

# Eye Movement and Visual Search: Are There Elementary Abnormalities in Autism?

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**Abstract** Although atypical eye gaze is commonly observed in autism, little is known about underlying oculomotor abnormalities. Our review of visual search and oculomotor systems in the healthy brain suggests that relevant networks may be partially impaired in autism, given regional abnormalities known from neuroimaging. However, direct oculomotor evidence for autism remains limited. This gap is critical since oculomotor abnormalities might play a causal role in functions known to be impaired in autism, such as imitation and joint attention. We integrate our oculomotor review into a developmental approach to language impairment related to nonverbal prerequisites. Oculomotor abnormalities may play a role as a sensorimotor defect at the root of impairments in later developing functional systems, ultimately resulting in sociocommunicative deficits.

**Keywords** Autism · Eye movement · Visual search · Joint attention · Language

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## Eye Movement in Autism: Why We Need to Know More

Despite decades of neuroscientific research on autism, the role of potential oculomotor abnormalities remains largely unexplored. There are some intriguing findings from a few studies indicating, for example, that autistic individuals show very unusual fixation trajectories when presented with complex social scenes (Klin, Jones, Schultz, Volkmar, & Cohen, 2002) or that saccade frequency may be abnormally elevated in autistic children even in the absence of a visual task (Kemner, Verbaten, Cuperus, Camfferman, & van Engeland, 1998). However, to date such results are few and far apart and are no more than tantalizing suggestions of a potentially fundamental abnormality of oculomotor systems in autism. The importance of this possibility—and the gravity of our lack of understanding—is magnified by the conceivable secondary effects that unexplored oculomotor abnormalities may have on other functional systems. For example, dysfunction of networks involved in saccade generation may result in increased saccade frequency at baseline (i.e., in the absence of saccade triggering stimuli). It is clearly conceivable that such abnormality would profoundly affect visual perceptual processing, predominantly in detrimental ways (as in impaired response to social and facial stimuli), but in positive ways for some types of task (such as visual search).

Language delay is one of the prominent features of autistic disorder and can serve as an example highlighting the need for oculomotor studies. As we will argue in detail below, language impairment in autism can only be understood through the study of functions that are *ingredients* of language acquisition, such as

polysensory integration, imitation, and joint attention (Müller, 2005). Arguably, it is not language as such, but these ingredient functions that are impaired in autism. Potential eye movement abnormalities would play a role because the ability to direct one's gaze to relevant regions of the visual field in well-timed saccades is fundamental to a person's ability to perceive and process stimuli crucial for accurate performance on an imitation or joint attention task. This consideration is not limited to the experimental setting. On the contrary, it is obvious that a typical word learning setting, in which a normally developing child will follow the mother's gaze to fixate on an object together with her while she pronounces the corresponding word, will be disrupted by dysfunctional eye movement.

A second consideration indicating the importance of visual search and oculomotor behavior in autism concerns the potential impact of atypical eye movement on behavioral and neuroimaging results. Even when one puts aside the possible fundamental role of oculomotor abnormalities in the development of autistic symptomatology sketched above and laid out in detail later in this chapter, atypical eye movement will still affect behavioral responses and hemodynamic activation effects. For example, Baron-Cohen et al. (1999) observed impaired performance and atypical activation patterns in autistic adults associated with the attribution of mental states to stimuli depicting pairs of eyes. In the absence of eye tracking data, the possibility remains that these behavioral and neurofunctional abnormalities may be due simply to the fact that autistic subjects do not visually scan such stimuli in the same way as controls (e.g., avoid looking at eyes). Quite probably, abnormalities of 'theory of mind' function are compounded by atypical oculomotor behavior.

In this review, we present a survey of the current knowledge on visual search and eye movement from the behavioral perspective and with regard to underlying neural systems. The review will contrast what is known for typically developing people with the much more limited evidence on people with autism. We will first discuss the main types of visual search and the underlying functional neuroanatomy, and will then turn to components of visual search, i.e., attention, saccadic eye movement, and smooth pursuit.

### Visual Search and Attention

Visual search makes demands on the orienting system and can thus be used to study the coordination of visual attention and eye movement. The visual search task,

whether it be the search for a letter in a list of words, a needle in a haystack, or a car in a parking lot, consists of a target embedded within a set of distracters. There are numerous variations of the basic visual search paradigm that have been employed experimentally. For example, a few of the major dimensions along which the task can vary are: similarity between target and distracters, orientation, set size, and velocity or direction of movement.

Feature binding, or temporal binding, is one of the central concepts of visual search. The term was originally defined cognitively as the proper conjunction of different sensory features into a whole percept (Treisman & Gelade, 1980), but it implies the cooperation of feature-specialized neuronal networks acting in synchrony to achieve coherency. In the visual search literature, tasks that require feature binding are *conjunctive* search tasks; tasks that do not are *feature* tasks. A simple feature task is one in which the target shares one dimension, like color, with all or some of the distracters, but differs in another dimension, like shape. Response time increases linearly with set size in a conjunctive search, but not in a feature search (Duncan & Humphreys, 1989; Treisman & Gelade, 1980; Wolfe, Cave, & Franzel, 1989).

Supposedly, feature searches are "parallel" searches because all items in the array can be processed simultaneously, causing the target to "pop out". Conjunctive searches, on the other hand, require serial search, or a more complex search that may involve focusing, disengaging, and then re-focusing of visual attention. Feature integration theory, originally proposed by Treisman and colleagues, is the theory that parallel and serial searches involve distinct types of processing (Hunt & Ellis, 1999), i.e., simple features are processed automatically, or in parallel, whereas a conjunction of multiple features requires that each feature be processed independently, or serially. It is thus the conjunction of dissimilar features, and not simply the number of features, that makes a search difficult enough to require a serial approach (Quinlan & Humphreys, 1987). According to Treisman's theory, integration of information across dimensions (for example, color and shape) cannot be performed automatically and thus calls for focused attention. The linear increase in response time as a function of set size in conjunctive searches would then be explained by the need to focus and re-focus attention on increasingly more items. Although there are obvious differences between conjunctive and feature searches, the parallel/serial dichotomy has been challenged by researchers (Desimone & Duncan, 1995; Duncan & Humphreys, 1989) who prefer to explain slowed search

in conjunctive tasks in terms of an efficiency continuum: the more complex the task, the more inefficient the system that underlies the search process.

In this review, we will employ the efficiency/inefficiency distinction in place of the parallel/serial distinction. The efficiency of a search can be manipulated by changing the distracters, e.g. increasing the target-distracter similarity, increasing the number of distracters, or increasing the heterogeneity of the distracters. An efficient search is one for which the target “pops-out” of the visual array and for which response time does not increase significantly when set size is increased. A feature search can be an inefficient search, a conjunctive search can be an efficient search, and vice versa. The efficiency of a search is determined by its level of difficulty, which is independent of, though often conflated with, feature binding (Nobre, Sebestyen, Gitelman, Frith, & Mesulam, 2002).

#### Is Visual Search a Consistent “Islet of Ability” in Autism?

Several studies indicate that individuals with autism show enhanced performance on conjunctive search tasks (O’Riordan, Plaisted, Driver, & Baron-Cohen, 2001; Plaisted, O’Riordan, & Baron-Cohen, 1998) and on the Embedded Figures Test [Johnson, 2002 (Unpublished manuscript); Jolliffe & Baron-Cohen, 1997; Shah & Frith, 1983]. Most of the current evidence for enhanced visual search in individuals diagnosed with autism comes from research with children, although a recent study by O’Riordan (2004) in autistic adults suggests that visual search remains a domain of relative strength in autism throughout life. However, individual variability may be substantial, even among high-functioning individuals with autism spectrum disorder (ASD). For instance, Kleinhans, Akshoomoff, and Delis (2005) found that 4 autistic subjects out of 14 were severely impaired on the visual scanning subtest of the Delis-Kaplan Executive Function System battery (Delis, Kaplan, & Kramer, 2001), whereas four subjects with Asperger’s disorder scored above normal. Superior visual search abilities may thus be task-specific or less consistent within the autistic population than is often assumed. Nonetheless, isolated strengths in visual search could provide insight into atypical oculomotor and attentional functioning in autism.

Plaisted et al. (1998) compared children with ASD to their typically developing peers using a visual search paradigm that consisted of both efficient (feature) and inefficient (conjunctive) searches. As expected, response time for both groups increased as a function of set size in the inefficient, but not in the efficient search

condition. This is consistent with the idea that target salience in an efficient (“pop-out”) search is unaffected by the number of distracters in the array because the target-distracter difference is maximized (Wolfe & Horowitz, 2004). Intriguingly, Plaisted et al. (1998) found that children with ASD were significantly faster and more accurate than controls in the inefficient search condition, regardless of set size. Even when nonverbal IQ is controlled, children with ASD show enhanced performance on inefficient visual search tasks (O’Riordan et al., 2001). As in the study by Plaisted and colleagues, O’Riordan et al. (2001) employed both a feature search and a conjunctive search. O’Riordan et al. (2001) also extended earlier findings by adding an inefficient feature search to the paradigm, enabling them to separate search difficulty and conjunction of features. Enhanced performance in the ASD relative to the control group was found for all inefficient search types. Interestingly, the difference was especially pronounced in the target-absent conditions. The results are interpreted in light of the unique item detection theory of superior visual search in autism, a theory which attributes superior performance to superior discrimination for objects of interest (O’Riordan et al., 2001), rather than to weak central coherence as stipulated by Frith (1989). Central coherence is the ability to integrate pieces into a whole precept, or, “the tendency to process incoming information in context for gist—pulling information together for higher level meaning, often at the expense of memory for details” (Happe, 1998, p. 2). Conversely, the unique item detection theory allows for mostly intact central coherence, but suggests that individuals with autism have a preference for detail when not specifically instructed to pay attention to the whole of a stimulus array (O’Riordan et al., 2001).

A second type of paradigm in the study of visual search is the Embedded Figures Test (EFT), which is an advanced visual search task. The test requires subjects to locate a simple shape within a complex figure and was originally designed to evaluate “field independency” (Witkin, Oltman, Raskin, & Karp, 1971). Field independency is the ability to separate relevant material from its context or, in visual search terms, to distinguish signal from surrounding noise (Verghese, 2001). Because subjects with ASD tend to do very well on the EFT, it has been suggested that ASD is characterized by a preference for local rather than global level processing (Mottron, Burack, Iarocci, Belleville, & Enns, 2003). The first report of enhanced EFT performance in autism was published in 1983 (Shah & Frith, 1983). The results from this study, often cited as evidence for weak central coherence in autism

(Frith, 1989; Happe, 1998), were subsequently replicated by Jolliffe and Baron-Cohen (1997) and by Johnson (2002, Unpublished manuscript). All three studies found that subjects with autism were significantly faster than control subjects when asked to find a target shape within the geometric pattern that contained it.

Very little imaging evidence is currently available to corroborate the behavioral findings of enhanced visual search in autism. To our knowledge, only two imaging studies of visual search have been conducted, both applying variations of the Embedded Figures Test (Manjaly et al., 2004; Ring et al., 1999). Ring et al. (1999) selected 10 figures from the original EFT for a functional MRI (fMRI) blocked design. Task conditions (embedded figures) were alternated with presentation of a baseline condition (blank screen). Between-group statistical analysis of the imaging data led the authors to conclude that the control group had greater activation in bilateral parietal cortex and right dorsolateral prefrontal cortex, while the ASD group had greater activation in the right occipital cortex and inferior temporal gyrus (Ring et al., 1999). However, Ring et al. used only a small set size (10 figures) and did not report response times. Onscreen duration for each figure was 15 seconds, potentially associated with long periods of uncontrolled cognitive events. A precise interpretation of the hemodynamic findings from this study is therefore difficult.

In a study only published as yet in abstract form, Manjaly et al. (2004) used an fMRI block-design to evaluate the neurofunctional correlates of visual search in 12 adults with ASD and 12 healthy control subjects. The task consisted of a previously validated set of figures (Manjaly et al., 2003) that was similar, though not identical to, the original Embedded Figures Test (Manjaly et al., 2004). As hypothesized, subjects with ASD were significantly faster and made fewer errors than controls in locating embedded figures. Neurofunctionally, the authors reported that the ASD group showed greater activation in the right temporoparietal junction, the right superior temporal gyrus, and the left cerebellum than did the control group. The control group exhibited greater activation in the left middle frontal gyrus, the left intraparietal sulcus, and the right lingual gyrus (Manjaly et al., 2004). Even when one puts aside the limited consistency of findings from this study and the earlier one by Ring et al. (1999), a crucial question of the visual search process remains unanswered: Are superior visual search and unusual regional brain activations in autism associated with atypical eye movement? In order to answer this

question, we will first need to review the neuroanatomy of visual search in greater detail.

### Neuroanatomy of Visual Search

In this section, we will review the brain circuitry for visual search in the typically developing brain and in relation to evidence of regional brain abnormalities in ASD.

Visual search elicits widespread activation because it relies on extensive, yet often overlapping, neural systems responsible for attention (and/or visual selection), eye movement, and object identification. The primary regions involved in visual search are the frontal eye fields (Gitelman, Parrish, Friston, & Mesulam, 2002; Muggleton, Juan, Cowey, & Walsh, 2003), the dorsolateral prefrontal cortex (Makino, Yokosawa, Takeda, & Kumada, 2004; Nobre, Coull, Walsh & Frith, 2003), the parietal lobe (Müller et al., 2003; Nobre et al., 2003; Nobre, Gitelman, Dias, & Mesulam, 2000), the temporal–parietal junction (Corbetta, Kincade, Ollinger, McAvoy, & Shulman, 2000; Hayakawa, Miyauchi, Fujimaki, Kato, & Yagi, 2003; Müller et al., 2003), the superior colliculus (Gitelman et al., 2002), and the cerebellum (Gitelman et al., 2002; Makino et al., 2004; Nobre et al., 2003; Wilkinson, Halligan, Henson, & Dolan, 2002). For many of these regions, no consensus has yet been reached regarding their role in attentional versus oculomotor functions. Nevertheless, the convergence of human and animal studies provides a general outline of the regions participating in visual search. In this section, we will briefly review the most important of these regions. More detailed discussions of their precise roles will follow in later sections regarding oculomotor and attentional components of visual search.

The literature documenting regional brain involvement in autism based on anatomical or functional anomalies is extensive (for reviews see Akshoomoff, Pierce, & Courchesne, 2002; Brambilla et al., 2003). Although not all results have been replicated, findings of abnormality in numerous subcortical, cerebellar, and cerebral cortical regions in at least some samples of ASD subjects suggest that autism is not only a pervasive disorder in view of its behavioral manifestations, but also a highly distributed disorder with regard to its neuroanatomical involvement. This section, however, will be strictly limited to regions known to participate in eye movement and visual search. Anatomical abnormalities do not per se imply functional impairment, but it remains important to determine

whether eye movement and visual search may be among the affected domains. Due to space limitations, we will not attempt a comprehensive review, but will solely refer to exemplary studies that establish the involvement of these brain regions of interest.

### Frontal Eye Fields and Prefrontal Cortex

The frontal eye field (FEF) is among the most reliably activated regions in visual search paradigms (e.g. Gitelman et al., 2002; e.g. Müller et al., 2003; Nobre et al., 2003). Because oculomotor paradigms show that the FEF plays a critical part in the process of saccade generation (Gaymard, Ploner, Rivaud, Vermersch, & Pierrot-Deseilligny, 1998; Luna et al., 1998), FEF activation during unconstrained visual search could be related to saccadic activity. However, several studies outline a broader role for the FEF, showing that FEF is responsible for visual selection, or saliency mapping, rather than saccade generation per se (Gitelman et al., 2002; Juan, Shorter-Jacobi, & Schall, 2004; Muggleton et al., 2003; Murphy, Thompson, Schall, 2001). According to Koch and Ullman (1985), the FEF—and possibly the lateral intraparietal region (LIP)—may support representations formed from the retinal image such that the level of neural activity at any given point encodes the saliency of items in a corresponding location in the visual field (Findlay & Gilchrist, 2003). Saliency might be based on stimulus properties such as similarity to the target or proximity to the current fixation.

Activation in the dorsolateral prefrontal cortex (DLPFC) is occasionally reported in imaging studies of visual search (Makino et al., 2004; Nobre et al., 2003). One possibility, consistent with a role for DLPFC in executive functions, is that this region is involved in the manipulation or monitoring of distractors in a visual search display (Makino et al., 2004). Alternatively, oculomotor paradigms (discussed in detail below) indicate that the DLPFC is a key player in the network underlying intentional eye movements. For example, the DLPFC may contribute to memory-guided saccades and the inhibition of unwanted reflexive saccades (Pierrot-Deseilligny, Muri, & Ploner, 2003).

Frontal lobe involvement in autism has been suspected for a long time (Damasio & Maurer, 1978). In recent years, evidence has accrued for growth abnormalities (early postnatal overgrowth followed by abnormally flat growth curves; Courchesne et al., 2001) being especially pronounced in frontal lobes (Carper, Moses, Tigue, & Courchesne, 2002). Carper and Courchesne (2005) further observed that this

pattern of early overgrowth, suspected to derail experience-driven regional cortical differentiation and fine-tuning of functional networks, was significant only in dorsolateral and medial (but not precentral or orbitofrontal) portions of the frontal lobes. This appears to be consistent with findings of abnormal white matter enlargement in children with autism specifically in radiate compartments of the frontal lobe (Herbert et al., 2004). The volumetric evidence suggests that FEF and regions of DLPFC, whose involvement in visual search was discussed above, are probably affected by growth abnormalities. However, Luna et al. (2002) observed normal levels of activation in FEF and SEF of autistic participants associated with an oculomotor delayed response task. A conservative interpretation based on currently preliminary evidence may suggest that while gray matter and cortical architecture in FEF and SEF are possibly not affected in autism, abnormalities in adjacent white matter are likely to impair the participation of these regions in functional circuits important for eye movement and visual search.

### Parietal Cortex

Like the FEF, the parietal lobe is consistently activated by visuo-spatial tasks and is considered part of the network integral to effective visual search. Functional subdivision within the parietal lobe is difficult because both spatial attention and saccade generation activate adjacent, and possibly overlapping, parietal regions. Variable results in the literature may be attributed to the specifics of individual task paradigms. Although task differences make inter-study comparison challenging, converging evidence on the role of the intraparietal sulcus and of posterior parietal regions yields a coherent account of search-related activation. For example, some of the variation in parietal lobe activation may be related to search efficiency. Both Nobre et al. (2003) and Donner et al. (2002) found activation in the posterior parietal lobe and intraparietal sulcus (IPS) for difficult search conditions when compared to easy feature conditions. Wilkinson et al. (2002) manipulated efficiency by varying distractor similarity and found activation patterns similar to Donner et al. (2002) and Nobre et al. . Bilateral superior parietal activation, including the IPS, was associated with a heterogeneous distractor condition but not a homogeneous condition. As reflected in response time data, the heterogeneous condition was the more difficult condition of the two (Wilkinson et al., 2002). These studies show that the IPS is reliably activated by inefficient and conjunction search tasks.

Abnormal growth patterns affecting gray and white matter (and therefore presumably compromising both local cortical integrity and interregional connectivity) are not limited to frontal lobes, but reflect a more global pattern in autistic brain development (Courchesne et al., 2001), also affecting parietal and temporal lobes (Carper et al., 2002). In one study, over 40% of a sample of older autistic children and adults had reduced gray or white matter volume in the parietal lobes (Courchesne, Press, & Yeung-Courchesne, 1993), correlating with impairments of visuospatial attention (Townsend & Courchesne, 1994). Two recent studies on visuospatial attention have shown reduced activity in parietal lobes in subjects with ASD (Belmonte & Yurgelun-Todd, 2003; Haist, Adamo, Westerfield, Courchesne, & Townsend, 2005). The findings by Haist and colleagues indicate specifically reduced inferior parietal activity associated with an impairment of rapid and automatic spatial attention, whereas voluntary top-down attentional regulation appears to be relatively spared.

#### Temporal Cortex

Activation in the temporal lobe related to visual search is less commonly reported than activation in the occipital-parietal-frontal network. In an MEG study, Hayakawa et al. (2003) reported activation in the posterior superior temporal sulcus (STS) for both pop-out and non pop-out search tasks. This finding was interpreted as a correlate of target identification, or “extraction of the target as the relevant stimulus” (Hayakawa et al., 2003). Shulman et al. (2003) observed activation in the temporal parietal junction (TPJ) during target-detection, but not during visual search (Shulman et al., 2003), and Müller et al. (2003) found TPJ activity during target presentation when subjects had been cued to target location. As Shulman et al. (2003) suggest, it may be that a subset of cells in the TPJ encode target stimulus properties, and that the enhanced BOLD response in the TPJ reflects activity in this subset of cells when stimuli resemble or match the target. In light of these findings, TPJ activation most likely corresponds to target identification, though the magnitude of that response may be mediated by variables such as expectation of target location (Corbetta et al., 2000) and search load (Müller et al., 2003).

While there is extensive evidence of involvement of the mediotemporal region in autism (Schumann et al., 2004; Sparks et al., 2002; Sweeten, Posey, Shekhar, & McDougle, 2002), little is known about potential structural abnormalities in lateral temporal regions presumed to participate in visual search. However,

several functional studies show that lateral temporal lobe impairment may be a characteristic of the autistic brain. For instance, a positron emission tomography (PET) study of autistic and typical children showed reduced regional blood flow during sleep in bilateral superior temporal cortex as well as right STS (Zilbovicius et al., 2000). Reduced activity in STS, an area previously identified in visual search tasks (Hayakawa et al., 2003), has also been observed in activation studies of autism (Castelli, Frith, Happe, & Frith, 2002; Gervais et al., 2004), though for conditions not obviously related to visual search (attributing mental states to interacting triangles; perception of biological sounds). Possibly linked to these functional findings, white matter compromise (reduced fractional anisotropy) in the left posterior lateral temporal lobe was seen in a diffusion-tensor MRI study (Barnea-Goraly et al., 2004).

#### Superior Colliculus

Activation in the superior colliculus (SC) has been examined by relatively few imaging studies, probably on account of its small size and deep location in the brain. However, the study by Gitelman et al. (2002) described earlier yielded evidence for superior colliculus activation for the “explore search” condition, when compared to saccade and central conditions. The implication is that SC activation is not solely limited to saccadic eye movements. It has been suggested that besides saccade generation the SC, like the FEF, is involved in the more general process of saccade target selection (Findlay & Gilchrist, 2003; McPeck & Keller, 2004). For example, multi-cell recording in the macaque SC during visual search reveals activity related to eye movement initiation, but also to selection of the next saccade target (McPeck & Keller, 2002). McPeck and Keller (2002) suggest that the transition from saccade target selection to saccade generation may take place in the SC.

Some authors have argued for brainstem involvement in autism (Hashimoto et al., 1995; Rodier, 2002). In postmortem studies, Bailey et al. (1998) and Rodier, Ingram, Tisdale, Nelson, and Romano (1996) found abnormalities in brainstem regions such as inferior olive (Bailey et al., 1998), facial nucleus, and superior olive (Rodier et al., 1996). However, there is currently no evidence specifically implicating the superior colliculus in autism. It should be noted, though, that due to its small size this structure is hard to visualize in functional neuroimaging studies with typically limited spatial resolution.

## Cerebellum

Both cerebellar hemispheres have been found to activate during visual search (Gitelman et al., 2002; Makino et al., 2004; Nobre et al., 2003; Wilkinson et al., 2002). Wilkinson et al. (2002) observed left cerebellar activity when subtracting a homogeneous distractor condition from a heterogeneous one, and bilateral cerebellar activity when comparing the heterogeneous distractor condition to baseline. Nobre et al. (2003) found that right cerebellar activity is common to search conditions of varying difficulty, but that left cerebellar activity is only found when efficient conditions are subtracted from inefficient conditions. Both the studies by Nobre et al. (2003) and Wilkinson et al. (2002) thus suggest that left cerebellar activity is associated with difficulty level, i.e. that inefficient conditions elicit activation in the left cerebellum more than efficient ones. These findings are consistent with the hypothesis that the cerebellum mediates rapid shifts of attention (Akshoomoff, Courchesne, & Townsend, 1997). Cerebellar involvement in search probably reflects in part activity of the oculomotor system. As discussed below, the cerebellum contributes to saccade and pursuit eye movement, although it remains unclear whether it is the attentional aspect of eye movement or the oculomotor component of attention-shifting that brings the cerebellum online.

In spite of the high individual and inter-study variability in brain morphology, the cerebellum has emerged as one of the more consistent sites of cellular and volumetric abnormality in autism (Brambilla et al., 2003; Palmen & van Engeland, 2004). The number of Purkinje cells in the autistic cerebellum has been found to be lower than normal in a majority of postmortem cases (Bailey et al., 1998; Kemper & Bauman, 1993; Lee et al., 2002; Ritvo et al., 1986). Furthermore, Fatemi et al. (2002) report a 24% decrease in the average *size* of Purkinje cells in autism, which suggests that cellular aberrations in the cerebellum can not solely be characterized by cell counts. The appearance of Purkinje cells may also vary with age. Kemper and Bauman (1993) found that subjects older than 22 years had neurons that were smaller and paler in color in comparison to a younger group. This finding is supported by MRI studies showing age-related changes in cerebellar structure (see Akshoomoff et al., 2002 for review), highlighting once again the developmental complexity of autistic pathogenesis.

In a study of 60 boys with autism (ages 2–16 years), Courchesne et al. (2001) found that the volume of cerebellar white matter in 2- to 3-year-old autistic subjects was 39% greater than in age-matched controls.

This study also suggests that the developmental time course of cerebellar white matter volume is atypical in autism. Between ages 2 and 16, white matter volumes in the control group increased by about 50% compared to only 7% in the autism group (Courchesne et al., 2001). Age-related changes and variability within the autistic population may in part explain why MRI studies of the cerebellar vermis have yielded contradictory results (as reviewed in Brambilla et al., 2003). Studies led by Courchesne et al. (1988) and Hashimoto et al. (1995) are among those showing a reduction in the size of cerebellar vermis lobules VI–VII. Garber & Ritvo (1989), Ciesielski, Harris, Hart, and Pabst (1997), and others did not replicate these findings. Nonetheless, it appears likely that the cerebellum is one of the sites of abnormality in autism.

## Oculomotor Components Of Visual Search

The above neuroanatomical review indicates that different brain regions participating in the visual search circuitry may be involved in more specific component processes of visual search. In this section, we will discuss in greater detail some of these component processes, focusing on saccadic eye movement and visual attention.

### Saccades

Eye movements can be involuntary and automatic, like the vestibulo-ocular and optokinetic reflexes, or under rudimentary volitional control, like the saccadic, pursuit, and vergence systems. As long as both subject and stimulus are stationary in a visual search task, saccades are the expected type of eye movement (Findlay & Gilchrist, 2003). The saccadic system permits rapid rotation of the eye in order to bring items of interest onto the fovea where objects can be processed with enhanced acuity.

Among the commonly measured parameters of saccadic eye movement are *amplitude* (size, or angular rotation, of the saccade), *duration* (time taken to reach the target), and *peak velocity* (Findlay & Gilchrist, 2003). Because the relationships between amplitude, duration, and peak velocity are relatively fixed (e.g., in normal subjects the relationship between duration and amplitude is linear), there is a main sequence for saccades that can be used to determine whether or not a saccade is normal (Knox, 2004b). *Accuracy* is determined by amplitude: a saccade of insufficient amplitude (hypometric) would undershoot the target and a saccade of excessively high amplitude (hypermetric)

would overshoot the target (Findlay & Gilchrist, 2003). *Latency* is the time that elapses between the appearance of a target and the onset of a saccade in response to that target (Findlay & Gilchrist, 2003).

### *Neural Mechanisms*

Saccadic eye movements can be driven by either top-down control, mediated by higher cortical systems, or by bottom-up input from subcortical regions. An exogenous, or reflexive, saccade occurs in response to a visual stimulus. An endogenous saccade is more intentional and may be guided by processes such as memory, prediction, and inhibition. The principal regions that make up the saccade system are the FEF, the supplementary eye field (SEF), DLPFC, the parietal eye field (PEF) in humans, lateral intraparietal cortex (LIP) in rhesus monkeys, SC, basal ganglia, and cerebellum.

### *Saccade System in Primates*

The substantia nigra pars reticulata and caudate regions of the basal ganglia are believed to exert influence on the SC through both inhibition and disinhibition of purposive saccade generation (see Hikosaka, Takikawa, & Kawagoe, 2000 for review). These inputs may be modulated by cognitive functions such as working memory (Hikosaka, Sakamoto, & Usui, 1983) and expectation (Apicella, Scarnati, Ljungberg, & Schultz, 1992; Hikosaka, Sakamoto, & Usui, 1989). Additionally, the SC receives saccade-related input from the FEF and the LIP. One way in which FEF-SC connectivity has been addressed is by electrical stimulation of the FEF when the SC is temporarily inactivated (Hanes & Wurtz, 2001) or while recording from cells in the SC (Schlagg-Rey, Schlag, & Dassonville, 1992). For example, Schlagg-Rey et al. (1992) recorded cellular activity in the intermediate layer of the macaque SC while applying microstimulation to FEF saccade-cells. Stimulation of the FEF elicited saccadic eye movement and excited cells in the SC that were known to encode the same vector as the electrically evoked saccades (Schlagg-Rey et al., 1992). As mentioned, the SC also receives significant input from areas of parietal cortex such as the LIP. In rhesus monkeys, autoradiographic tracing illuminates LIP fibers that traverse the posterior limb of the internal capsule and terminate in the oculomotor layers of the SC (Gaymard, Lynch, Ploner, Condy, & Rivaud-Pechou, 2003). When humans with small lesions to this region of the internal capsule are tested on

oculomotor paradigms, they show significant deficits in unpredictable-saccade conditions but not in memory- or predictable-saccade conditions (Gaymard et al., 2003). In addition to the extensive connectivity between cortical eye fields and the SC, there is direct communication between the FEF and LIP (Ferraina, Pare, & Wurtz, 2002; Stanton, Bruce, & Goldberg, 1995). Furthermore, Stanton et al. (1995) report evidence that the LIP may be functionally organized by saccade amplitude. Projections from FEF regions associated with large saccades terminate in cytoarchitecturally different areas of the LIP than projections from FEF regions associated with small saccades (Stanton et al., 1995).

Finally, there is evidence for cerebellar involvement in saccade generation. Lesions restricted to the oculomotor vermis of the cerebellum (centered in lobules VI and VII) cause significant disruptions in saccade amplitude and velocity in rhesus monkeys (Takagi, Zee, & Tamargo, 1998). The caudal fastigial nucleus, which receives projections from the oculomotor vermis, is also thought to play a role in the control of saccadic eye movement (see Robinson & Fuchs, 2001 for review). The physiological picture, then, is one in which saccadic eye movement is supported by a network that includes cerebellum, basal ganglia, superior colliculus, and cortical eye fields.

### *Lesion and Imaging Evidence in Humans*

Frontal regions associated with saccadic eye movement in humans are FEF, SEF, and DLPFC. Patients with DLPFC lesions are impaired on intentional saccade tasks, with fewer anticipatory saccades in predictive saccade tasks and reduced accuracy on anti-saccade and memory-guided saccade tasks (Pierrot-Deseilligny et al., 2003). Consistent with the hypothesis of DLPFC involvement in memory-guided saccades, rTMS applied to DLPFC during the memory phase of a saccade task (prior to saccade generation) disrupts both the amplitude and the direction of subsequent saccades (Brandt, Ploner, Meyer, & Leistner, & Villringer, 1998). Correspondingly, fMRI detects enhanced signal in the DLPFC during the instruction phase of anti-saccade trials relative to pro-saccade trials, but no difference in DLPFC activation during actual saccade execution (DeSouza, Menon, & Everling, 2003). The above studies suggest that the contribution of the DLPFC to the saccade system is related to executive components such as preparatory set and maintenance of task-relevant information.



The FEF and SEF play a central part in the network associated with saccadic eye movement. Contrary to traditional localization of the FEF in Brodmann area 8, recent fMRI studies place the FEF posterior to area 8 in the precentral sulcus (Kato & Miyauchi, 2003; Luna et al., 1998). Specifically, Kato et al. found activation centered in the superior precentral region for saccade-only trials when contrasted with blink-only trials. The BOLD response in the FEF corresponds to saccade generation, but may also correspond to the initial phase prior to target presentation. Cornelissen et al. (2002) demonstrated that activity in the FEF for both pro- and anti-saccade trials precedes target presentation, which may be indicative of preparatory set prior to movement initiation. The SEF has been localized in the medial portion of the superior frontal gyrus (Luna et al., 1998). Inter-subject consistency in activation during self-paced saccades suggests that the upper part of the paracentral sulcus may be a stable landmark for the SEF (Grosbras, Lobel, Van de Moortele, LeBihan, & Berthoz, 1999).

The eye-movement related region within the posterior part of the superior parietal lobe, sometimes referred to as the parietal eye field (PEF), is thought to be roughly homologous to the LIP in macaques (Koyama et al., 2004). In fMRI paradigms, this region activates during visually-guided (reflexive) saccade tasks (Berman et al., 1999; Mort et al., 2003) and also during voluntary saccade tasks (Mort et al., 2003). Specifically for visually-guided saccades, Luna et al. (1998) found saccade-related activation in precuneus and along the intraparietal sulcus in superior and inferior parietal cortex. Konen, Kleiser, Wittsack, & Bremmer, and Seitz (2004) observed activation in the posterior part of the intraparietal sulcus (IPS) for both unpredictable (reflexive) saccade and predictable (voluntary) saccade conditions, but activation in the anterior part of the IPS only for the predictable saccades (Konen et al., 2004). Current evidence thus suggests that voluntary and reflexive saccades activate at least partly overlapping regions in posterior parietal cortex.

The role of the cerebellum in the saccade network has also been demonstrated in human studies. Hayakawa, Nakajima, Takagi, Fukuhara, and Abe (2002) conducted a detailed fMRI analysis of cerebellar activation during horizontal and vertical visually-guided saccades. For all of the subjects studied, significant activation was found in the vermis between the primary fissure and the horizontal fissure (vermis declive and folium) and in parts of the superior semilunar lobule (Hayakawa et al., 2002). In a second fMRI study, visually guided saccades, relative to

fixation, were found to activate the posterior vermis within lobuli VI–VIII (Nitschke et al., 2004). In contrast, memory-guided saccades, relative to fixation, were associated primarily with activation in the left cerebellar hemisphere (Nitschke et al., 2004).

### *Saccadic Eye Movement in Autism*

Since eye movement is necessary for unconstrained visual search (Findlay & Gilchrist, 2003), the study of the oculomotor system may provide an explanation for the visual search superiority often seen in children and adults with autism. Although there are inconsistencies in the literature (see Sweeney, Takarae, Macmillan, Luna, & Minshew, 2004), a number of studies indicate that saccadic eye movement is abnormal in individuals with autism (e.g., Goldberg et al., 2002; e.g., Kemner et al., 1998; van der Geest, Kemner, Camfferman, Verbaten, & van Engeland, 2001). In several of these studies, a visually-guided saccade task was used in conjunction with intentional-saccade tasks such as the memory-guided, predictive, and anti-saccade tasks. The visually-guided task is designed to assess target-elicited saccades, the saccades that are driven by exogenous stimuli and may not require intentional mechanisms. However, task performance is still contingent upon the ability to generate a saccade in coordination with a specific visual stimulus. The reflexive saccade system at rest, or in the absence of task constraints, has not been well-studied in autism despite initial evidence of abnormalities (Kemner et al., 1998).

The first study of basic oculomotor function in autism relied on electrooculography (Natsopoulos, Kiosseoglou, Xeromeritou, & Alevriadou, 1988; Rosenhall, Johansson, & Gillberg, 1988). The authors used a non-predictive (visually-guided) saccade task and recorded saccade velocity, latency, and accuracy. Subjects maintained central fixation until a light emitting diode was illuminated in the periphery. Although abnormalities were found in only 6 of the 11 children with autism, most of the autistic children had reduced accuracy and lower peak velocity compared to the control group. No differences in latency were found. A second EOG study by Minshew, Luna and Sweeney (1999), which used a visually-guided saccade task similar to the task used by Rosenhall et al. (1988), found no difference between autism and control groups in accuracy, velocity, or latency. Additionally, Minshew and colleagues recorded eye movement during an anti-saccade task (in which subjects had to suppress reflexive saccades by generating a

saccade away from the stimulus) and a delayed-response saccade task (in which subjects had to maintain central fixation while simultaneously encoding the peripheral location of a stimulus). Compared to the control group, the autism group made more response suppression errors in both the anti-saccade task and the delayed-response saccade task. The subjects with autism also showed reduced accuracy of saccades to the location of a previously occurring stimulus in the delayed-response saccade task (Minsheiw et al., 1999).

The finding of increased response suppression errors in autism was replicated by Goldberg et al. (2002). For this study, eye movement was recorded using infra-red oculography, a method with better spatial resolution than EOG (Knox, 2004a). The paradigm consisted of a saccade task, an anti-saccade task, a memory-guided saccade task, and a gap/overlap task. Compared to the control group, the autism group made more response suppression errors in the anti-saccade and memory-guided saccade tasks. In the memory-guided saccade task, but not the anti-saccade task, the autism group had overall higher response latencies. In the predictive saccade task, the autism group had a significantly higher variance in latency and made fewer saccades overall. The results from the gap/overlap task—a task previously implemented by van der Geest et al. (2001) which comprises three conditions—are more difficult to interpret. In the gap condition, the central fixation point disappears before the onset of the peripheral target. In the overlap condition, the central fixation point remains visible after the onset of the peripheral target. The target onset and disappearance of the central fixation point are simultaneous in the null condition (Goldberg et al., 2002). When measuring latency, a “gap effect” is obtained by subtracting the gap condition from the overlap condition (van der Geest et al., 2001). Goldberg and colleagues found that mean latency for all three conditions (gap, null, and overlap) was greater for the autism group than for the control group. However, there was no significant group difference in the gap effect (Goldberg et al., 2002). Conversely, van der Geest et al. (2001) found no significant difference in mean latency between groups, but a significantly reduced gap effect in a group of high-functioning autistic boys (compared to typically developing boys). Thus, the only two studies of autism employing the gap/overlap paradigm have yielded exactly opposite findings.

While current evidence suggests that oculomotor abnormalities are mostly found for conditions that involve facial stimuli (Klin et al., 2002; Pelphrey et al., 2002) or require top-down executive control, (such as

response inhibition or working memory; Minsheiw et al., 1999), results from a study by Kemner et al. (1998) suggest that more elementary abnormalities of the oculomotor system may also exist in autism. Kemner and coworkers evaluated eye movement in autistic children using an oddball task that consisted of frequent, rare, and novel pictures. Saccadic frequency in the autism group, as compared to comparison groups, was significantly greater both during presentation of frequently occurring stimuli and during inter-stimulus intervals. In contrast, normal controls showed greater saccadic activity during the presentation of novel stimuli. Increased saccadic activity during inter-stimulus intervals may indicate a basic deficit in eye movement that is stimulus independent (Kemner et al., 1998). Potentially related to this finding, Nowinski, Minsheiw, Luna, Takarae and Sweeney (2005) recently reported that intrusive saccades during fixation—although overall in the normal range in autistic individuals—were characterized by greater amplitude and reduced intersaccade intervals when the fixation target was remembered (rather than present). Nonetheless, it remains unclear what precise conditions may prompt increased frequency or atypical characteristics of saccades in autism. Note that Goldberg et al. (2002) reported *decreased* saccadic frequency in the autism group during a predictive saccade task. One possibility that is consistent with the current literature would imply that saccade frequency at baseline (during “rest”) and during tasks requiring little effort is abnormally increased in autism. Seemingly contradictory findings, as in the predictive saccade task (Goldberg et al., 2002), may be related to executive components, consistent with evidence for executive impairment in autism (Minsheiw, Sweeney, & Luna, 2002).

### Smooth Pursuit

This review focuses on the eye movements associated with static search displays. When a search array consists of moving objects (e.g., Nobre et al., 2003), both saccadic and smooth pursuit eye movements are expected. If stimuli are in motion, it is the smooth pursuit system that keeps the fovea trained on the stimulus by slowly rotating the eyes. The initial phase of smooth pursuit, typically defined as the first 100 ms, is called the open-loop stage because it is thought to be primarily driven by exogenous information. The closed-loop stage of pursuit is the phase in which feedback, such as predictive or performance-based information, is used to update eye movement. During either stage, express saccades are sometimes needed to

correct performance if the stimulus movement is under- or overestimated. The neuronal network that serves smooth pursuit includes many of the same areas that underlie saccade generation (Krauzlis, 2004), even though areas like the FEF may comprise functionally distinct smooth pursuit and saccade sub-regions (Rosano et al., 2002).

### *Smooth Pursuit in Autism*

Although a few studies exist (Hideo, 1987; Kemner, van der Geest, Verbaten, & van Engeland, 2004; Takarae, Minshew, Luna, Krisky, & Sweeney, 2004), smooth pursuit in autism has not been well-documented. A recent paper by Takarae et al. (2004) details evidence for impaired smooth pursuit in individuals with autism. Eye movements of 60 autistic individuals and 94 typical individuals were recorded during presentation of a foveofugal step-ramp task, a pure ramp task, and an oscillating target task. All tasks began with central target presentation. In the foveofugal ramp task, the target appeared centrally before stepping 3° right or left and then continuing to move at a constant speed away from center. Autistic participants had significantly reduced gain for the initial catch-up saccade in the foveofugal step-ramp task, and reduced pursuit gain during the ensuing open-loop stage for targets in the right visual field. The reduction in saccade gain to the right visual field was especially pronounced in younger autistic participants. Results from all three tasks evidenced a bilateral reduction in closed-loop pursuit gain for the autism group, in contrast to the control group, that was independent of target direction and velocity. Closed-loop deficits also varied with age: the reduction in closed-loop pursuit gain was more pronounced in autistic individuals over age 16 years (Takarae et al., 2004).

### Attentional Components of Visual Search

The relationship between visual attention, or visual selection, and saccadic eye movement is not fully understood, but these systems are clearly interdependent. Attentional processes can be classified as either overt or covert. Visual search typically involves overt fixation on an item of interest, with simultaneous covert attention to items in the periphery. Analogously, eye movement during visual search entails fixation, followed by saccade preparation and saccadic shifts to a new target. The question remains how the dual orienting processes of attention and eye movement interact. It has been theorized that eye movement

and attention may co-occur as independent processes; that covert attention may be a by-product of saccade preparation; or that a shift in covert attention may precede a saccadic eye movement (Findlay & Gilchrist, 2003, p 42). In support of this third alternative, enhanced visual discrimination at saccade targets has been found *prior* to actual saccades (Deubel & Schneider, 1996; Kowler & Blaser, 1995). Whatever the relationship, it is clear that both eye movement and attention need to be considered when interpreting the neurological response to tasks that require visual orienting. Visual selection and the saccades that are generated in response to information obtained through covert mechanisms may be dissociable, but they work in tandem and are best understood as part of a larger system.

Although rarely considered jointly in studies of autism, effective orienting relies on both saccade generation and the ability to shift attention. Because processes like covert attention and preparation for saccade generation are difficult to separate, the question of whether impairments in autism are specifically and selectively attentional or whether they may be related to eye movement abnormalities has yet to be answered (see Akshoomoff et al., 1997). Research on attention shifting in individuals with autism has mostly relied on cue-to-target and bimodal paradigms, making it difficult to extrapolate to visual search. Courchesne et al. (1994) found that children and adolescents with autism are able to focus attention in a single modality, but show significant impairment in executing rapid shifts of attention between sensory modalities. The decrement in performance for the attention shifting task could not be explained as an inability to understand the task, because subjects with autism were able to achieve near-normal levels of accuracy when given more time. Individuals with autism are also slower or impaired when shifting attention between spatial locations (Harris, Courchesne, Townsend, Carper, & Lord, 1999; Townsend, Courchesne, & Egaas, 1996; Wainwright-Sharpe & Bryson, 1993, 1996). For example, Harris and colleagues recorded the performance of children with autism on a spatial orienting task. The onset of a visual display was followed by either an attention-directing cue (one of the two boxes flanking the central fixation point brightened) or no cue (neither box brightened). Trials were cued validly, invalidly, or not at all and children were instructed to press a button as soon as the target was detected in either of the two boxes. Compared to typical children, children with autism were slower at detecting validly cued targets (Harris et al., 1999).

Another study found that an ASD group showed greater susceptibility to distracters in certain conditions, as measured by response time (Burack, 1994). The conditions in this study varied with respect to the presence or absence of a window highlighting the target and with respect to the number (0,1,2,4) and location of distracters. The target, either a plus sign or a circle, always appeared in the middle of the screen and subjects were instructed to press a button corresponding to the specific target stimulus. The hypothesis, according to which the window would offset the target and facilitate performance by aiding the focus of attention, was confirmed for both the autism and the comparison groups. However, when the distracter variable was introduced, it was found that subjects in the autism group failed to benefit from the presence of a window. In other words, subjects with autism were particularly susceptible to the distracters—even when the distracters were far away from the target—and this susceptibility was reflected in increased response times (Burack, 1994). These results may appear inconsistent with the general view of enhanced visual search ability in autism and highlight the need for oculomotor data that would permit a better explanation of such unexpected behavioral findings.

### Development of Oculomotor Function and Attention

Since ASD is a developmental disorder with prenatal or early postnatal onset (Courchesne & Pierce, 2005; Miller et al., 2005), the question arises what causal role potential oculomotor abnormalities could play in the development of autistic symptomatology. In this section, we will very briefly sketch developmental milestones of oculomotor functions in typically developing children.

Although infants are not born with fully mature oculomotor systems, observed eye movements indicate that the basic mechanisms are in place. Infants are able to visually track a moving stimulus at 8 weeks (Richards & Holley, 1999) or possibly even as young as 2 weeks of age (Lengyel, Weinacht, Charlier, & Gottlob, 1998). By 4 months of age, infants may be capable of learning to inhibit reflexive saccades to a peripheral distractor when it predicts the location of a more interesting target elsewhere (Johnson, 1995). This might suggest that the saccadic system in young infants is not driven solely by exogenous information; rather, by 4 months of age infants may be able to use endogenous information (such as memory and prediction) to inhibit an automatic response in favor of a more interesting stimulus. Csibra, Tucker, and Johnson

(1998) studied event-related potentials in 6-month-old infants presented with a version of the gap/overlap task and found left frontal positivity suggestive of prefrontal involvement in saccadic eye movement. However, the authors also found that in 6-month-olds there was a marked absence of the pre-saccadic event-related potential (ERP) components seen in adults. Although cortical involvement in the oculomotor behavior of infants may be rudimentary, elementary functions of both the saccadic and pursuit systems are present.

Many of the significant changes in saccadic response time (Fukushima, Hotta, & Fukushima, 2000; Munoz, Broughton, Goldring, & Armstrong, 1998) and visual tracking (Richards, 2001) that occur over the course of development may be related to the maturation of attention (see Colombo, 2001 for review). Specifically, the endogenous system has been shown to have a slower developmental time course than attentional mechanisms such as basic alertness, attention to object features, and exogenously-driven spatial orienting (Colombo, 2001). Sustained attention, which requires inhibition of reflexive orienting, is endogenous in nature and can be studied in relatively young infants. Over the first few months of life (Richards, 1989), and even well into the third year (Ruff & Lawson, 1990), significant changes are seen in the duration and frequency of sustained attention.

In summary, while attentional systems related to oculomotor function undergo prolonged development throughout childhood, basic components of oculomotor behavior, such as saccadic and smooth pursuit eye movements, are present in infancy. Impairment of these early and basic oculomotor components could thus impact later developing sensorimotor, attentional, and cognitive functions.

### Is Oculomotor Impairment an Elementary Precursor to Autistic Symptomatology?

The current evidence reviewed above is too limited for a definitive and confirmatory answer to the question raised in this final section. Conversely and more importantly, there is little compelling evidence that would indicate the integrity of basic oculomotor functions. The urgent need for more extensive and systematic studies of oculomotor behavior in autism advocated here derives from this lack of reassuring evidence of integrity in the presence of tentative results indicating abnormalities.

Given the complexity of the neural systems underlying visual search and oculomotor behavior in the normal brain, as reviewed above, autism is unlikely to

involve a singular impairment affecting the entire system of oculomotor behavior and visual search. Much more likely, some components may be impaired, others intact or even functionally above normal; some neural components in subcortex, cerebellum, or cerebral cortex may be impaired, others intact. Furthermore, many previous oculomotor studies of autism are probably confounded by executive and social task components (for example, Klin et al., 2002; Minschew et al., 1999) and thus do not unequivocally address the issue of primary defects of eye movement. This underscores the need for more direct evidence on visual search and eye movement in autism. The current lack of evidence in this regard is particularly striking as any functional domain involving visual perception and visual search relies on the efficiency of the oculomotor system. In this section, we will first discuss how oculomotor abnormalities may affect visual and attentional functions relevant to social development and will then examine potential secondary effects on language acquisition.

### Face Perception

For some domains that can be regarded subdomains of visual perception, such as face perception, the relevance of oculomotor studies is obvious. Face perception is the functional domain that has thus far attracted the highest level of scrutiny in neuroimaging studies of autism. Three studies (Hubl et al., 2003; Pierce, Müller, Ambrose, Allen, & Courchesne, 2001; Schultz et al., 2000) reported reduced activity in the ‘fusiform face area’ (FFA) in autism during face perception. However, even if reduced FFA activity during face perception in autism were a consistent finding (contrary to some more recent evidence; Hadjikhani et al., 2004; Pierce, Haist, Sedaghat, & Courchesne, 2004), it would remain unclear whether these findings would be in any way unexpected. Avoidance of eye contact is a prominent feature of autism (Baird et al., 2000). More specifically, children with autism look at faces much less frequently than typically developing peers and when they look at faces they tend not to focus on parts containing core features, such as the eyes, that typically developing children preferentially look at (Klin et al., 2002; Pelphrey et al., 2002). Domain-specific stimulation is therefore strongly reduced in autism. Experience and activity strongly influence the neurofunctional organization of cerebral cortex during development, as demonstrated in animal (e.g., Sur & Leamey, 2001) and human studies (e.g., Elbert, Pantev, Wienbruch, Rockstroh, & Taub, 1995; e.g., Sadato, Okada, Honda, & Yonekura, 2002). Therefore, any reduction in FFA

involvement during face perception in autism is most parsimoniously explained as a reflection of normal experience-based neurofunctional plasticity. This highlights the need for caution in interpreting atypical regional activation patterns in autism as reflections of localized brain defects. However, the true underlying issue remains unresolved: Which elementary early-onset neurofunctional abnormalities may actually explain reduced face perception experience in autism. The oculomotor system is one potential candidate, i.e., early-onset eye movement abnormalities may affect the emergence of face processing abilities in autism. As we have previously argued in detail (Müller, 2005), explanatory models of complex processing impairments (affecting, for example, face perception) are most promising when taking account of sensorimotor component functions. From this perspective, examination of the oculomotor system is a prerequisite for theories of specific face perception impairments in autism.

### Joint Attention

While for domains such as face processing the need for eye tracking evidence is straightforward, this may be much less the case for complex domains that are not primarily visual, such as language. However, it is known that one of the crucial precursors and predictors of language acquisition is joint attention (Markus, Mundy, Morales, Delgado, & Yale, 2000), which again underscores the importance of oculomotor behavior.

The ability to participate in joint attention is a pivotal accomplishment of early childhood. Joint attention refers to the triadic relationship between two or more individuals sharing an experience concerning a third party, object or event. For example, child (A) is looking at a dog. Child (B) arrives and child (A) verbally engages child (B) by looking at child (B) sharing eye contact and then looking and possibly pointing to the dog. A verbal tag such as “look it’s a dog” may be attached to coincide with the pointing. The interaction between child (A) and (B) described above serves the purpose of establishing common ground between two or more individuals who are overtly aware that they are both focused on the same entity or event.

Joint attention supports learning through imitation, social sharing of experiences, and word-object mapping (Mundy, Sigman, Ungerer, & Sherman, 1986). In a longitudinal study of a crucial component function of joint attention, Brooks and Meltzoff (2005) recently reported that gaze following at age 9–11 months was significantly correlated with word acquisition and phrase comprehension later in life (at ages

14–18 months). Joint attention also mediates understanding of others as intentional agents who have valuable information and provide emotional cues with their facial expressions and pragmatic information in their speech about a shared object, event or third party (Tomasello & Kruger, 1992). Research comparing autistic, developmentally delayed, and typically developing children has illuminated a failure to share joint focus of attention as a specific feature of autism (Bacon, Fein, Morris, Waterhouse, & Allen, 1998; Dawson, Meltzoff, Osterling, Rinaldi, & Brown, 1998; Mundy et al., 1986; Sigman, Kasari, Kwon, & Yirmiya, 1992). This may be in part explained by the absence of gaze cueing effects in children with ASD (Johnson et al., 2005). It has been established that improvements in joint attention significantly affect other areas of functioning, including the critical areas of language use and social relationships. There is evidence that language outcomes and successful independent living rely on the levels of joint attention exhibited by children with autism (Charman, 2003). A longitudinal study (Howlin, Goode, Hutton, & Rutter, 2004) found that good outcomes in adulthood as assessed by the ability to live independently, maintain employment, gain friendships, and obtain formal education were associated with the individuals having had some speech at age five. Thus the ability of children to engage in joint attention and thereby learn language affects later outcomes.

Children with autism do not exhibit typical patterns of joint attention bids or responses (Carpenter, Pennington, & Rogers, 2002), failing to jointly attend to social stimuli in the same manner as their peers. For example, children with autism do not show a typical change in orienting (Dawson et al., 1998) even when their peers are in distress (Dawson et al., 2004). Indeed, children with autism fail to orient to most social stimuli (Dawson et al., 1998). They are impaired in their own gestural communication, making use of imperative pointing but not protodeclarative pointing (Camaioni, Perucchini, Muratori, Parrini, & Cesari, 2003). Further, individuals with autism show impoverished imitation of another's actions (Williams, Whiten, & Singh, 2004). Despite normal memory for the actions to be imitated, individuals with autism show impairments in imitating facial expressions and limb movements (Carpenter et al., 2002; Rogers, Bennetto, McEvoy, & Pennington, 1996; Rogers, Hepburn, Stackhouse, & Wehner, 2003).

There is evidence that developmental sequences related to joint attention are abnormal in autism. Carpenter, Nagell, and Tomasello (1998) found that typically developing children follow a sequence of

learning that adheres to the following pattern: *Joint engagement*  $\Rightarrow$  *Communicative gestures*  $\Rightarrow$  *Attention following*  $\Rightarrow$  *Imitative learning*  $\Rightarrow$  *Referential language*. By contrast, children with ASD follow a distinctly different pattern: *Imitative learning*  $\Rightarrow$  *Referential language*  $\Rightarrow$  *Joint engagement*  $\Rightarrow$  *Attention following*  $\Rightarrow$  *Communicative gestures*.

The disrupted pattern of development seen in autism would be consistent with inappropriate or delayed word-object mapping and a failure to link people's verbalizations to specific situations. Whereas it is established that joint attention plays a crucial role in autistic language delays (Charman, 2003), much less is known about the causes of joint attention impairment in autism. Oculomotor anomalies remain an obvious candidate for examination. Potential oculomotor defects are likely to affect an autistic child's ability to engage in joint attention. For example, abnormal saccade generation will make it harder or impossible for an autistic child to follow the mother's gaze in what would normally be a triadic joint attention setting. Such defects could thus be one of probably several elementary sensorimotor impairments setting off a cascade of disturbances in later developing systems, indirectly affecting social cognition, language acquisition, and other domains.

Abnormalities in social cognition, such as joint attention, have been cited as indications of failed interregional specialization within a social brain network (Johnson et al., 2005), in the context of findings on two face-sensitive ERP components. These components (N170 and N290) are modulated by eye gaze direction in children with autism at the age of 2–5 years, indicating incomplete functional differentiation between gaze and face processing. In typically developing children such modulation is also seen, but much earlier in life (around age 4 months), whereas it is absent in older children and in adults. Johnson et al. (2005) interpret these findings as a reflection of reduced specialization within a 'social brain network' in autism. However, this approach does not directly address the question of how an oculomotor system that is over-specialized for certain tasks and under-specialized for others early in life might affect later development in domains such as joint attention. The studies reviewed here suggest more fundamental abnormalities of eye movement and visual attention, regardless of their relevance for social cognition.

#### Language Acquisition

The discussion of joint attention underscores that interactive mechanisms of language acquisition are

likely to be compromised in autism (Baltaxe & Simmons, 1975). The child's ability to take advantage of experiential settings in language learning is part of what our group has described as *language ingredient functions* (Müller, 2005). These are neurocognitive functions that emerge before the onset or during the course of language acquisition and that are prerequisites for normal language development. Among the candidates for such ingredient functions are visuomotor and audiomotor integration, motor planning and preparation, imitation and action understanding, joint attention, as well as working memory and response inhibition. While none of these functions are fully developed at the outset of typical language acquisition, the presence of basic abilities in each of these domains contributes to the child's ability to learn words and communicate in phrases. The strongest evidence for convergence of these ingredient functions in language acquisition comes from neurobehavioral and neuroimaging studies discussed below. Based on this model, language delay in autism can be explained ontogenetically with reference to impairments of these more elementary ingredient functions (see Müller & Basho, 2004 for detailed discussion). Besides the evidence of joint attention serving such a function (as discussed above), there is overall consensus that imitation is delayed in autism (Williams et al., 2004). This may reflect more general delays in sensorimotor integration. Imitation deficits have been shown correlate with impaired social cooperation in young children with autism (Rogers et al., 2003).

The concept of language ingredients is supported by neuroimaging evidence for Broca's area, which has long been considered a "language area". However, left inferior frontal cortex participates in a multitude of apparently nonlinguistic functions. For instance, left inferior frontal activation has been found for imitation (Buccino, Binkofski, & Riggio, 2004; Iacoboni et al., 1999), action imagery (Binkofski et al., 2000) and observation (Buccino et al., 2001), motor preparation (Krams, Rushworth, Deiber, Frackowiak, & Passingham, 1998), complex motor planning (Fincham, Carter, van Veen, Stenger, & Anderson, 2002), rule shifting (Konishi et al., 1998), response selection (Thompson-Schill, D'Esposito, Aguirre, & Farrah, 1997), as well as response conflict and inhibition (Kemmons, Villalobos, Gaffrey, Courchesne, & Müller 2005; Miller & D'Esposito, 2005; Rubia et al., 2001). Adult cognitive neuroscience has few answers for this surprising overlap of functional specializations in Broca's area. From a developmental perspective, such functional overlap is much less surprising. The issue is not so much why in adults all those non-linguistic functions *also* involve

inferior frontal cortex, but rather how those apparently nonlinguistic functions serve as language ingredients during development. From a developmental perspective, it is reasonable to hypothesize that left inferior frontal cortex becomes crucially involved in language learning from the second year onwards because a number of functional pathways providing vital components for language learning converge in this brain area (Müller & Basho, 2004).

Although Broca's area is the currently best-studied neural substrate subserving convergent language ingredient functions, it is probably not directly involved in eye movement and visual search. Our discussions above, however, highlight how suspected oculomotor abnormalities in autism may affect the development of at least some of the mentioned ingredient functions, such as joint attention and imitation, and thus have indirect negative impact on language acquisition and cognitive development.

The question remains why conditions such as congenital blindness, which may affect some proposed ingredient functions directly by disrupting sensory components, do not result in absence of language acquisition or the emergence of autistic symptomatology. It has been reported that blindness is indeed associated with a much higher prevalence of autism than seen in the general population (Ek, Fernell, & Jacobson, 2005). However, in developmental disorders affecting only a single sensory modality or a localized part of the brain, ingredient functions are expected to benefit from compensatory effects of developmental plasticity (Elbert, Heim, & Rockstroh, 2001; Kolb & Gibb, 2001). For example, the effects of absent visual input on compensatory plasticity in congenital blindness have been demonstrated by Sadato et al. (2002). Compensatory plasticity appears to be much less effective in the autistic brain, most likely due to diffuse or distributed regional involvement (as discussed in Müller & Courchesne, 2000).

## Conclusion

The general context of our review on eye movement and visual search relates to the developmental logic of a disorder such as autism. The field of autism research has in the past decade been heavily influenced by findings from neuroimaging and other neuroscientific research in adult patients, often for technical reasons. This carries the risk of a "lesion-deficit" perspective that is common in adult neuropsychology (Thomas & Karmiloff-Smith, 2002). However in autism research, it is not sufficient to map out areas involved in complex

task conditions (e.g., those considered to reflect ‘theory-of-mind’) and interpret abnormality in the adult autistic individual as an “explanation” of the impairment (Müller, 2005). In order to achieve a truly explanatory model of autism, priority should be given to the study of developmental precursors of more complex and relatively late emerging functions, such as language or social cognition.

Our approach sketched in the previous sections aims at such a developmental account of sociocommunicative impairment in autism. It is currently supported by evidence that is robust for some ingredient functions, such as joint attention, but no more than suggestive for others, such as visuomotor coordination, imitation, and the role of the mirror neuron system. Our review serves to establish that neural systems of eye movement and visual search are further obvious candidates for language ingredients and therefore potentially crucial elements of an explanatory model of language delay and cognitive impairment in ASD. Specifically, oculomotor defects in autism could be among the most basic elements in a developmental chain of events, causally preceding impairments in other language ingredient functions, such as imitation, joint attention, or complex motor planning. A comprehensive theory of socio-communicative impairment in autism therefore has to take into account the potential role of an atypical or defective functional brain organization for eye movement and visual search.

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