



# Eye tracking studies of normative and atypical development ☆

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## Abstract

This paper reviews the use of eye tracking measures (saccades, smooth-pursuit eye movements, fixations during scene and face perception, and pupillary dilation) to study typical and clinical populations of children and adolescents and evaluates the use of these measures. The studies are evaluated with a focus on points that may be of general interest to developmentalists (the contribution of contextual and temporal factors in performance, methods of analyzing age-related differences, and the role of the psychometric properties of the tests in interpretation of differences across age and clinical groups). Some limitations of eye tracking are pointed out (e.g., the nature of the relation between oculomotor and other motor systems, constraints in making inferences about the brain from psychophysiological data). Finally, the potential of eye tracking measures for probing normative and abnormal development is explored.

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The eyes have been a window to the mind in typical and clinical populations for more than a century. We take in the world through our eyes, and almost everything we do during the time we are awake involves eye movements. Furthermore, although

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we are not aware of it, our pupils dilate rapidly all the time in response to a large array of cognitive and emotional stimuli, whether internally or externally generated. Thus, our eyes reveal a great deal about what we are thinking and feeling, and eye tracking measures can harness this potential to improve our understanding of the mind and its development.

Eye tracking provides a non-invasive method for elucidating a wide variety of cognitive processes, from visual-spatial attention to object perception, memory, and language. Eye tracking measures can also be helpful in examining socio-emotional processes, such as motivation, response to different types of rewards, and aspects of social information processing. The basic characteristics of eye tracking measures are well delineated, which allows researchers to make stronger inferences about specific cognitive and emotional processes from eye tracking tasks. Eye tracking also enables measurement of variables that are difficult to obtain through other methods, such as direct assessment of where individuals are looking at in static or dynamic visual stimuli and momentary recruitment of cognitive resources in accordance with task demands. Because the neural substrates of eye movements are fairly well established, eye tracking has also been used to make inferences about how the brain processes information. Although there is a very large and sound body of research on eye tracking in adults and non-human primates, this research has so far been vastly underutilized in research with children and adolescents. This article will provide an overview of eye tracking studies in healthy and clinical populations of children and adolescents, with the hope that these measures can be added to the toolkit of developmentalists as they seek to understand cognitive and social processes and their neural substrates in typical and atypical development.

In the first section, I will briefly review key eye tracking measures (saccades, smooth-pursuit eye movements, eye movements during face and scene perception, and task-specific pupillary dilations) to provide some basic background information for the rest of the paper. The next two sections will summarize studies in which these measures have been used in typical and several atypical populations of children and adolescents. In the final section, I will evaluate studies that have used eye tracking as a research tool, emphasizing points that may be of general interest to developmentalists, list limitations of eye tracking as a tool, and end with its potentials for addressing developmental questions.

The goal of the paper is not to review how eye tracking measures have contributed to understanding of specific developmental or clinical phenomena or models and theories of normative and atypical development (which would require placing these studies in the context of a broader body of research that does not involve eye tracking or children), but to provide a sense of why and how these measures have been used in typical and atypical populations of children and adolescents and to appraise these measures as tools for probing development.

Eye movements in children have been reviewed by Hainline (1988), so this paper will focus on research conducted since 1988. I will also limit the review to studies of participants from 4 to 5 years of age through adolescence. For reviews of eye tracking in infancy, the reader is referred to Haith (2004), Simion and Butterworth (1998), and Von Hofsten (2004). To limit the scope of the manuscript, I will not cover research on eye movements that have been recorded during reading (for reviews, see Liversedge & Findlay, 2000; Rayner, 1998; Starr & Rayner, 2001) or in learning disabilities.

## Eye tracking measures

The first eye tracking devices were built in the late 1800s and early 1900s. These devices required attaching objects to the eye ball to track its movements, and it is hard to imagine the experience as being pleasant for the participants. Huey (1898), for example, required participants to “bite into a mass of partially cooled sealing-wax attached to a mouth-piece fastened in the head frame” (p. 584) to keep their heads still, and Delabarre (1898) anesthetized the eyeball “by applying two or three drops of a two to three per cent solution of cocaine” (p. 572). These early devices were used in experimental investigations of psychological phenomena, such as visual illusions, as well as reading. Diefendorf and Dodge (1908) were among the first to apply eye tracking to the study of eye movements in schizophrenia. Electro-oculography was developed in the 1920s (Schott, 1922, cited in Young & Sheena, 1975). In the 1930s and 1940s, corneal reflection methods were used to study eye movements of healthy individuals to pictures, including visual art and advertisements (Brandt, 1945; Buswell, 1935). Brandt (1945) also studied eye movements of good and poor students as they were solving problems (e.g., in algebra, geometry, geography) and made suggestions to teachers based on his observations. As noted by Rayner (1978) in his review of eye movements in reading, eye tracking studies during this period had a strong applied emphasis. In the 1950s, a more accurate method of recording eye movements was developed that involved attaching an eye monitoring device to a contact lens placed over the eye (Young & Sheena, 1975). Yarbus (1965/1967) used this method to undertake an intensive investigation of eye movements, especially eye movements to pictures. In the 1950s and 1960s, eye movements and pupillary dilation were used to study a host of cognitive processes in adults and children in the context of the Cognitive Revolution (e.g., Mackworth & Bruner, 1970). As noted earlier, Hainline (1988) provides a review of eye movements in children from these decades. Many of the relatively standardized eye movement paradigms used today in healthy and clinical populations began to be developed in the 1970s and 1980s (e.g., Hallett & Adams, 1980; Holzman, Proctor, & Hughes, 1972). Interest in the neurobiological bases of saccades increased with the demonstration that patients with frontal lesions had difficulty making saccades away from visual targets (Guitton, Buchtel, & Douglas, 1985).

An extremely thorough review of the history of pupillometry research is provided by Loewenfeld (1993). Other reviews of this area focus to a greater extent on cognitive bases of pupillary dilations in healthy adults (Beatty, 1982) and in adults with schizophrenia (Steinhauer & Hakerem, 1992).

Today, eye tracking with children and adolescents is conducted with non-invasive video-based eye monitors, with automated recording of eye movements (see Fig. 1).

This section will provide a brief overview of basic eye movement measures. Table 1 provides a summary of common eye tracking tasks, typical measures used with these tasks, and what these measures assess. Although some technical details are included in this section, the discussion is geared primarily toward readers who are not familiar with eye tracking methods.

### *Saccades*

Saccades are ballistic eye movements aimed at bringing objects into foveal vision. Thus, a saccade to an object normally coincides with an overt shift of visual-spatial attention to

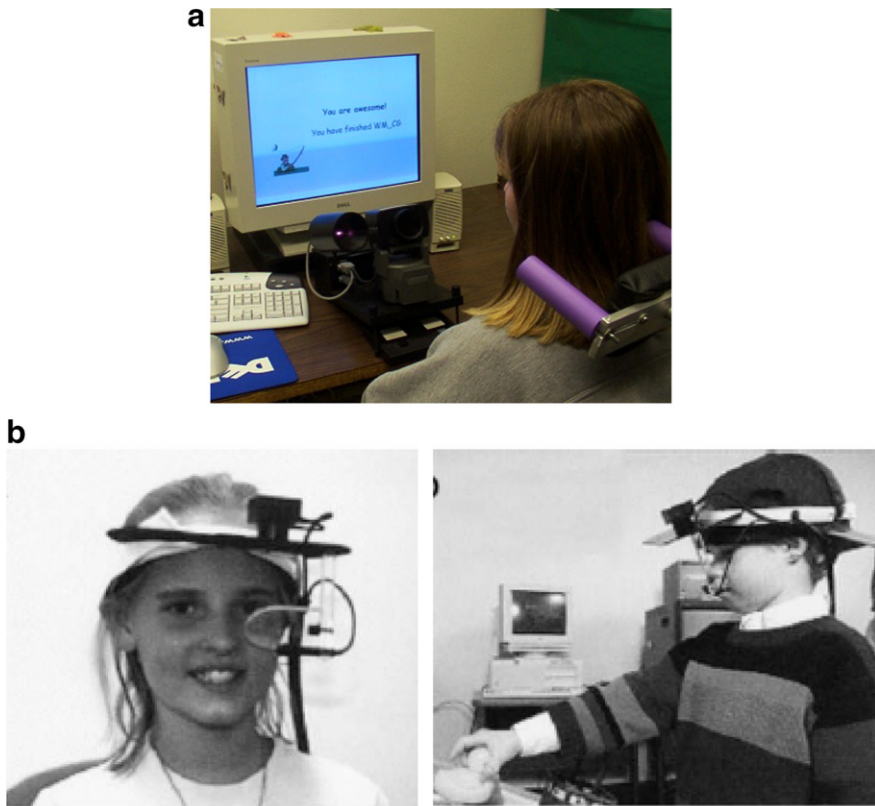


Fig. 1. (a) Remote tabletop and (b) head-mounted eye monitors (Trueswell et al. (1999), reprinted with permission from Elsevier).

that object. Saccades can be divided roughly into externally versus internally guided saccades. Externally guided saccades are often assessed through visually guided saccade tasks (labeled *prosaccades* in the rest of this paper), where participants are instructed to look at a visual stimulus as soon as it appears.

In some studies reviewed below, saccade tasks included conditions in which there was a 200-ms gap or overlap between a fixation point and the target stimulus. The reduction of saccadic RTs in the gap condition compared to those in a typical condition with no gap (sometimes called the “null” condition) is termed the gap effect. It has been hypothesized that the reduced saccadic RTs typically observed in the gap task reflect early disengagement of attention from fixation and non-specific response preparation processes due to the warning provided by the offset of fixation, whereas elevated RTs reflect the longer period of time needed to disengage attention from fixation (e.g., Fischer & Weber, 1993; Kingstone & Klein, 1993; Pratt, Bekkering, & Leung, 2000; Spantekow, Krappmann, Everling, & Flohr, 1999).

Internally guided saccades are executed in the absence of a visual stimulus. Saccades made to a location opposite from a visual stimulus (antisaccades) or to the predicted (predictive saccades) or remembered location of a visual stimulus (memory-guided saccades) fall under this category.

Table 1  
Summary of common eye tracking tasks and measures

Task	Common measures	What is measured?
All saccade tasks	Duration, peak velocity & amplitude of the saccade	Basic dynamics of saccades
	Gain (saccade amplitude/target amplitude)	Spatial accuracy of the saccade (hypometric saccades undershoot the target, whereas hypermetric saccades overshoot the target)
	Latency to initiate the saccade (RT)	Speed of processing and movements of visual–spatial attention
	Variability of saccadic RTs	Variability of speed of processing and movements of visual–spatial attention
	Frequency of express saccades (saccades with very short RTs, i.e., between 80 and 130 ms)	Disengagement of visual–spatial attention
	Corrective saccades (saccades to the correct location after an initial error)	Ability to monitor performance, perceive and correct errors
	Premature saccades (saccades prior to target onset despite instructions to fixate)	Ability to inhibit disallowed saccades
Gap & overlap tasks	Gap effect (reduction in average saccadic RT when there is a temporal gap between fixation and target)	Disengagement of visual–spatial attention & non-specific response preparation
	Increase in average saccadic RT when there is a temporal overlap between fixation and target	Engagement of visual–spatial attention at fixation
Antisaccade task	Accuracy (whether the saccade was in the correct direction)	Ability to inhibit a saccade to the disallowed location
Memory-guided saccade task	Spatial accuracy of the saccades (distance error)	Accuracy of visual–spatial working memory
Predictive saccade task	Frequency of predictive saccades for targets whose location and/or timing is predictable	Ability to form an internal representation of the target and to predict its occurrence
Active fixation tasks	Intrusive saccades during fixation	Ability to maintain fixation, sustained attention
Pursuit	Root-mean square error (the difference between target and gaze position during pursuit)	Overall efficiency of the pursuit system and its interaction with the saccadic system
	Position gain (gaze position/target position)	Overall efficiency of the pursuit system and its interaction with the saccadic system
	Velocity gain (gaze velocity/target velocity)	Efficiency of pursuit, independent of catch-up and intrusive saccades
	Compensatory saccades (to catch up with the target)	Inefficiency in pursuit, compensated for by the saccadic system
	Intrusive saccades (saccades that anticipate the target's location and "square wave jerks")	Disruption of pursuit by the saccadic system due to failure to inhibit saccades
	Initiation phase (pursuit during the first 100–120 ms)	Visually guided, dependent on bottom-up information by onset of target movement
	Maintenance phase (pursuit after the first 100–120 ms)	Internally guided, dependent on top-down information about target velocity

(continued on next page)

Table 1 (continued)

Task	Common measures	What is measured?
Scene/face perception tasks	Location and sequencing of fixations	Allocation of visual–spatial attention across the scene/face
	Duration of fixations	Duration of processing foveal and parafoveal information and deciding where to look next
	Distance between fixations	Breadth of visual–spatial attention
Pupillary dilation tasks	Peak pupillary dilation	Allocation of resources
	Latency to peak pupillary dilation	Speed of processing

Note. RT, response time.

The neurobiological bases of prosaccades are depicted in Fig. 2. Internally guided saccades are mediated by additional regions (for reviews, see Carpenter, 1988; Everling & Fischer, 1998; Hikosaka, Takikawa, & Kawagoe, 2000; Leigh & Kennard, 2004; Leigh & Zee, 1999; Pierrot-Deseilligny, Gaymard, Müri, & Rivaud, 1997).

The main measures extracted from saccadic eye movements include duration, peak velocity, amplitude, gain (saccade amplitude/stimulus amplitude), and latency to initiate the saccade. Once initiated, saccades take 30–120 ms to complete for amplitudes up to 45° (Alik, Toom, & Luuk, 2003; Carpenter, 1988; Morrone, Ross, & Burr, 2005). The peak velocity of prosaccades ranges from 200 to 400°/s, depending on amplitude (Smit, van Gisbergen, & Cools, 1987; Van Gelder, Lebedev, & Tsui, 1997). Prosaccades are initiated approximately 180–250 ms after stimulus onset, depending on the task.

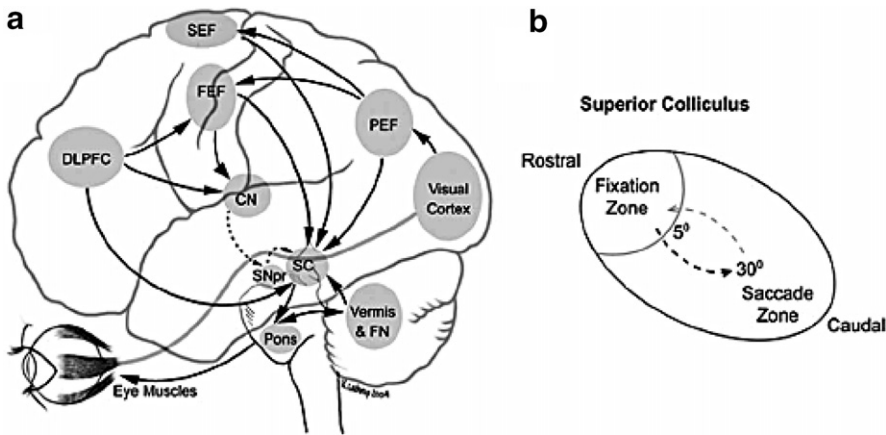


Fig. 2. Lateral view of human cerebral cortex and projections to superior colliculus (SC) involved in saccade triggering. (a) Cortical and subcortical areas involved in oculomotor control, with excitatory and inhibitory pathways depicted in solid and broken lines, respectively. Direct excitatory pathways to the SC shown are from the dorsolateral prefrontal cortex (DLPFC), frontal eye fields (FEF), parietal eye fields (PEF), and supplementary eye fields (SEF). Indirect cortical input from the DLPFC and FEF are through the caudate nucleus (CN), which inhibits the substantia nigra pars reticulata (SNpr) and which, in turn, inhibits the SC. Cerebellar connections and pontine connections to the SC are also shown. (b) Retinotopic map of left SC depicting fixation neurons in rostral portions and saccade neurons in caudal portions. Directional coding is shown with upward direction in superior and downward direction in inferior regions. Figure and figure caption reprinted from Reilly et al. (2005), with permission from the Society of Biological Psychiatry.

*Pursuit*

To track small objects that move relatively slowly and smoothly, we use smooth-pursuit eye movements (Fukushima, 2003). These are smooth, non-ballistic movements that match gaze velocity to target velocity and keep the object within foveal vision. The neural substrates of smooth-pursuit eye movements are displayed in Fig. 3 (for reviews, see Fukushima, 2003; Krauzlis, 2005; Krauzlis & Stone, 1999; Thier & Ilg, 2005). Although the pursuit and saccade systems have separable neural substrates, they nevertheless work in an

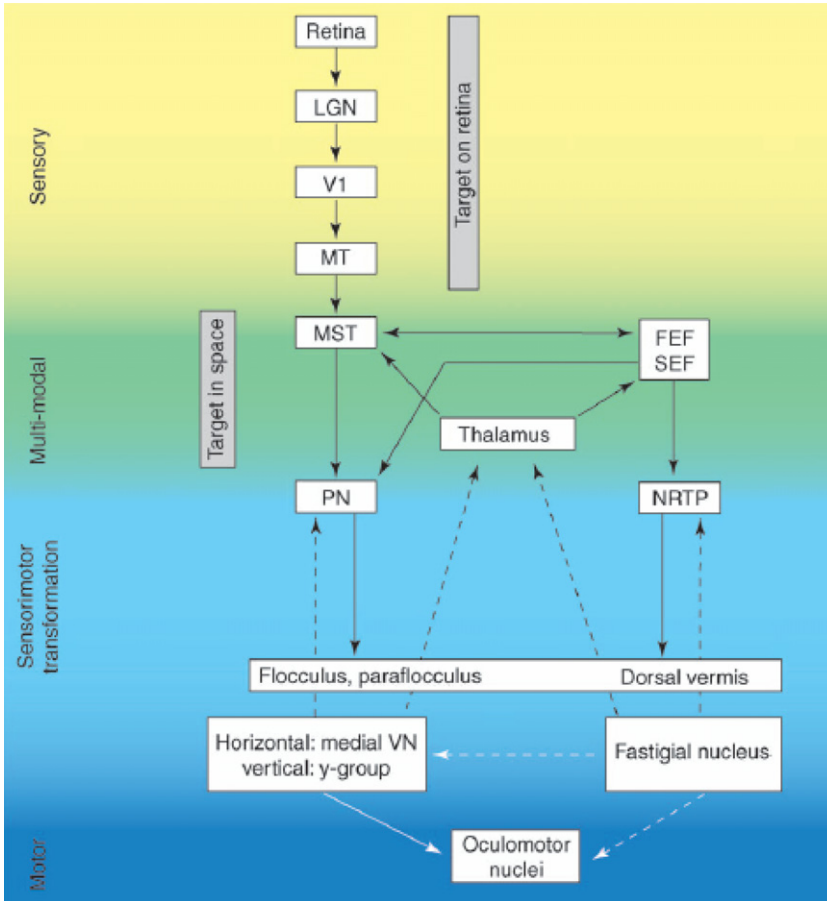


Fig. 3. The major substrates of smooth-pursuit eye movements and their connections. Broken lines indicate connections that are still hypothetical or have not been elucidated in sufficient detail. The scheme considers observations, not discussed in the main text, that suggest that signals for horizontal and vertical smooth-pursuit are dealt with by different parts of the vestibular complex: namely, horizontal smooth-pursuit by medial vestibular nuclei; and vertical smooth-pursuit by the y-group—a small cell group that caps the inferior cerebellar peduncle and that, similar to vestibular complex neurons, receives primary vestibular afferents. *Abbreviations:* FEF, frontal eye field; LGN, lateral geniculate nucleus; MST, middle superior temporal; MT, middle temporal; NRTP, nucleus reticularis tegmenti pontis; PN, pontine nuclei; SEF, supplementary eye field; V1, primary visual cortex; VN, vestibular nuclei. Figure and figure caption reprinted from Thier and Ilg (2005), with permission from Elsevier.

integrated fashion in tracking moving objects (e.g., Fukushima, 2003; Krauzlis & Stone, 1999; Liston & Krauzlis, 2003; Missal & Keller, 2002). The two systems rely on overlapping neural substrates, and are affected similarly by a variety of experimental manipulations and lesions. When the pursuit system is unable to foveate the object continuously, saccades are used to catch up with the moving object or to anticipate its location.

Smooth-pursuit eye movements are usually assessed by instructing participants to visually track a small stimulus that moves at a relatively slow and predictable velocity along a horizontal path. There is still controversy regarding the most appropriate measures to assess the integrity of the pursuit system (Hutton & Kennard, 1998). Nevertheless, the main quantitative measure used in most recent studies is gain [defined as peak (or mean) gaze velocity divided by peak (or mean) target velocity]. Low gain scores suggest difficulty in matching gaze to target velocity, indicating inefficiency in the functioning of the pursuit system. Performance can also be assessed by root-mean square error (RMSE; Clementz, Iacono, & Grove, 1996), which is based on the difference between gaze and target position throughout tracking. RMSE is calculated by taking the square of the difference between target and gaze positions at each artifact-free point during pursuit, averaging the squares, and then taking the square root of this average.

Additional measures of performance include compensatory and intrusive saccades during tracking (Hutton & Kennard, 1998; Ross, Hommer, Radant, Roath, & Freedman, 1996). A high frequency of compensatory saccades (to catch up with the target) indicates difficulty in matching gaze to target velocity, and suggests that an impairment or immaturity in the pursuit system is being compensated for by the saccadic system. In contrast, intrusive saccades (anticipatory saccades or pairs of saccades that move the gaze away from the target and back, i.e., “square wave jerks”) may reflect disruption of pursuit by the saccadic system because of a failure to inhibit the saccadic system appropriately during pursuit.

Pursuit performance can also be divided into initiation and maintenance phases (e.g., Avila, Hong, Moates, Turano, & Thaker, 2006; Fukushima, 2003; Thier & Ilg, 2005). Initiation of pursuit (“open-loop” pursuit) occurs during the first 100–120 ms and is hypothesized to depend on bottom-up information provided by the onset of the target’s movement (“retinal motion signal”). In contrast, maintenance of pursuit (“closed-loop” pursuit) once the system locks on to the target is thought to rely on top-down guidance of the pursuit system based on an internal representation of the target velocity.

### *Eye movements during sceneface perception*

Whereas simple paradigms requiring participants to look at a few stimuli are informative regarding the cognitive and neural correlates of saccades, eye movements during scene or face perception are more useful for examining information processing in more naturalistic contexts. We normally make 3–4 saccades a second and pause in between (fixate) for 300–400 ms at a time, to take in the information at the fovea and to decide where to fixate next. As demonstrated decades ago by Buswell (1935) and Yarbus (1965/1967), people look at informative regions when shown a picture of a scene or of a face. Furthermore, when given more time to look at the picture, they return again and again to these informative regions rather than covering the whole area of the picture (e.g., see Fig. 4). It is especially under these conditions that it becomes obvious that eye movements do not reflect a passive type of perception but represent active, goal-directed movements. Reviews



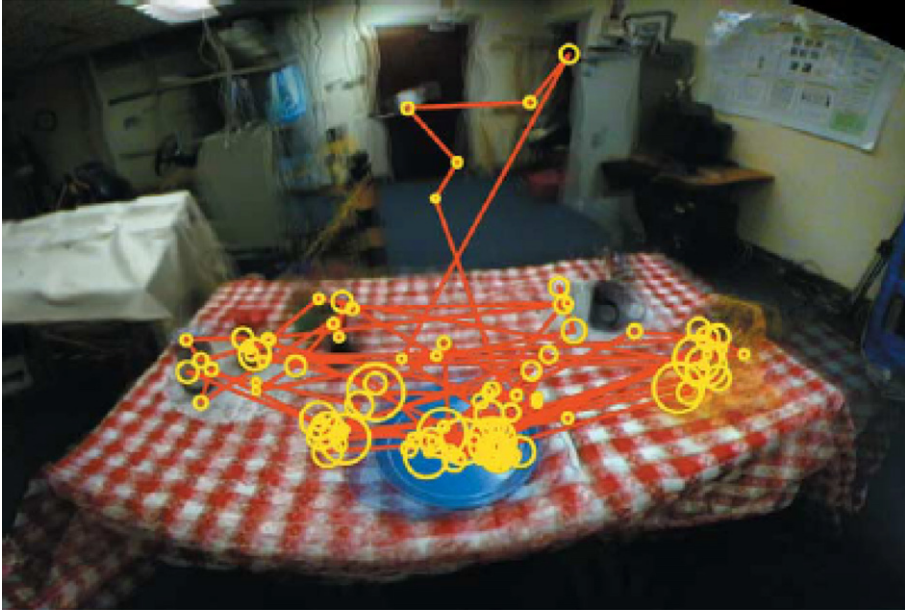


Fig. 4. Fixations made by an observer while making a peanut butter and jelly sandwich. Images were taken from a camera mounted on the head, and a composite image mosaic was formed by integrating over different head positions using a method described in Rothkopf and Pelz (2004) (The reconstructed panorama shows artifacts due to the incomplete imaging model that does not take the translational motion of the subject into account.) Fixations are shown as yellow circles, with diameter proportional to fixation duration. Red lines indicate the saccades. Note that almost all fixations fall on task-relevant objects. Figure and figure caption reprinted from Hayhoe and Ballard (2005), with permission from Elsevier.

of recent research on eye tracking during scene perception can be found in Henderson (2003), Hayhoe and Ballard (2005), Henderson and Hollingworth (1999), and Land and Furneaux (1997).

The main measures used in scene/face perception paradigms include the location, duration, and sequencing of fixations, and distance between fixations. The location and sequencing of fixations are used to infer what individuals are attending to and in which order. The duration of fixations is used as a measure of speed of processing foveal and extrafoveal information, and distance between fixations is used to estimate the width of the attentional spotlight. In general, fixation duration increases and saccadic amplitude decreases as task difficulty and the need to gather more fine-grained information increases.

### *Pupillary dilation*

The main factor that regulates pupillary diameter is amount of light. However, pupillary diameter also varies as a function of task-specific recruitment of cognitive resources. Researchers who use the construct of resources to account for developmental changes in cognition argue that older children either have more resources or use their resources more efficiently than younger children (for reviews, see Bayliss, Jarrold, Baddeley, Gunn, & Leigh, 2005; Cowan et al., 2005). However, a critical shortcoming of these accounts has

to do with the hypothetical nature of the construct of resources (Meyer & Kieras, 1997). When the amount and allocation of resources are not measured directly and inferred from behavioral data, explanations run the risk of circularity. Therefore, measures of task-specific pupillary dilation can be invaluable in grounding resource theories in actual psychophysiological data.

The relation between pupillary diameter and task-specific pupillary dilation has been likened to that between spontaneous electroencephalogram records and event-related potentials (ERPs; Beatty, 1982). *Tonic* changes in pupillary diameter are influenced by general factors, such as level of arousal, anxiety, and stress. Task-specific pupillary dilations are *phasic* changes in pupillary diameter time-locked to the onset of stimuli or to responses. Correlations between pupillary dilations and indices of autonomic function (e.g., heart rate, galvanic skin response) are not high, consistent with the idea that neural control of pupillary dilation lies at the intersection of the autonomic and central nervous systems (Loewenfeld, 1993). Similarly, although pupillary dilations and ERPs covary, they are not perfectly correlated, indicating that they reflect different aspects of processing (Steinhauer & Hakerem, 1992).

Pupillary dilations show a remarkable sensitivity to working memory load on tasks such as the digit span, where they increase linearly with each increase in memory load, reaching a peak just before participants repeat back the digits, and level off or decrease when the number of digits to be remembered exceeds memory span (Granholm, Morris, Sarkin, Asarnow, & Jeste, 1997; Kahneman & Beatty, 1966; Kahneman, Onuska, & Wolman, 1968; Peavler, 1974). In mental rotation tasks, pupillary dilation also increases linearly with angular disparity (Just, Carpenter, & Miyake, 2003). Phasic pupillary dilations increase with task difficulty in many other tasks (Beatty, 1982; Beatty & Lucero-Wagoner, 2000; Brown et al., 1999; Granholm, Chock, & Morris, 1998, 2000; Just et al., 2003; Minassian, Granholm, Verney, & Perry, 2004; Nuthmann & van der Meer, 2005; Schlemmer, Kulke, Kuchinke, & van der Meer, 2005; Siegle, Steinhauer, & Thase, 2004; Van der Meer, Friedrich, Nuthmann, Stelzel, & Kuchinke, 2003). Importantly, the pupil dilates even to the *absence* of expected stimuli, indicating clearly that dilations are not passive reactions to perceptual stimuli (Qiyuan, Richer, Wagoner, & Beatty, 1985).

Task-specific pupillary dilations are mediated by rapid interactions among the frontal cortex, the thalamus, and the reticular activating system, resulting in activation of sympathetic pathways and inhibition of parasympathetic pathways that terminate in the muscles controlling pupillary diameter (Beatty & Lucero-Wagoner, 2000) [The sympathetic nervous system, which prepares the organism for the “fight or flight” response (Loewy, 1990), mediates pupillary dilation and other visceral functions related to arousal and expenditure of energy. The parasympathetic nervous system supports functions related to conservation of energy, such as increased blood flow to the gastrointestinal system, as well as pupillary constriction]. The neurobiological bases of resources are also hypothesized to include interactions among the prefrontal cortex, thalamus, and mesencephalic reticular formation (Dehaene, Kerszberg, & Changeux, 1998; Heilman, Watson, Valenstein, & Goldberg, 1987; Robbins, 1997). Thus, phasic pupillary dilations constitute one of the few relatively direct measures of resource recruitment (Kahneman, 1973). In particular, they have been hypothesized to reflect cortical modulation of the reticular system and are interpreted as reflecting the momentary mental effort or recruitment of cognitive resources in accordance with task demands (reviewed in Beatty, 1982; Beatty & Lucero-Wagoner, 2000). The effect of task difficulty on pupillary dilation is hypothesized to be

mediated primarily by cortical inhibition of the parasympathetic pathway, rather than activation of the sympathetic pathway (Steinhauer, Siegle, Condray, & Pless, 2004), and increasing dilation with parametric increases in task difficulty on a digit sorting task is paralleled by increasing activation in the middle frontal gyrus, measured through functional brain imaging (Siegle, Steinhauer, Stenger, Konecky, & Carter, 2003b). The middle frontal gyrus, one of the three major gyri in the frontal lobes, lies in a front-to-back direction in between the superior and inferior gyri. It contains the dorsolateral prefrontal region, often identified as Brodmann's area 46/9.

As reviewed in Karatekin, Marcus, and Couperus (2007), there is a decrease in both resting pupillary diameter and baseline diameter on cognitive tasks from middle childhood to young adulthood. One reason for this decrease may be a decrease in central inhibition of the parasympathetic pathway. Consistent with this hypothesis, studies of the autonomic regulation of the cardiac system also show changes that indicate maturation of the autonomic nervous system during this age range. Importantly, these changes have been related to maturation of the parasympathetic branch and a gradual lessening of the influence of the sympathetic branch. However, age-related changes in task-specific pupillary dilations do not appear to be related to the absolute size of pupillary diameter. Thus, developmental changes in pupillary dilation in response to increasing demands to recruit cognitive resources may be mediated, at least in part, by the maturation of the parasympathetic branch, and/or the central inhibition of the parasympathetic branch, perhaps as a result of maturation of the middle frontal gyrus. In contrast, developmental reductions in baseline or resting pupillary diameter could be related to a developmental reduction in the dominance of the sympathetic branch.

Task-specific pupillary dilations are quantified through peak dilation and pupillary waveforms during a brief period (e.g., 1–2 s) after stimulus onset or prior to response. Depending on the task, different components of pupillary waveforms over longer periods of time (e.g., 10–20 s) can also be analyzed (e.g., Granholm & Verney, 2004; Siegle, Steinhauer, Carter, Ramel, & Thase, 2003a). Another measure is latency to peak dilation, an index of processing speed (Beatty & Lucero-Wagoner, 2000).

## **Eye tracking in children and adolescents: Normative development**

### *Saccades*

There has been a great deal of research on the development of saccades, especially pro- and antisaccades, in childhood and adolescence (summarized in Table 2). The main concern of a number of these studies has been to delineate the shape of the developmental trajectory on saccade tasks, using large samples and statistical techniques that go beyond simple correlations. In general, results indicate that the basic dynamics of prosaccades (peak velocity and duration), and the subcortical regions mediating them, are mature by age 4–6. In contrast, prosaccade RTs decrease gradually through at least adolescence, and antisaccade RTs and errors show much steeper rates of improvement during this period (see Fig. 5). However, the shape of the trajectory and the age at which performance reaches maturity differ across studies for both pro- and antisaccades, probably due to differences in sample sizes, task parameters and the method of analyzing age-related changes. These RT reductions can be attributed, in part, to the increasing efficiency in information transmission that accompanies myelination. In addition, increasing control over visual

Table 2  
Saccadic eye movement studies in normative development

Study	Age	<i>N</i>	Task <sup>a</sup>	Analysis of age-related changes	Results <sup>b</sup>
Salman et al. (2006a)	8–19	39	Prosac (100 tr.)	Linear stepwise regression	Sac RT: ↓ Gain & peak velocity: ns
Fischer et al. (1997a)	8–70	281	Pro-o (200 tr.), anti-g (200 tr.) Task order not rep	Participants classified into 10 age groups, increasing bin width with age; Omnibus ANOVAs, followed by visual inspection	Prosac RT: ↓ until 15–20, ↑ after 30 Antisac RT: steep ↓ between 9 and 15, continued to ↓ until 25, ↑ after Antisac errors: ↓ until 20, ↑ after Frequency of express sac on pro: ns
Klein (2001)	6–28	199	Pro-g/o, anti-g/o (100 tr./condition) Tasks in counterbalanced order	Participants classified into 1-year age groups ( <i>N</i> = 1–16/bin); multiple regression	Prosac RT: ↓ Antisac RT & errors: ↓ to a greater extent than prosac RTs Difference between anti and prosac RTs: ↓ Gap effect: ↓ Express sac: ns
Klein and Fischer (2005a)	Same as Klein (2001)				Express prosac & express antisac errors: ns Antisac errors & prosac RTs: ↓
Munoz et al. (1998)	5–79	168	Pro-g/o (120 tr.), anti-g/o (240 tr.) Prosac always followed by antisac	Participants classified into 11 age groups increasing bin width with age ( <i>N</i> = 8–28/bin); Omnibus Kruskal–Wallis, followed by visual inspection	Sac RTs: U-shaped curve, shortest in 18–22 5- to 8-year-olds: more sac RT variability, express sac in overlap, antisac errors, prosac hypometria; larger gap effect & difference between pro & antisac RTs Peak velocity: ns Sac duration: ns until age 60
Fukushima et al. (2000) <sup>c</sup>	4–13	99	Prosac (60 tr.)	Samples sizes ranged from 3 to 26 across ages <i>T</i> tests (uncorrected), correlations	Sac RT: plateaued by 12 Amplitude and peak velocity of sac: ns Antisac errors & RTs: ↓
	20–38	22			
	7–10, 12 Adults	59 15	Antisac (60 tr.)		

	6–8 Adults	10 11	Pro and antisac with warning (0, 300, 600 or 1000 ms; 40 tr./task) Task order not rep		Adults & children benefited similarly from warning, but children benefited less on both pro- and antisac
Malone and Iacono (2002)	11 17	674 616	Anti (20 tr; 17 tr. for 167 participants) Pro always followed by anti	Model fitting (along with heritability estimates)	Antisac errors ↓ Contributions of genetic & environmental influences similar between ages
Eenshuistra et al. (2007)	8–9 11–13 22	19 19 21	Pro- and antisac, with working memory load and gap & overlap conditions (54 tr. each)	ANOVAs	Prosac RT: ↓; Antisac errors & RTs: ↓ Younger children more affected than adults by working memory manipulation but not fixation offset manipulation
Ross et al. (1994a, 1994b)	7–15	53	Pro (≤30 s), predictive (≤30 s), fixation (≤30 s) Tasks always in this order	Participants classified into four 2-year-age groups ANOVAs, followed by post-hoc tests Regression for predictive and fixation tasks to find best-fitting curves and age of maturation	Sac RT on all tasks: ↓ linearly Premature sac on predictive task ↓ until 12 Sac during fixation: ↓ until 10
Luna et al. (2004)	8–30	245	Prosac (54 tr.), antisac (36 tr.), MGSs (1, 2, 4 or 8 s; 6 tr./delay) Tasks always in this order	Participants divided into seven age groups. First 6 groups spanned 2 years each ( $N = 20\text{--}30/\text{bin}$ ) seventh group spanned 6 years ( $N = 62$ ) Multiple regression, model fitting	Inverse regression best fit for sac RTs, antisaccade errors, distance errors of MGS Antisac errors and spatial error of initial MGS mature at 14, sac RTs at 15, distance errors of final MGSs at 19 Peak velocity and gain of prosac: ns
Kramer et al. (2005)	8–9 10–12 13–15 16–18 19–25	25 25 25 25 25	Prosac (60 tr.), antisac (120 tr.), oculomotor capture <sup>d</sup> (180 tr.) Pro- and antisac presented on a different day than capture Task order counterbalanced	ANOVA, followed up with Tukey's HSD	Antisac errors: equally high in the three younger groups, lower & similar in older groups Oculomotor capture errors: ns Prosac errors: infrequent & ns Prosac RTs: ↓ until 13–15, ns after AntisacRTs: ↓ until 16–18, ns after

(continued on next page)

Table 2 (continued)

Study	Age	N	Task <sup>a</sup>	Analysis of age-related changes	Results <sup>b</sup>
Luna et al. (2001)	8–13	11	Pro (54 tr.), anti (36 tr.)	Inverse curve fit	Antisac errors: ↓
	14–17	15	Task order not rep		Antisac RTs: ns
	18–30	10	Participants administered tasks in blocks on the day before scan, in trials alternating with fixation during scan; behavioral results recorded only on tasks administered prior to scan		Peak velocity, spatial accuracy, amplitude, & duration of antisac: ns
Scherf et al. (2006)	10–13	9	Prosac (40 tr. in scanner), MGS (5 s, 40 tr. in scanner)	ANOVA, inverse curve-fit	Prosac RTs and spatial accuracy: ns
	14–17	13	Participants administered slightly different versions of the task prior to during scan; behavioral results recorded only on tasks administered prior to scan		MGS RTs: main effect of age, but pairwise comparisons ns
	29.5 (10.6)	8			Accuracy of initial MGS: children < adolescents children = adults Accuracy of final MGS: ns Inverse curve fit: accuracy of initial MGS: ↑ Accuracy of final MGS: ↑ Sac RT: ns
Klein and Feige (2005)	7–8	12	Pro-o (100 tr.), anti-o (100 tr.)	ANOVA	Pro- and antisac RTs: ↓ (no age × task interaction)
	10–11	11	Task order counterbalanced		Antisac errors: ↓ after 10–11
	13–14	11			
	15–16	12			
	17–18	12			

*Abbreviations:* ↑, increased with age; ↓, decreased with age. Anti-g/o, antisaccade task with gap and overlap conditions. Anti-o, antisaccade task with overlap condition only. HSD, honestly significant difference. MGSs, memory-guided saccades. Not rep., not reported. Ns, not significant. Pro-g/o, prosaccade task with gap and overlap conditions. Pro-o, prosaccade task with overlap condition only. RT, response time; Sac., saccade(s) or saccadic. Tr., trials.

<sup>a</sup> The numbers in parentheses refer to task parameters (number of trials, task duration, or warning or delay period).

<sup>b</sup> All results refer to analyses of age-related differences.

<sup>c</sup> On the antisaccade task, a subgroup of the 7- to 10-year-olds and 12-year-olds who took part in the antisaccade study were compared to a subgroup of the adults. In the study on the effects of warning signals, the participants were a subgroup of the participants in Study 1.

<sup>d</sup> In the oculomotor capture task, small gray circles with the number 8 on them were presented on an imaginary circle for 1000 ms, after which all but one of the circles changed to red and the numbers changed to letters. The participants were instructed to report the orientation of the letter on the circle whose color had not changed. On most of the trials, a new red circle appeared simultaneously with the color change. This new circle did not have any informational value, but was expected to “capture” attention because of its sudden onset. On the remaining trials, the additional circle remained on screen throughout the trial. An error was counted in the initial saccade was made to the distractor.

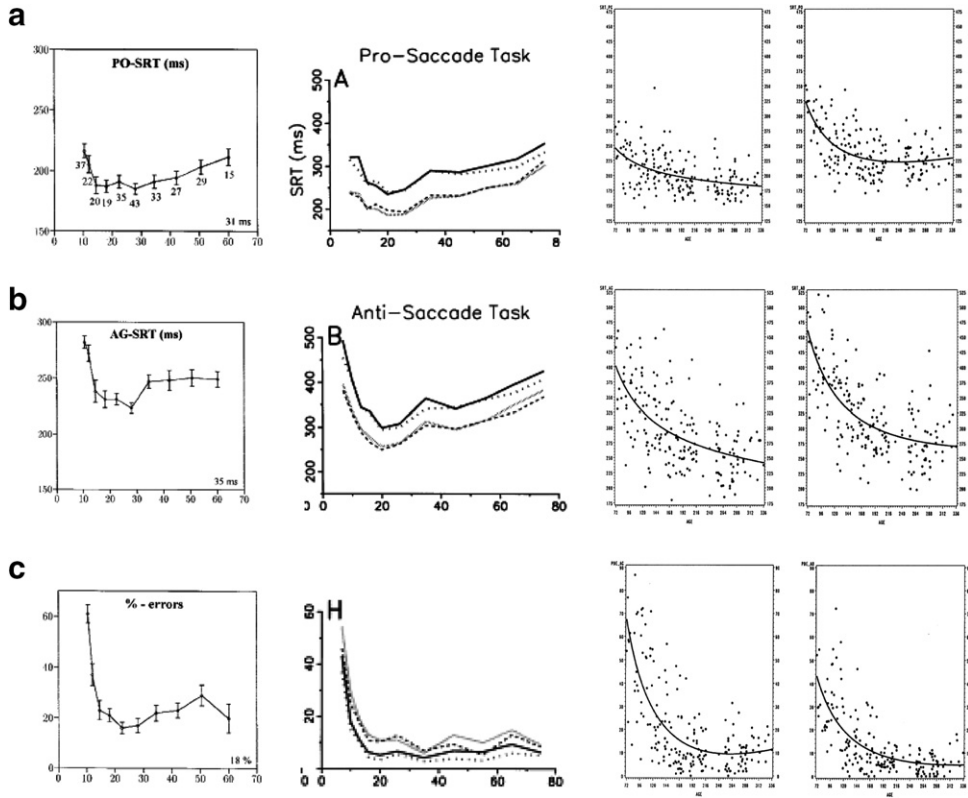


Fig. 5. Age-related changes in (a) prosaccade RTs, (b) antisaccade RTs, and (c) antisaccade errors in three studies. The first column presents data from Fischer et al. (1997a), the second from Munoz et al. (1998), and the third from Klein (2001). The figure from Fischer et al. (1997a) is reprinted with permission from Elsevier, and the figures from Munoz et al. (1998) and Klein (2001) are used with kind permission of Springer Science and Business Media.

attention contributes to increasingly faster responses to visual stimuli in both pro- and antisaccade paradigms. Finally, maturation of working memory capacity and inhibitory skills are both likely to play a role in improvements in the ability to execute antisaccades. Several studies have used saccades to make inferences about cognitive processes that go beyond the oculomotor system, and three studies have examined age-related changes in the neural substrates of saccades.

One study of saccades in children focused on prosaccades (Salman et al., 2006a). Results indicated that saccadic RTs decreased from age 8 to 19, but there were no age-related differences in gain or peak velocity. The magnitude of the decrease with age ranged from 25 to 60 ms, depending on target amplitude and direction. The authors concluded that the brainstem and cerebellar regions mediating accuracy and peak velocity of prosaccades were mature by age 8, whereas oculomotor regions involved in fast responding to visual targets (including disengagement and shifting of attention, and sensorimotor transformations) continue to mature through adolescence.

Several studies have compared the development of pro- and antisaccades in large samples. In one of these studies (Fischer, Biscaldi, & Gezeck, 1997a, 1997b), participants between 8 and 70 years of age were examined. Prosaccade RTs improved gradually with age until 15–20 and increased slightly after 30. Antisaccade RTs and errors showed steep decreases between 9 and 15, and continued to improve until age 25. A factor analysis of six eye movement variables and age yielded two factors, which were interpreted as evidence for separable automatic and voluntary components of saccade generation. Results suggested that the voluntary component (derived mostly from the antisaccade task) has a more protracted course of development than the automatic component (derived mostly from the prosaccade task).

Klein and colleagues have conducted a series of studies on pro- and antisaccades. In the first study (Klein, 2001), pro- and antisaccade tasks with gap and overlap conditions were administered to participants between ages 6 and 28. Visual inspection of the data indicated that prosaccade RTs improved gradually between 6 and 28, and that antisaccade RTs and errors showed much steeper rates of improvement. The authors note that the factor analysis by Fischer et al. (1997a, 1997b), which included age as one of the variables, assumed a linear relationship between age and performance, whereas their (i.e., Fischer et al.'s) data showed non-linear relationships. Klein et al. further examined the nature of the relationship between age and saccade variables using multiple regression and found different relations for different variables. In most instances, a non-linear relation between age and the variable ( $\text{age}^{-1}$ ) provided a better fit with the data than a linear relation. The effects of the experimental variables (pro vs. anti and gap vs. overlap) changed non-linearly with age, becoming smaller in older participants. A principal components analysis conducted on 22 variables derived from the two tasks yielded five factors. Of these, the two factors related to the antisaccade task showed the strongest relation to age, whereas factors related to the prosaccade task and premature responses were not related to age.

The data from the same participants were re-analyzed to examine express saccades (saccades with RTs of 80–130 ms; Klein & Fischer, 2005a). Based on data from adults, it had been argued previously that express saccades can be distinguished from regular saccades in terms of their neural substrates and that they reflect the state of attentional engagement. Principal components analyses indicated that both express prosaccades and “express errors” on the antisaccade task loaded on the same factor, which showed little developmental change. Antisaccade errors and prosaccade RTs loaded on another factor, which was not related to express errors and which showed developmental change. The authors used these developmental differences to bolster the argument that express saccades are distinct from regular saccades.

In a study of pro- and antisaccades in 5- to 79-year-olds (Munoz, Broughton, Goldring, & Armstrong, 1998), results showed a U-shaped curve for saccadic RTs, with 18- to 22-year-olds showing the fastest RTs. The 5- to 8-year-olds had the largest variability in saccadic RTs, the greatest frequency of express saccades in overlap, and the largest gap effect. Results were interpreted as indicating that young children have poor voluntary control over fixation, leading to express saccades or to extremely long saccadic RTs. The youngest children had a high frequency of antisaccade errors (close to 50%), which decreased to 10% by age 15 and stabilized after age 20. The 5- to 8-year-olds also showed hypometria on the prosaccade task. However, peak velocity and duration of the saccades did not change until age 60, suggesting maturation of the brainstem regions involved in saccade dynamics by



age 5. Results were attributed to development of cortico–cortical connections in the frontal cortex throughout adolescence.

Fukushima, Hatta, and Fukushima (2000) also examined pro- and antisaccades in children and adults. Peak velocity of prosaccades was at adult levels by 4 years of age, but saccadic RTs did not plateau until age 12. Antisaccade errors and RTs both decreased with age, but had not reached adult values by age 12. In a second study, the effects of an auditory warning signal during fixation were compared between 6- and 8-year-olds and adults. Adults and children benefited similarly from the signal, but the children benefited to a lesser extent than the adults on both pro- and antisaccade tasks. Results were interpreted as indicating that the brainstem structures involved in saccade dynamics (especially the paramedian pontine reticular formation) are mature by age 4, whereas frontal association areas involved in pro- and antisaccade RTs and antisaccade errors (frontal and supplementary eye fields, and the dorsolateral prefrontal cortex) reach maturity later.

Pro- and antisaccades were examined in a sample of female twins to examine genetic and environmental contributions to performance at different ages (Malone & Iacono, 2002). Results showed that error rates decreased from 45% to 29% between 11 and 17 years of age ( $d = 0.81$ ). However, the contributions of genetic and environmental influences did not appear to differ between the two ages.

Finally, a recent study tested if improvements in working memory capacity or inhibitory control were responsible for improvements in antisaccade performance with age (Eenshuistra, Ridderinkhof, Weidema, & van der Molen, 2007). The authors concluded that the developmental improvement in antisaccade accuracy from age 8 to young adulthood was better explained by improvements in working memory capacity than in inhibitory control.

The primary goal in several other studies was to draw inferences about cognitive development that go beyond the saccadic system. In a study by (Ross, Hommer, Breiger, Varley, & Radant, 1994a, 1994b), three tasks were used to examine the development of visual–spatial attention in 7- to 15-year-olds. On the first task, children made prosaccades to targets appearing at unpredictable locations. On the second task, the location, but not the timing, of the target was predictable, and the children were instructed to refrain from making saccades until the target appeared. On a third task, the children fixated a visual stimulus, without any distractors. Results showed that ability to suppress saccades during fixation leveled off by age 10, the ability to inhibit premature saccades to predictable targets continued to improve until age 12, and saccadic response time (RT) on all tasks decreased linearly between age 7 and age 15. However, there was a great deal of variability at each age. The authors concluded that visuospatial attention had different developmental trajectories depending on whether children were required to ignore internal or external distractors.

In a larger study (Luna, Garver, Urban, Lazar, & Sweeney, 2004), 8- to 30-year-olds were examined on pro- and antisaccades, and memory-guided saccades. Compared to linear, exponential, quadratic, and cubic functions, inverse regression functions (which allowed for both steep improvements in performance and asymptotes) provided the best fit to the data. RTs on all tasks fit this model, as well as errors on the antisaccade task, and distance errors on the memory-guided task. The age at which the data of the children did not differ from those of adults was taken as an indication that performance had matured. Antisaccade errors and distance errors of initial memory-guided saccades reached this criterion at age 14, saccadic RTs on all tasks at age 15, and distance errors

of final saccades at age 19. Correlational analyses suggested that processing speed (inferred from saccadic RTs across all tasks) developed independently from response inhibition (as indicated by antisaccade errors) and working memory (inferred from distance errors of memory-guided saccades), but that working memory contributed to the development of response inhibition and that the development of processing speed and response inhibition both contributed to the development of working memory. The authors point out that improvements on these tasks likely depend on maturation of distributed neural circuits, and suggest that their results indicate “progressively more efficient use of brain circuitry” through adolescence (p. 1369).

A third study examined the development of different types of inhibition in 8- to 25-year-olds by comparing age-related changes on the pro- and antisaccade tasks to those on an oculomotor capture task (Kramer, Gonzalez de Sather, & Cassavaugh, 2005). Although both the antisaccade and oculomotor capture tasks require participants to avoid “capture” by the sudden onset of a visual stimulus, the antisaccade task makes greater demands on top-down attentional control. Antisaccade errors were equally high in the three younger groups, and were significantly lower in the two older groups. In contrast, errors on the oculomotor capture task did not vary with age. The authors suggest that “the ability to exert top-down control in opposition to attentional capture engendered by stimulus-driven influences (e.g., the sudden appearance of a new object in the visual field) exists by 8 years of age. However, the ability to maintain multiple top-down sets (e.g., inhibit an eye movement to a salient stimulus and move the eyes in the opposite direction ...) seems to take substantially longer to develop” (p. 768).

Only three studies so far have examined the neural correlates of saccades in children. Luna and Colleagues (2001) compared three age groups on pro- and antisaccade tasks while they underwent functional brain imaging. The day before scanning, participants were administered a block of antisaccades and a block of prosaccades. In the scanner, they were administered one task consisting of alternating 30-s blocks of prosaccades and active fixation, and another task consisting of alternating 36-s trials of pro- and antisaccades. Performance in the scanner was not measured but was assumed to be similar to performance on the previous day. Results of the tasks administered in the lab indicated that antisaccade error rates decreased with increasing age, whereas RTs and other dynamics of the antisaccades did not change with age. There were no age-related changes in brain activation in the supplementary eye fields, insula, precuneus or anterior cingulate during the antisaccade task. However, compared to adults, there was less activation in children and adolescents in the superior frontal eye fields, intraparietal sulcus, thalamus, cerebellum, and superior colliculus. The younger children showed less activation in the basal ganglia than adolescents or adults. On the other hand, compared to adults, children had greater activation in the supramarginal gyrus, which lies in the inferior parietal lobe. Surprisingly, the adolescents showed greater activation than either children or adults in the dorsolateral prefrontal cortex. Results were interpreted as indicating that age-related changes in antisaccade performance “is influenced by the maturation of integrated function among the neocortex, striatum, thalamus, and cerebellum,” possibly through synaptic pruning and myelination (p. 791).

In a subsequent study, these researchers (Scherf, Sweeney, & Luna, 2006) compared participants drawn from those who took part in the previous study on a memory-guided saccade task. As in the previous study, slightly different versions of the eye movement tasks were administered before and during scanning, although behavioral results were

recorded only on tasks administered prior to the scan. There were no age-related changes in prosaccade RTs. The behavioral results for memory-guided saccades differed somewhat depending on whether an ANOVA or an inverse curve-fit was used for analyses (see Table 2). A complex set of neuroimaging results was obtained, as 22 regions were analyzed in each hemisphere, using three different methods of analysis (visual inspection of proportion of significantly active voxels per region, correlations between age and activation in each region, and voxelwise ANOVAs comparing the three ages). The authors interpreted the findings as indicating that there are both quantitative and qualitative changes with age in the neural substrates of visual-spatial working memory, possibly due to synaptic pruning and myelination.

Finally, Klein and Feige (2005) examined age-related changes in the contingent negative variation (CNV) to the warning stimulus on pro- and antisaccade tasks in 7- to 18-year-olds. When the warning stimulus predicts the imperative stimulus with certainty, the CNV reflects both contingency formation and response preparation, and previous studies had indicated that the CNV is larger before anti-compared to prosaccades. Results showed that the topography of the CNV changed with age on both tasks, with a lateral-posterior source in 7- to 11-year-olds, an anterior-central source (similar to the adult pattern) in 17- to 18-year-olds, and transitional patterns in 12- to 16-year-olds. Results were specific to the CNV: the young children showed another slow negative potential (related to stabilization of gaze after saccade execution) at anterior-frontal leads, suggesting that they were able to activate these regions as effectively as adults for another purpose. The authors concluded that “the cognitive functions supported by the anterior-central CNV generating structures are supported by different cortical regions (possibly located in the inferior parietal lobe) in children” (p. 8).

### *Pursuit*

In pursuit studies with healthy children, eye tracking has been used as a tool to make inferences about the development of the smooth-pursuit system and its interactions with the saccadic system. The results of pursuit studies in children are summarized in Table 3. As can be seen in this table, there are quite a few discrepancies across studies in target velocity and whether it was constant or not, operational definitions of pursuit performance (particularly for intrusive and compensatory saccades), and the method of analyzing age-related differences. With these caveats in mind, the studies reviewed in this section suggest that there are either small or no differences in gain between young children (around age 7) and adults for slow targets, but that performance continues to improve through adolescence for faster targets (e.g., see Fig. 6). Results are inconsistent for intrusive and catch-up saccades.

The results of one study (Accardo, Pensiero, Da Pozzo, & Perissutti, 1995) are too detailed to include in Table 3. In this study, performance was compared between 7- and 12-year-olds and 30- and 38-year-olds. Velocity gain, assumed to reflect the functioning of the pursuit system, was close to perfect in adults for 0.2- and 0.4-Hz targets, but decreased with each increase in velocity. Children had smaller gains than adults at all velocities, but the differences were larger at higher velocities. Position gain, assumed to reflect the interaction of the pursuit and saccadic systems, was at ceiling levels in both children and adults for 0.2- and 0.4-Hz targets. The adults' position gain did not decline until target velocity reached 1.2 Hz, whereas the children's gain began to decline at 0.8 Hz. The

Table 3  
Smooth-pursuit eye movement studies in normative development

Study	Age <sup>a</sup>	<i>N</i>	Target velocity <sup>b</sup>	Duration	Measures	Analysis <sup>c</sup>	Results <sup>c</sup>
Haishi and Kokubun (1995)	3–4	9	0.3, 0.5, 0.7 Hz (sinusoidal)	15 s/frequency	Power ratio (an estimate of how smooth the eye movements are); Phase difference	ANOVA	Power ratio: no effect & no interaction between age and frequency among the three child groups Phase: no effect of age within child groups, but interaction; eye movements lagged behind target in 3- to 4-year-olds, ahead of target in 5- & 6-year-olds
	5	12					
	6	7					
	22–37	5					
Langaas et al. (1998) <sup>d</sup>	5–7 Undergraduates	24 8	0.3 Hz/11.3°/s (sinusoidal)	Two 30-s cycles	Gain, saccades	Gain: Mann–Whit. <i>U</i> Saccades: ANOVA & Scheffe	Gain: ~1 in both groups, ns. Sac ( $\geq 0.5^\circ$ ): children > adults Sac ( $< 0.5^\circ$ ): ns
Accardo et al. (1995)	7–12 30–38	10 10	0.2, 0.4, 0.8, 1.0, 1.2 Hz (cosinusoidal)	5-min total	Velocity & position gain	Visual inspection	Velocity gain: adults > children for all frequencies See text for more information
Salman et al. (2006b) <sup>e</sup>	8–19	38	0.25 (15.5°/s) (sinusoidal) 0.5 (31°/s) (sinusoidal)	20 cycles/freq	Gain & phase	Correlation	Gain ↑ for both 0.25- and 0.5-Hz Gain reached adult level by mid adolescence Phase: ns

Katsanis et al. (1998) <sup>f</sup>	11–12 17–18 34–63	62 39 36	0.4 Hz (sinusoidal)	30 s	Gain, RMSE, saccades	MANOVA, chi-sq., post-hoc tests Levene test for homogeneity of variance	Compared to the older groups, the youngest group had lower gain, higher RMSE, more anticipatory ( $\geq 5^\circ$ ) sac, greater variability in gain & RMSE Catch-up sac: ns
Ross et al. (1993)	11.4 (7–15)	53	6, 12°/s (constant)	<30 s/frequency	Gain, saccades	Correlation	6°/s target. Sac.: ns 12°/s target. Intrusive sac.: ns 12°/s target. gain ↑ & catch-up sac. ↓
Ross et al. (1994a, 1994b)	11.5 (8–15)	51	9°/s (constant); target stepped to right or left, then moved smoothly in the same or opposite direction	<30 s/task	Initiation & maintenance gain	Correlation, ANOVA	Initiation phase: ns M. gain for opposite-direction targets: ↑ M. gain for same-direction targets: ns
Takarae et al. (2004) <sup>g</sup>	$M = 19.3$ ( $SD = 11.3$ )	94	4 to 32°/s (constant) Foveofugal and pure step-ramp tasks; oscillating target task	Foveofugal: 32 tr. Pure: 40 tr Oscillating: 22 s at each of four frequencies	Maintenance gain	Inverse regression	Inverse regression models yielded results for maintenance gain on all tasks

*Abbreviations:* ↑, increased with age; ↓, decreased with age. chi-sq., chi-square. *M*, mean *M*. gain, maintenance gain. Mann–Wh. *U*, Mann–Whitney *U*; ns, not significant. RMSE, root-mean square error. Sac., saccade(s).

<sup>a</sup> Numbers within parentheses refer to standard deviation or range, when available.

<sup>b</sup> Target velocity refers to peak target velocity in studies in which velocity was not constant.

<sup>c</sup> All analyses and results refer to those involving age-related differences.

<sup>d</sup> Participants were controls compared to children born prematurely or with developmental coordination disorder.

<sup>e</sup> The authors also examined vertical pursuit. Results showed a great deal of inter-subject variability and weaker developmental changes than for horizontal pursuit.

<sup>f</sup> The younger two groups consisted of the first-borns of male twin pairs. Eleven of the adults were parents of the younger participants.

<sup>g</sup> Participants were controls compared to children with autism, reviewed below.

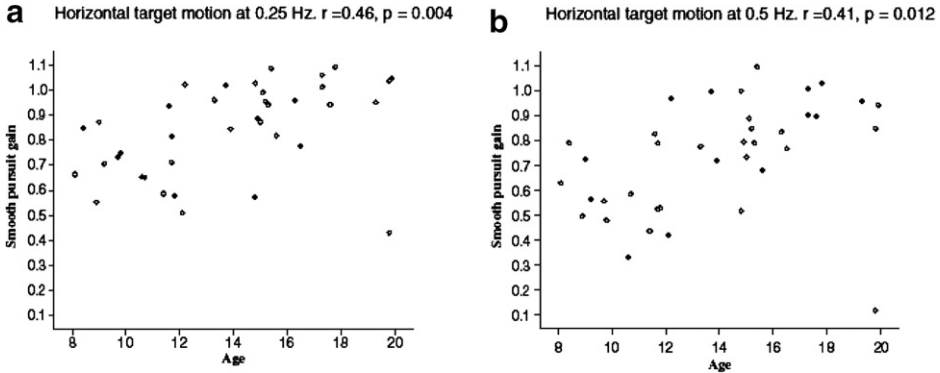


Fig. 6. Smooth-pursuit gain as a function of age and two target speeds, 0.25 Hz (a) and 0.5 Hz (b; Salman et al., 2006b). Reprinted with kind permission of Springer Science and Business Media.

discrepancy in the results for velocity versus position gain was interpreted as indicating immaturity of the pursuit system in the 7- to 12-year-olds despite efficient interactions between the pursuit and saccade systems for slow targets. The children's values were also more variable than those of adults for both velocity and position gain.

#### *Eye movements during scene and face perception*

Because there are no standard paradigms for assessing eye movements during scene or face perception, there is a great deal of diversity in the studies reviewed here and less of a sense of accumulating knowledge about a specific research question. All of the developmental studies in this area have included 1–4 age groups, with small to medium sample sizes.

Eye movements have been used in several studies to examine how children interpret and resolve linguistic ambiguities. Most tasks used with adults in research on this topic involve reading and are not suitable for children. Therefore, eye movements were particularly useful in extending this research to younger ages. In one study (Trueswell, Sekerina, Hill, & Logrip, 1999), eye movements of 5-year-olds ( $N = 16$ ) and 18- to 22-year-olds ( $N = 12$ ) were examined as participants were responding to spoken instructions to move objects around a table. The instructions involved syntactic ambiguities in the early part of the sentence, which could then be revised with the contextual information presented (e.g., “put the frog on the napkin in the box”). The adults took into account the contextual information as they were listening to the instructions and changed their original interpretation when appropriate. The children, however, rarely changed their original interpretation, as indicated by the location of their fixations during the course of the instruction. In contrast, their fixation patterns were very similar to those of adults for syntactically unambiguous sentences. The findings were interpreted as indicating that children rely on local linguistic information to resolve ambiguities, and have difficulty revising their initial interpretations and coordinating multiple sources of linguistic and contextual information as they process language.

In another study (Sekerina, Stromswold, & Hestvik, 2004), 4- to 7-year-olds ( $N = 16$ ) and adults ( $N = 18$ ) listened to sentences containing referentially ambiguous pronouns

(e.g., “the boy washed him”) and chose a picture corresponding to their interpretation of the sentence. The resolution of the ambiguity depended on whether the participants used contextual information (“sentence-external referent”) or not (“sentence-internal referent”). Along with behavioral data, the researchers analyzed participants’ fixations on different regions of the pictures as they listened to the sentences. Results indicated that the adults were immediately aware of the ambiguity. Their fixations alternated between the two interpretations during the 1 s after the pronoun, although they tended to choose the more easily accessible, sentence-internal referent. The behavioral responses of the children indicated that the majority did not take contextual information into account. Their eye movements, however, revealed that they became aware of the ambiguity toward the end of the trial, by the time the adults had already resolved it. Thus, as in the study by Trueswell et al. (1999), the children were less likely than adults to revise their original interpretations and had difficulty coordinating linguistic and contextual information.

Two studies of eye movements in young children were aimed at examining the role of shared storybook reading in emergent literacy. In one study (Evans & Saint-Aubin, 2005), two experiments were conducted on 48- to 61-month-olds to determine how much the children looked at the text as they were read storybooks with pictures. Five children participated in Experiment 1, and 10 in Experiment 2. The layout of the print in relation to the pictures and the richness of the pictures’ color were manipulated, and the total duration of fixations on print versus pictures was analyzed. Contrary to common assumptions about shared reading having a specific effect on promoting children’s print knowledge, the results indicated that the children’s fixations fell overwhelmingly on the pictures.

These results were replicated in another study (Justice, Skibbe, Canning, & Lankford, 2005), in which 50- to 69-month-olds ( $N = 10$ ) were presented with storybooks on a computer screen while they were listening to adults read the stories. The salience of the print versus the pictures was manipulated, and the number and total duration of fixations on print and pictures were analyzed. The proportion of fixations falling on print was 3% when the pictures were salient and increased to only 7% when the print was made more salient. Total durations were 2.5% and 6%, respectively. Thus, the eye tracking data revealed that children rarely attended to print on their own as they were looking at storybooks.

Two developmental studies examined eye movements during face perception. In one study (Schwarzer, Huber, & Dümmler, 2005), two experiments were conducted to examine holistic and analytic modes of processing faces. In Experiment 1, 6- to 8-year-olds ( $N = 24$ ) and 9- to 10-year-olds ( $N = 20$ ) were compared to 17- to 35-year-olds ( $N = 20$ ; median age = 24). Participants were instructed to assign pictures of schematic faces to one of two categories. The faces were constructed so that they could be categorized using either an analytical or a holistic strategy. Which strategy participants used was deduced from their responses. Results showed that most of the participants, regardless of age, used an analytical strategy. In Experiment 2, holistic processing was induced through the use of photographs of faces. Participants were 6- to 7-year-olds ( $N = 27$ ), 9- to 10-year-olds ( $N = 27$ ), and 21- to 39-year-olds ( $N = 21$ ; median age = 24). Most of the youngest participants used either a purely analytical strategy (24%) or other strategies that relied on analytical processes in an inconsistent manner (57%). The proportion of holistic processors increased from 19% in the youngest group to 44% in the 9- to 10-year-olds and 63% in the adults, consistent with other studies showing a shift with age from analytical to holistic face processing. In both experiments, the fixations of the holistic processors tended to fall

on the eyes and the nose, whereas the fixations of the analytical processors were more distributed across facial features. Although the adults made fewer fixations than the younger groups (consistent with holistic processing), there were no age-related differences in gaze time to different facial regions.

The development of facial expression perception was examined in another series of studies comparing 8-year-olds, 12-year-olds and adults (Marcus, 2005). Although adults performed differently than children on behavioral measures (showing better accuracy for identifying emotions in inverted faces, sharper distinctions between emotional categories, and a stronger bias away from angry faces), there were few differences between the age groups in terms of eye movements to faces depicting different emotions.

Finally, we used eye tracking to examine incidental and intentional spatial sequence learning (Karatekin, Marcus, & Couperus, 2007; Karatekin, Marcus, & White, 2007). We tested four age groups [8- to 10-year-olds ( $N = 35$ ), 11- to 13-year-olds ( $N = 28$ ), 14- to 17-year-olds ( $N = 13$ ), and young adults ( $N = 24$ )] on a serial reaction time task. Participants were administered 5 blocks of trials in which a stimulus appeared in one of four boxes. They were instructed to look at the stimulus and to press the corresponding button. Unbeknownst to them, the second, third and fifth blocks contained 10 repetitions of a 10-step sequence, and the first and fourth blocks contained stimuli in pseudo-random order. The dependent variables included manual responses and oculomotor anticipations (looks to the target location prior to target onset), and incidental learning indices (improvement in performance with increasing exposure to the sequence and interference on the fourth, pseudo-random, block). As in a previous study with adults (Marcus, Karatekin, & Markiewicz, 2006), participants spontaneously tried to anticipate the target location from the beginning of the task, regardless of age, and oculomotor anticipations and RTs showed learning effects similar to those in the manual modality. There were few age-related differences in sequence learning indices in either the manual or oculomotor modalities. However, compared to older participants, the youngest group had a more shallow rate of decrease in oculomotor RTs with increasing exposure to the sequence on the first sequence block, hinting at an age-related difference in rate of learning. On a final block in which participants were explicitly instructed to learn a sequence, all measures showed age-related differences, including between adolescents and adults. Within-block analyses of the manual and oculomotor variables showed that the adults were initially slowed down, but they improved quickly. In contrast, instructions to learn the sequence explicitly did not benefit the adolescents, and hurt the performance of the youngest group. Taken together, the results suggested that the search for regularities and the ability to rapidly learn a sequence incidentally are mature by ages 8–10. In contrast, the ability to learn a sequence intentionally, which requires cognitive resources and strategies, continues to develop through adolescence.

### *Pupillary dilation*

To my knowledge, there are only three studies in which pupillary dilation was used as an index of cognitive processes in children and adolescents. In a 1970 study, pupillary dilation was examined as a function of difficulty of mental arithmetic problems in 10 “educable retardates” (mean age = 10.6,  $SD = 0.11$ ) and 10 controls (mean age = 11.0,  $SD = 0.6$ ) (Boersma, Wilton, Barham, & Muir, 1970). As expected, dilation was greater for difficult compared to easy problems, with dilation increasing during the response period of 20 s in



controls. Although there were no group differences in dilation before the presentation of the problems or during the first part of the response period, the “retardate” group had smaller dilations than controls during the rest of the response period, particularly for the difficult questions. In addition, increases in dilation were related to accuracy in both groups. The authors attributed the results in the “retardate” group to a greater degree of attentional fluctuation.

We used pupillary dilation in two studies to examine development of attention and working memory. In one study (Karatekin, 2004), top-down control over attention was investigated on a dual task in 10-year-olds ( $N = 15$ ) and adults ( $N = 21$ ). The tasks were an auditory digit span (with three sequence lengths) and a simple visual RT task. In four conditions, participants performed neither (No-task), one (Digit Span or RT Only), or both tasks (Dual). Dependent variables were digit span accuracy, manual RT, and pupillary dilation to digits. As expected, the behavioral results indicated that children were less accurate and slower than adults. The pupillary results are depicted in Fig. 7. At both ages, the slopes of the functions relating pupillary dilation to the presentation of the digits were flat in No-task and RT Only, linear and increasing in Digit Span Only and Dual, and shallower in Dual than in Digit Span Only. In addition, the slopes were shallower in children

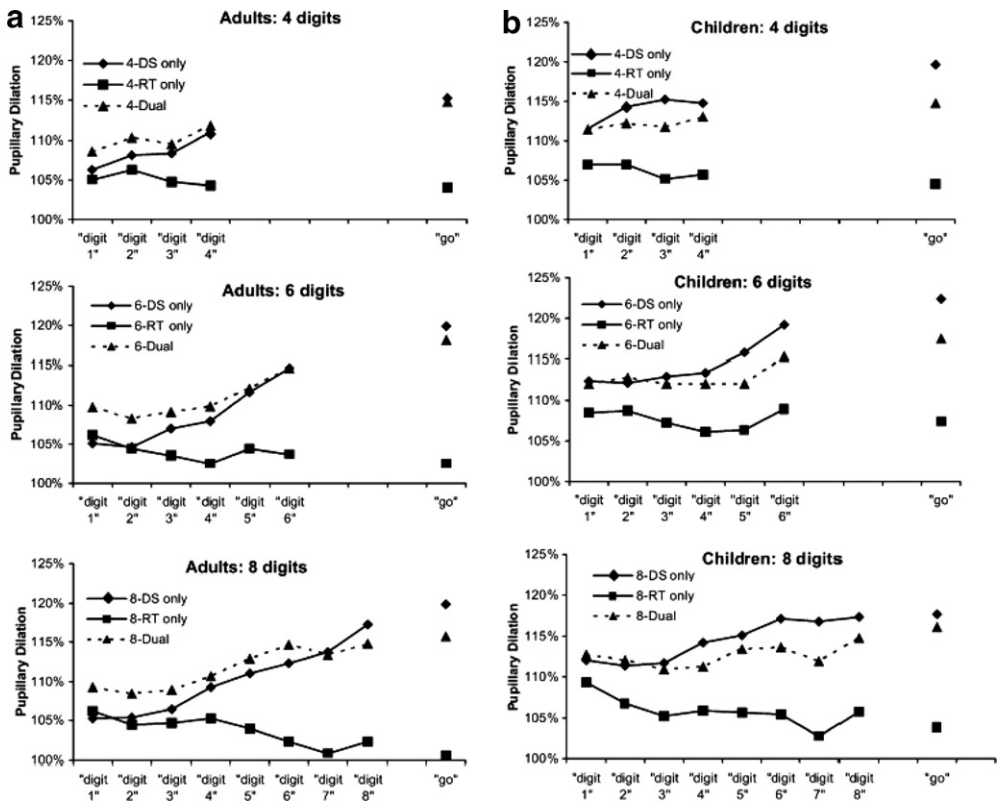


Fig. 7. Pupillary dilation (% increase over no-task) to the auditory stimuli as a function of condition and sequence length in (a) adults vs. (b) 10-year-olds. Figure and figure caption reprinted from Karatekin (2004), with permission from Elsevier.

Table 4  
Eye tracking studies in children and adolescents with schizophrenia-spectrum disorders or at risk for schizophrenia

Study	N	Groups	Age <sup>a</sup>	M:F	IQ <sup>b</sup>	Task <sup>c</sup>	Results <sup>d</sup>
Jacobsen et al. (1996)	17	COS	14.5 (10–18)	59:41	85 (17)	Pursuit (11°/s), (constant, 5 cycles)	Gain: COS < ADHD = C
	18	ADHD-III-R	12.6 (9–15)	94:6	111 (17)		RMSE: COS > ADHD > C
	22	Control	13.5 (9–18)	73:27	116 (18)		Anticipatory ( $\geq 4^\circ$ ) sacc: COS > ADHD = C Back-up sac: COS > ADHD = C Catch-up sac: ns
Kumra et al. (2001) <sup>e</sup>	29	COS	14.6 (2.5)	55:45	7.3 (3.7)	Pursuit (17°/s) (constant, 10 cycles)	Gain: COS & Psy NOS < C
	20	Control COS	14.6 (2.3)	55:45	11.2 (2.1)		RMSE: COS & Psy NOS > C
	26	Psy NOS	13.7 (3.5)	85:15	7.8 (3.1)		Catch-up sac: COS > C
	18	Control NOS	13.1 (3.4)	72:28	15.8 (2.5)		All anticipatory sac: ns Large (>4°) anticipatory sac: ns
Ross et al. (1996)	13	At risk	10.6 (6–15)	39:61	9.3 (2.6)	Pursuit (12°/s, constant, not rep.)	Gain: At-risk < C
	19	Control	11.1 (6–15)	47:53	11.9 (2.8)		RMSE: At-risk > C Small (~2°) anticipatory sac: at-risk > C Catch-up sac: ns
Ross et al. (1999)	13	At risk	10.6 (6–15)	39:61	9.3 (2.6)	Pursuit (12°/s or 17°/s, <sup>f</sup> constant, 3 min) <sup>g</sup>	Anticipatory sac: COS, adult-onset sz, and both
	19	Control	11.1 (6–15)	47:53	11.9 (2.8)		sets of parents >age-matched C
	10	COS	10.3 (7–15)	70:30	Not rep.		COS group had more anticipatory sac than
	14	Adult-onset sz	21 (16–29)	Not rep.	Not rep.		adult-onset sz & at-risk children.
	20	Parents of COS	(28–53)	Not rep.	Not rep.		Bilineality (pursuit impairments in both parents)
	28	Parents of adult sz	(40–81)	Not rep.	Not rep.		greater in
10	Control adults	(22–45)	Not rep.	Not rep.	parents of COS than in parents of adult-onset sz		
Ross (2003)	49	COS	10.4 (2.5)	71:29	Not rep.	Pursuit (17°/s, constant, not rep.),	Gain: COS < at-risk = C
	60	At risk	10.5 (2.4)	48:52	Not rep.		Large (>4°) anticipatory sac: COS > at-risk = C
	80	Controls	11.5 (2.5)	47:53	Not rep.		Small (1–4°) anticipatory sac COS > at-risk > C Catch-up sac: ns

Ross et al. (2005)	45	COS	10.4 (2.4)	73:27	Not rep.	MGSs (1 & 3 s, tr. not rep.)	Premature MGSs: COS > at risk = C Spatial accuracy at 1 s: COS < at-risk = C Spatial accuracy at 3 s: ns Sac RT: ns
	58	At risk	11.2 (3.1)	53:47	Not rep.		
	84	Control	11.3 (2.7)	46:54	Not rep.		
Schreiber et al. (1997)	21	At risk	13.0 (2.6)	48:52	116 (16)	Visually guided saccades (targets stepped 10–60°; 109 tr.)	Sac of at-risk group were hypometric, particularly for 40° to 60° targets. Frequency of hypometric sac ↓ in C but not in at-risk
	21	Control	13.0 (2.7)	48:52	117 (12)		
Karatekin and Asarnow (1998)	13	COS	14.4 (3.3)	54:46	89 (17)	Visually guided saccades (72 tr./task) Parallel & serial search (216 tr./task)	RT of visually guided sac: ns Parallel search rate: ns Serial search rate: COS = ADHD > C RT of first sac in parallel search: ns RT of first sac in serial search: ADHD > COS = C
	28	ADHD-III-R	13.9 (3.1)	68:32	105 (16)		
	38	Control	14.1 (2.7)	47:53	106 (17)		
Karatekin and Asarnow (1999)	13	COS	14.5 (3.3)	54:46	89 (17)	Eye movements during scene perception (5 pictures, 8 s/picture)	COS looked at fewer relevant, but not more irrelevant regions than C; COS stared more for global, but not for structured questions ADHD had shorter fixations than C for questions requiring detailed analysis
	30	ADHD-III-R	13.8 (3.1)	63:37	106 (15)		
	26	Control	13.2 (2.4)	46:54	105 (18)		

*Abbreviations:* III-R, the children were diagnosed using the DMS-III-R, which did not specify ADHD subtypes. ADHD, Attention-deficit/hyperactivity disorder. C, controls. COS, childhood-onset schizophrenia. MGSs, memory-guided saccades. NOS, not otherwise specified. Not rep., not reported. Ns, not significant. Psy NOS, psychotic disorder not otherwise specified. RMSE, root-mean square error. RT, response time. Sac, saccade(s) or saccadic. Sz, schizophrenia. Tr., trials.

<sup>a</sup> Ages refer to mean ages, numbers within parentheses refer to standard deviation or range, when available. Age was used as a covariate in Jacobsen et al. (1996), Ross et al. (1996,2003,2005).

<sup>b</sup> IQ was estimated from Vocabulary subtest scores in Kumra et al. (2001), Ross et al. (1996, 1999).

<sup>c</sup> Information in parentheses refers to task characteristics, including number and duration of trials. In pursuit studies, the information in parentheses refers to, in order, target speed, whether it was constant or not, and total duration or cycles for the task.

<sup>d</sup> All results refer to those involving group differences.

<sup>e</sup> Different control groups were used for the schizophrenia and psychosis NOS groups to provide matching on age and gender.

<sup>f</sup> The task was the same as in Ross et al. (1996); however, the parents of the two schizophrenia groups were presented with targets moving at 16.7° rather than 12°/s.

<sup>g</sup> Pursuit performance was recorded in 60-s intervals for the adults and 30-s intervals for the children in repeated trials until 3 min of usable data were obtained.

than adults in both Digit Span Only and Dual. These findings suggested that although the 10-year-olds allocated their attention between tasks and across increasing memory loads in a similar manner as adults, their ability to recruit sufficient resources at higher loads was not yet fully mature.

In a second study, we examined the regulation of cognitive resources in 10-year-olds ( $N = 32$ ) and young adults ( $N = 72$ ) on spatial  $n$ -back tasks assessing sustained attention and spatial working memory (Karatekin, Marcus, & Couperus, 2007; Karatekin, Marcus, & White, 2007). One third of the participants were administered 0-back twice, a third was administered 0-back first and 1-back second, and a third was administered 1-back first and 0-back second. Performance was assessed with behavioral measures (accuracy, RT) and pupillary dilation. Repeated administration of 0-back led to a decrease in pupillary dilation and increase in RT variability, revealing a subtle vigilance decrement. Effects of repeated administration of 0-back were similar between ages. Compared to adults, children's sensitivity ( $d'$ ) and RTs were not disproportionately affected by 1-back. However, they showed a disproportionately higher response bias ( $c$ ) and larger pupillary dilations to hits on 1-back, suggesting that they were not as effective as adults in extracting information about target frequency when there was a working memory load. Thus, on these relatively simple tasks of sustained attention and working memory, 10-year-olds appeared to recruit resources in a manner similar to adults.

### **Eye tracking in children and adolescents: Atypical development**

In this section, eye tracking studies will be reviewed in three disorders in which a fair number of such studies have accumulated. There is a growing body of research on eye movements in learning disabilities (e.g., Descroches, Joannis, & Robertson, 2006; Fischer, Hartnegg, & Mokler, 2000; Fukushima, Tanaka, Williams, & Fukushima, 2005; Hutzler, Kronbichler, Jacobs, & Wimmer, 2006). This research will not be reviewed here to limit the scope of this review. Eye tracking has been used in several other disorders in children and adolescents, including developmental coordination disorder and prematurity (Lang-aas, Mon-Williams, Wann, Pascal, & Thompson, 1998), neurofibromatosis (Lasker, Denc-kla, & Zee, 2003), obsessive-compulsive disorder (Rosenberg et al., 1997), fragile X and Turner syndromes (Lasker, Mazzocco, & Zee, 2007), depression and anxiety (Jazbec, McClure, Hardin, Pine, & Ernst, 2005).

#### *Schizophrenia*

One of the most robust findings in the schizophrenia literature is an impairment of smooth-pursuit eye movements, not only in actively psychotic individuals but also in remitted patients and in unaffected relatives of individuals with schizophrenia. These findings indicate that pursuit impairments reflect a genetic vulnerability to the disorder (for reviews, see Broerse, Crawford, & den Boer, 2001; Holzman, 2000; Hutton & Kennard, 1998; Reuther & Kathmann, 2004; Trillenberg, Lencer, & Heide, 2004). Thus, studies of youth with schizophrenia-spectrum disorders (who have a more severe and genetically loaded, but not qualitatively different, form of the disorder; for a review, see Asarnow & Karatekin, 2000) and high-risk offspring of parents with schizophrenia have focused mostly on pursuit abnormalities. As can be seen in Table 4, the results of pursuit studies in youth with schizophrenia-spectrum disorders or at genetic risk for schizophrenia have

shown fairly consistently that schizophrenia and the risk for schizophrenia are associated with reduced gain. However, the operational definitions and the findings related to intrusive and catch-up saccades are not as consistent. For comparisons to normative development, it should be noted that all of the studies reviewed in this section included targets moving at a constant velocity of 11 to 17°/s, whereas the velocity and the nature of motion of the target differed considerably across the normative studies. In addition, given the rarity of schizophrenia in children and the difficulties involved in recruiting at-risk children, most of these studies included participants across a wide age range, and their data were averaged in group comparisons.

Pursuit performance was assessed in participants with childhood-onset schizophrenia compared to controls and participants with Attention-Deficit/Hyperactivity Disorder (ADHD) in Jacobsen et al. (1996) and controls and participants with Psychotic Disorder Not Otherwise Specified in Kumra et al. (2001). The pattern of deficits was taken to indicate support for continuity between the adult- and childhood-onset forms of schizophrenia in both studies, as well as for the specificity of deficits to schizophrenia (Jacobsen et al., 1996) and similarity between adolescents with schizophrenia and those diagnosed with a less well-defined psychotic disorder (Kumra et al., 2001). Typical results from schizophrenic, ADHD and control participants are depicted in Fig. 8.

Ross and colleagues have conducted a series of studies examining pursuit in children with schizophrenia and children at risk for the disorder by virtue of having a parent with schizophrenia. In a study comparing children of schizophrenic parents to controls (Ross et al., 1996), results indicated that the pursuit system was intact in the at-risk group but that it was being affected by intrusive (anticipatory) saccades. The authors suggested that

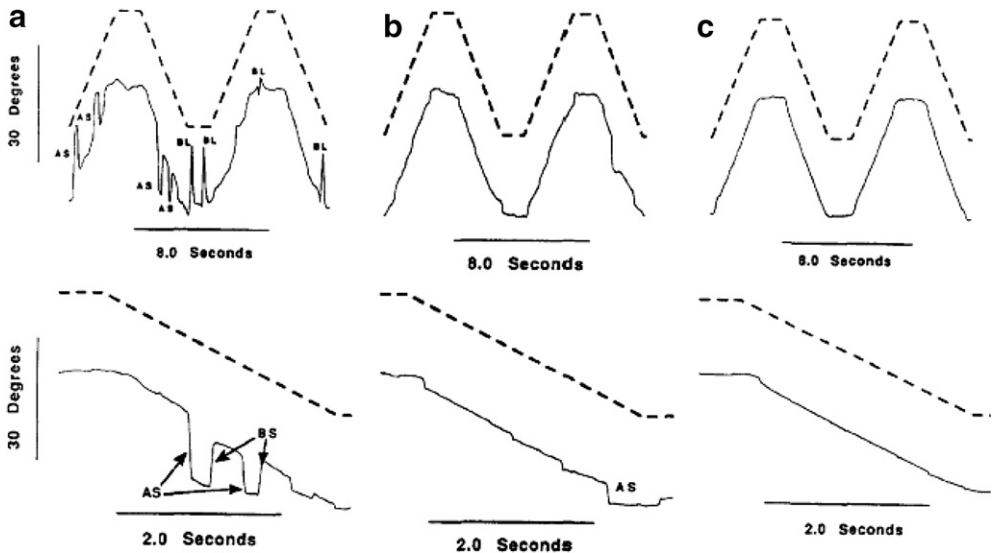


Fig. 8. (a) Eye tracking record from a 15-year-old medication-free subject with childhood onset schizophrenia. Top, 16-s segment. Bottom, enlargement of the first descending ramp. (b) Eye tracking record from a 10-year-old subject with ADHD. Top, 16-s segment. Bottom, enlargement of the first descending ramp. (c) Eye tracking record from a 15-year-old normal subject. Top, 16-s segment. Bottom, enlargement of the first descending ramp. AS, anticipatory saccade; BS, back-up saccade; BL, blink artifact. Figure and figure caption reprinted from Jacobsen et al. (1996), with permission from the Society of Biological Psychiatry.



high frequency of small anticipatory saccades. Because the frequency of these saccades was not correlated with age in the whole sample and because their frequency in the 6- to 15-year-old controls was only slightly higher than the frequency observed in a study of healthy 25- to 50-year-olds, the authors concluded that the neural substrates of small anticipatory saccades are mature by age 6. Therefore, they suggested that this endophenotype for schizophrenia is present and static years before the age of risk for developing the full-blown disorder.

The same children were compared in another study (Ross, Heinlein, Zerbe, & Radant, 2005) on a memory-guided saccade task. The impairments in the schizophrenia group, and the lack of expected impairments in the at-risk group, were taken to support continuity between the childhood- and adult-onset forms of schizophrenia as well as the lack of sensitivity of impairments to genetic risk in children.

Schreiber and Colleagues (1997) found hypometria for visually guided saccades in adolescent offspring of schizophrenic parents compared to controls, but only for high-amplitude targets. The authors noted that this pattern of hypometria was not consistent with an abnormality in the brainstem or cerebellum, but that it could point to dysfunction in the frontal eye fields or the principal sulcus within the dorsolateral prefrontal cortex.

Asarnow and I used eye tracking in two studies to examine visual–spatial attention in childhood-onset schizophrenia. In one study (Karatekin & Asarnow, 1998), we tested if visual search impairments in schizophrenia are due to a delay in initiation of search or a slow rate of serial search. We determined the specificity of these impairments by comparing children with schizophrenia to children with ADHD and controls. The hypotheses were tested within the framework of Treisman’s feature integration theory by administering children parallel and serial search tasks adapted from Treisman and Souther (1985). Search rate was estimated from the slope of the search functions, and duration of the initial stages of search from the time to make the first saccade on each trial. Participants were also administered three control tasks to assess the integrity of prosaccades; there were no group differences in saccadic RT on these tasks. Both the schizophrenia and ADHD groups had intact parallel search rates and slowed serial search rates. Contrary to expectations, ADHD, but not schizophrenic, children were delayed in initiation of serial search. We interpreted the results as indicating dysfunction in both schizophrenia and ADHD in the distributed circuitry mediating visual–spatial attention, particularly the prefrontal cortex and thalamus.

In a second study (Karatekin & Asarnow, 1999), we investigated exploratory eye movements to thematic pictures in schizophrenic, ADHD, and control children. For each picture, participants were asked three questions varying in amount of structure (e.g., “what is happening?” vs. “how many animals are there?”). Time spent viewing relevant and irrelevant regions, fixation duration, and distance between fixations were measured. The schizophrenic children looked at fewer relevant, but not more irrelevant, regions than controls. They showed a tendency to stare more when asked to decide what was happening but not when asked to attend to specific regions. Thus, lower levels of visual attention (e.g., basic control of eye movements) were intact in schizophrenic children. In contrast, they appeared to have difficulty with top–down control of selective attention in the service of self-guided behavior. There were few differences between the control and ADHD groups, except for a trend in the ADHD group toward shorter fixations than controls on the question requiring the most detailed analysis.

*Pervasive Developmental Disorders (PDD)*

Table 5 summarizes eye tracking studies in PDD. Because profound social difficulties are among the defining characteristics of PDD, several studies of PDD focused on fixation patterns to socially relevant stimuli. There are discrepancies in the results of these studies, with two showing no or minimal impairment, and two showing abnormal gaze patterns. Nevertheless, these studies have enabled researchers to go beyond gross descriptions of abnormalities in gaze in individuals with PDD and to quantify the extent and nature of the abnormalities in eye movements to static and dynamic scenes and to different aspects of faces. Other studies examined saccades and pursuit. Some of the consistent findings were that the dynamics of saccades (duration, peak velocity) were normal, pursuit was abnormal, and antisaccade errors and premature saccades were more frequent in the PDD compared to the control groups. There are discrepancies across the studies in terms of saccadic RTs and the gap effect. Even so, these studies provide a starting point for characterizing the nature of cognitive and neurobiological impairments in PDD. It is important to note that in most of these studies (1) the samples were quite small, (2) average IQ in the PDD group was in the normal range, whereas the majority of individuals with PDD have low IQs, (3) the overwhelming majority of the participants were male, and (4) as in the schizophrenia studies, the age ranges of the samples were quite large.

In one of these studies on scene perception (Van der Geest, Kemner, Camfferman, Verbaten, & van Engeland, 2002a), children with PDD were compared to controls on fixation patterns to cartoon-like drawings of scenes that included small human figures. The instructions were to look carefully at each picture. Although both groups looked more at the human figures than the rest of the scenes, there were no group differences on other variables. The authors concluded that the gaze abnormalities observed in individuals with PDD in everyday life could be due to the unique demands of social interactions.

In a related investigation that likely included the same participants as in Van der Geest, Kemner, Verbaten, and van Engeland (2002b), children with PDD were compared to controls on fixations to photographs of faces (Van der Geest et al., 2002a). In the first study, the faces depicted four emotions. In the second study, neutral faces were presented upright or inverted. The children were instructed to look carefully at the faces. The dependent variables included number and duration of fixations on five facial regions. The groups did not differ in fixations to the different regions for upright faces, regardless of the emotion depicted. However, the controls spent less time looking at inverted compared to upright faces, whereas the fixation times of the PDD group did not vary as a function of orientation. These results were interpreted as indicating that the abnormal gaze behavior in autism in everyday life may be due to the demands of social interactions, and that autistic children may not be processing faces in a holistic fashion. As noted earlier, there may be a gradual developmental shift from analytic to holistic processing of faces in healthy individuals from 6 years of age to young adulthood (Schwarzer et al., 2005). Thus, the PDD group in Van der Geest et al. (2002a), who had an average age of 10 years, appear to be delayed or deviant in terms of this developmental trajectory.

In another investigation of face processing, Dalton et al. (2005) conducted two studies in which participants were instructed to look at faces while they underwent functional magnetic resonance imaging. In the first study, the focus was on emotion discrimination from photographs of faces. The PDD group was less accurate than controls, particularly when the face was directed at the viewer rather than turned slightly away and when the



Table 5  
Eyetracking studies in autism

Study	<i>N</i>	Groups	Age <sup>a</sup>	M:F	IQ	Task <sup>b</sup>	Results <sup>c,d</sup>
Van der Geest et al. (2002a)	16	PDD (10 HFA)	10.6 (2.1)	Not rep.	93 (17)	Scan drawings of 25 scenes (10 s/scene)	Duration & number of fixations: ns Average & total scanpath length: ns Time to look at human figure: ns Total duration & number of fixations on human figure: ns
	14	Control	9.9 (1.5)		97 (10)		
Van der Geest et al. (2002b)	17	10 HFA, 7 NOS	10.6 (2.1)	94:6	95 (15)	Scan 16 faces depicting emotions, Scan 12 upright, 12 inverted faces (10 s/face)	Fixations to different regions for upright: ns Fixation duration for upright > inverted in C but not in PDD
	17	Controls	10.1 (1.3)	94:6	98 (11)		
Dalton et al. (2005)	11	Autism or Asp	15.9 (4.7)	100:0	94 (19)	Emotion discrimination from 40 faces (3 s/face) Facial recognition (10 familiar, 10 not)	Accuracy: PDD < C Duration of fixations on eyes: PDD < C Fixations on other regions: ns
	12	Control	17.1 (2.8)	100:0	Not assessed		
	16	Autism or Asp	14.5 (4.6)	100:0	92 (28)		
	16	Control	14.5 (4.6)	100:0	123 (13)		
Dalton et al. (2007)	12	Autism or Asp	14.4 (4.8)	67:33	110 (16)	Facial recognition Same as Dalton et al. (2005)	Accuracy for faces: PDD < Sibling, C Duration of fixations on eyes: PDD, sibling < C Fixations on other regions: ns
	10	Sibling	13.1 (3.0)	70:30	122 (15)		
	12	Control	14.2 (3.6)	83:17	116 (8)		
Klin et al. (2002)	15	HFA	15.4 (7.2)	100:0	101 (25)	Face/scene perception during five video clips (30–60 s/clip)	Proportion of time on eyes: PDD < C Proportion of time on mouths, bodies, objects: PDD > C
	15	Controls	17.9 (5.6)	100:0	103 (20)		
Kemner et al. (1998)	10	Autism	10.3 (1.3)	80:20	72 (15)	Visual oddball task (80% frequent, 10% infrequent, 10% novel stimuli; 1 s/stimulus; 140 tr.)	Sac to frequent stimuli: PDD > ADHD, C Sac during 2 inter-trial intervals: PDD > ADHD, C C: more sac to novel than to other stimuli; no difference in PDD or ADHD Dyslexia: fewer sac to infrequent than to frequent stimuli
	10	ADHD-III	8.9 (1.6)	100:0	94 (10)		
	10	Dyslexia	10.0 (1.4)	100:0	96 (7)		
	10	Control	10.7 (1.3)	80:20	98 (9)		

(continued on next page)

Table 5 (continued)

Study	N	Groups	Age <sup>a</sup>	M:F	IQ	Task <sup>b</sup>	Results <sup>c,d</sup>
Van der Geest et al. (2001)	16	10 HFA, 6 NOS	10.9 (2.2)	100:0	98 (16)	Pro-g/o (60 tr./cond), order counterbalanced	Sac RT in gap & overlap: ns
	15	Control	10.3 (1.4)	100:0	97 (10)		Gap effect: PDD < C
Landry and Bryson (2004)	15	13 aut, 2 asp	5.6 (3.8–7.6)	Not rep.	70 (29)	Shift (10 tr.) & disengage (10 tr.), presented in mixed order	Sac RT on Shift: ns
	13	Down syndrome	5.5 (3.5–8.0)		65 (15)		Sac RT on Disengage: PDD > C
	13	Control	3.6 (2.1–6.2) <sup>a</sup>		110 (22)		
Goldberg et al. (2002)	11	HFA	13.8 (1.5)	73:27	99 (11)	Predictive (90 tr.), anti (42 tr.) MGSs (1.5–3 s, 40 tr.), Pro-g/n/o (25 tr./cond)	Express sac on gap: PDD < C
	11	Control	14.4 (1.5)	73:27	113 (14)	Tasks always presented in that order	Antisac errors: PDD > C Premature MGSs: PDD > C Predictive sac: PDD < C Sac RT on gap/null/overlap & MGSs: PDD > C Gap effect: ns RT, amplitude, & peak velocity of predictive sac.: ns RT, velocity, & spatial accuracy of antisac: ns Velocity & spatial accuracy of MGSs: ns
Minshew et al. (1999)	26	HFA	20.2 (8.5)	96:4	105 (13)	Prosac (54 tr.), antisac (36 tr.), MGSs (1, 2, 4, 8 s; 6 tr./delay)	Antisac errors: PDD > C
	26	Control	20.0 (8.7)	96:4	101 (18)	Tasks always presented in that order	Premature MGSs: PDD > C Spatial accuracy of MGSs: PDD < C Peak velocity, duration, RT of all sac: ns
Luna et al. (2007)	61	HFA	8 to 33	Not rep.	111 (17)	Same as Minshew et al. (1999)	Prosac peak velocity: ns
	61	Control	8 to 33	Not rep.	111 (14)		Prosac spatial accurach: PDD < C Prosac and antisac RT: ns RT of memory-guided sac: PDD > C Antisac errors: PDD > C Spatial accuracy of MGSs: PDD < C

Nowinski et al. (2005)	52	HFA	17 (8 to 46)	92:8	106 (13)	Active fixation (15–30 s)	Rate of intrusive sac: ns Intrusive sac in PDD had larger amplitudes & shorter latency to return to fixation than in C
	52	Control	18 (8 to 45)	92:8	109 (12)		
Rosenhall et al. (1988)	11	8 autism 3 “autistic-like”	13 (9–16)	55:45	60–100	Prosac (90 tr.), Pursuit (10, 20, 30 or 40°/s; 12 or more tr./velocity) Order not rep	Sac RT: ns Pursuit: failure to complete task
	26	Control	10 (7–13)				
Scharre and Creedon (1992)	34	Autism	median 7.5 (2–11)	94:6	ave to sev. retarded.	Pursuit (follow a cube moved by experimenter)	Pursuit: only 15% could perform the task well
Takarae et al. (2004)	60	HFA	20.1 (11.2)	88:12	102 (16)	Pursuit (4–32°/s) 3 tasks (32 tr., 40 tr., 22 s; constant) Task order not rep	Initiation gain for targets moving into right visual field: PDD < C; left visual field: ns Maintenance gain on all tasks: PDD < C
	94	Control	19.3 (11.3)	84:16	108 (13)		

*Abbreviations:* ADHD-III, attention/deficit hyperactivity disorder, diagnosed based on DSM-III. Asp., Asperger’s syndrome. Ave. to sev. retarded, average to severely retarded. C, control. HFA, high-functioning autism. MGSs, memory-guided saccades. Ns, not significant. PDD, Pervasive Developmental Disorder. NOS, Pervasive Developmental Disorder not otherwise specified. PDD, Pervasive Developmental Disorder. Pro-g/n/o, prosaccades, with gap, null and overlap conditions. RT, response time. Sac., saccade(s) or saccadic. Tr., trials.

<sup>a</sup> Ages refer to mean ages, numbers within parentheses refer to standard deviation or range, when available. In Landry and Bryson (2004), the control group was significantly younger than the other two groups. However, age was not entered into the analyses. In all other studies, age was reported to not differ significantly between groups, and none of these studies used age as a covariate in the analyses.

<sup>b</sup> Information in parentheses refers to task characteristics, including number and duration of trials. In pursuit studies, the information in parentheses refers to, in order, target speed, whether it was constant or not, and total duration or cycles for the task.

<sup>c</sup> All results refer to those involving group differences.

<sup>d</sup> All participants are referred to as PDD in the results column to ease comparison across studies.

face was emotional rather than neutral. In a second study, participants saw photographs of their family members or friends and of strangers, and decided whether the face familiar. The control group performed at ceiling level, whereas the autistic group was less accurate (84%). Eye tracking results for both studies showed that the duration of fixations on the eyes was shorter in the autistic than in the control group, but that the groups did not differ on fixation time for other regions. Compared to controls, the autistic group showed greater activation in the amygdala in response to the facial stimuli in general and greater activation in both the amygdala and orbitofrontal gyrus for emotional faces in particular. In both studies, duration of time spent fixating the eyes was positively correlated with amygdala activation in the autistic group. The authors suggested that hyperactivation in neural circuits mediating emotions cause “negatively valenced hyperarousal” and heightened sensitivity to social stimuli in autism, which leads to reduced fixations on eyes, which in turn helps to reduce the overarousal.

In a subsequent study, biological siblings of the PDD group were administered the same tasks (Dalton, Nacewicz, Alexander, & Davidson, 2007). The siblings did not meet criteria for PDDs. Results showed that the siblings’ gaze and brain activation patterns were similar to those of the PDD group and significantly different from those of the control group. The siblings also had smaller amygdala volumes than controls. These results suggest that abnormalities in face processing may constitute an endophenotype for PDDs<sup>1</sup>.

In another study (Klin, Jones, Schultz, Volkmar, & Cohen, 2002), adolescents with autism and controls viewed video clips of scenes depicting social interactions. The dependent variables were the number and duration of fixations on the mouths, eyes, body, and objects. Proportion of time fixating the eyes was significantly lower in the autistic (25%) than in the control group (65%), with no overlap between groups. Significant differences were also observed for the other regions, with the autistic group fixating more than controls on the mouths, bodies, and objects. Measures of social competence were positively correlated with fixations on the mouth (suggesting a focusing of attention on speech rather than the social cues from the eyes) and negatively correlated with fixations on objects in the autism group.

Several studies have examined saccades in PDD. In the first study (Kemner, Verbaten, Cuperus, Camfferman, & van Engeland, 1998), children with autism were compared to children with ADHD or reading disorder and controls on a visual oddball task. The high frequency of saccades in the autistic children were interpreted as indicating a weak attentional engagement system. The authors suggested that these results could point to a dysfunction in the frontal eye fields and/or the superior colliculus.

To further test the hypothesis that autism is associated with a weak attentional engagement, another study included a prosaccade task with gap and overlap conditions (Van der Geest et al., 2001). The autistic group showed a smaller gap effect than controls, probably due to slightly faster overlap RTs in the autistic group. The authors attributed this result to a difficulty with engaging attention at fixation in autism and suggested that the difficulty could stem from dysfunction in the superior colliculus or the parietal lobe.

These results were not replicated in another study using a somewhat similar task (Landry & Bryson, 2004) involving shift and disengagement conditions. In this study, children with

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<sup>1</sup> This is a new study that came out since I originally submitted this paper.

PDD or Down syndrome and controls were presented with a central stimulus, followed by a stimulus on the right or the left. On the Shift task, there was a 250-ms gap between the stimuli. On Disengage, the central stimulus remained on screen throughout the trial. The children were instructed “to look at the screens.” The groups did not differ on the shift trials. However, the autistic group showed a disproportionate lengthening of saccadic RTs on the Disengage task, which included their failure to look at the peripheral stimulus throughout the 8-s trial duration on 20% of the trials. Results suggested that the autistic group had difficulty with disengaging attention from fixation.

In another study that included internally and externally guided saccade tasks (Goldberg et al., 2002), adolescents with PDD were compared to controls on gap/null/overlap, anti-saccade, memory-guided and predictive saccade tasks. The pattern of results on these tasks was interpreted as indicating dysfunction in circuits including the dorsolateral prefrontal cortex, and the frontal eye fields, with possible impairments in the parietal cortex, basal ganglia, and cerebellum as well.

In a similar study (Minshew, Luna, & Sweeney, 1999), participants with autism and controls were tested on pro- and antisaccade and memory-guided saccade tasks. Based on the pattern of findings, the authors ruled out impairments in the cerebellum, pons, and superior colliculus, and suggested that the results could be explained by neocortical impairments.

More recently, the same group administered pro-, antisaccade, and memory-guided saccades to a larger sample of participants with and without PDDs from 8 to 33 years of age (Luna, Doll, Hegedus, Minshew, & Sweeney, 2007). In addition to showing generally poor performance on these tasks, the PDD group also showed differences in the developmental trajectories of antisaccade and memory-guided saccade performance. In general, although the control group continued to improve through young adulthood, the PDD group showed less or no improvement.

In a study of fixation (Nowinski, Minshew, Luna, Takarae, & Sweeney, 2005), autistic and control groups were compared on the ability to maintain fixation on central and peripheral stimuli. The autistic group did not show an increased rate of intrusive saccades for central or peripheral targets or an impairment in the extent of foveopetal drift while fixating peripheral stimuli. However, the metrics of their intrusive saccades were different, particularly when fixating the remembered location of the central stimulus. Results were interpreted as indicating an absence of gross cerebellar dysfunction, but a subtle imbalance between excitatory and inhibitory regulation of eye movements in the brainstem, or in the inhibitory cerebellar input to the brainstem affecting the metrics but not frequency of intrusive saccades.

There have been three studies of pursuit in autism. In the first study (Rosenhall, Johansson, & Gillberg, 1988), children were administered pursuit and visually guided saccade tasks. There was no group difference in saccadic RTs. The authors found too much variability in the control group on the pursuit task and failure to complete the task in a majority of the autistic children.

In a second study (Scharre & Creedon, 1992), autistic children were administered a battery of basic visual function tasks, including pursuit. The authors report that only 15% of the children could perform smooth pursuit, and that the remaining children “demonstrated a series of saccadic fixations in place of smooth pursuit movements” (p. 437). The authors report that the children also had high rates of refractive errors, strabismus, and impaired optokinetic nystagmus.

Three pursuit tasks were used in a third study (Takarae, Minshew, Luna, Krisky, & Sweeney, 2004). The autistic group showed reduced gain during initiation, but only for targets moving into the right visual field, pointing to an impairment in left extrastriate areas processing visual motion information (MT/V5) and/or the areas to which they transmit this information. The autistic group also showed reduced gain during maintenance on all tasks, regardless of target direction and speed. The authors also report that reduced gain during maintenance was more apparent for participants over 15 years of age. Results were interpreted as indicating dysfunction in the frontal eye fields, the basal ganglia, and/or the cerebellum.

## ADHD

Difficulties with inhibition and “executive functions” are among the central features of ADHD. Thus, many of the eye tracking studies in children and adolescents with ADHD have compared the integrity of internally versus externally guided saccades, especially antisaccades. These studies are summarized in Table 6. The most consistent finding is that individuals with ADHD make more premature saccades on a number of different tasks and more errors on the antisaccade tasks, reflecting difficulties with inhibition. Peak velocity of saccades was also found to be reduced in two out of three studies. There are inconsistencies across studies in terms of saccadic RTs. Pursuit was tested in three studies (one study by Jacobsen et al. (1996), is summarized in the Section Schizophrenia). Two studies showed reduced RMSE, and one study that included an attentional manipulation designed to enhance performance showed no difference between control and ADHD groups. No impairments were observed in gain in the two studies in which it was measured. Importantly, these studies have highlighted the fact that individuals with ADHD have impairments in relatively lower-level functions (such as peak velocity of saccades, or ability to maintain fixation on a visual stimulus). These results demonstrate that executive functions do not adequately encompass all the deficits observed in this disorder (cf. Willcutt, Doyle, Nigg, Faraone, & Pennington, 2005). Results of studies on medication effects are inconsistent, probably due to selection bias, order effects and small samples. Other factors that should be noted in these studies are that (1) as in studies of PDDs, the majority of the participants were male, (2) studies differed in the extent to which participants had different subtypes of ADHD and comorbid conditions. These subtypes and comorbid conditions were not always reported, and their effects were usually not analyzed.

There have been two studies of pro- and antisaccades in ADHD using relatively large samples. In the largest study of oculomotor function in ADHD, children and adults with ADHD were compared to controls (Munoz, Armstrong, Hampton, & Moore, 2003). Children were between 6 and 16 years of age, and the adults were between 18 and 59. Participants were tested on pro- and antisaccade tasks, with gap and overlap conditions, and a modified prosaccade to assess active fixation (the gap between the fixation and stimulus varied between 0 and 800 ms). The ADHD groups performed worse than controls on most indices of oculomotor function in both tasks. In addition, performance on both the antisaccade and prolonged fixation tasks improved through adolescence, reaching adult levels only at age 16 (see Fig. 10). The developmental trajectory of the ADHD group lagged behind that of controls for both tasks and reached an asymptote later. There was also a great deal of variability in the ADHD group on antisaccade errors. Results were inter-

Table 6  
Eye tracking studies in ADHD

Study	N	Groups	Age <sup>a</sup>	M:F	IQ (or est'd IQ)	Task <sup>b</sup>	Results <sup>c,d</sup>
Mokler and Fischer (1999) <sup>e</sup>	76	ADHD	10.3 (0.3)	80:20	Not rep.	Pro-g/o (80–120 tr.), anti-g/o (160–240 tr.) active fixation (300–450 tr.) Pro- & antisac administered in first session, fixation in second. Prosac always followed by anti	Prosac: RT, CV of RT, duration: ADHD > C
	75	Control	10.7 (0.3)	53:47	Not rep.		Prosac RT: peak velocity: ADHD < C
	38	ADHD	31.0 (1.4)	47:53	Not rep.		Gap effect: ns
	105	Control	34.1 (1.1)	44:56	Not rep.		Prosac: proportion of express saccades: ns Antisac: errors, RT, CV of RT: ADHD > C Active fixation: intrusive sac: ADHD > C
Klein et al. (2003) <sup>f</sup>	46	ADHD	11.3 (7–15)	89:11	102 (11)	Pro-g/o, anti-g/o (100 tr./condition) Order counterbalanced	Prosac and antisac RTs: ADHD > C
	46	Control	11.3 (7–15)	83:17	106 (16)		Premature sac on all tasks: ADHD > C Antisac errors: ADHD > C Corrective sac on antisac task: ADHD < C Express sac in pro-g: ADHD < C
Rothlind et al. (1991)	20	ADHD-III-R	10.5 (6.9–13.9)	100:0	99 (10)	Pro-null/o, anti-null/o (10 tr./condition)	Pro- and antisac RTs: ns
	21	Control	9.9 (2.8)	100:0	111 (13)		Antisac errors: ns Asymmetry in sac RTs in both tasks in C, but not ADHD
Mostofsky et al. (2001b)	11	TS + ADHD-III-R	11.7 (7.8–14.3)	100:0	Not rep.	Pro-& antisac (60 tr./condition), MGSs (4.5–5 s; 60 tr.) Task order not rep	Prosac RT: TS, TS + ADHD > C
	14	TS	10.8 (8.4–14.6)	100:0	Not rep.		Prosac RT variability: TS + ADHD > TS, C
	10	Control	10.6 (8.1–12.6)	100:0	Not rep.		Antisac errors and premature MGSs: TS + ADHD > TS
Habeych et al. (2006) <sup>g</sup>	12	At risk + ADHD	(10–12)	Not rep.	Not rep.	Anti-g/n/o (99 tr./cond) Task order not rep	Antisac errors on gap & null: at risk + ADHD > at-risk
	55	At risk	(10–12)	67:33	111 (12)		Antisac RTs in null: at risk + ADHD > at risk
	12	Control	11.2 (10–12)				Antisac RTs in gap & overlap: ns Peak velocity of antisac in null: At risk + ADHD > at-risk Peak velocity of antisac in gap & overlap: ns

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Table 6 (continued)

Study	N Groups	Age <sup>a</sup>	M:F	IQ (or est'd IQ)	Task <sup>b</sup>	Results <sup>c,d</sup>
Castellanos et al. (2000)	32 ADHD-C	8.8 (6–13)	0:100	Not rep.	Pursuit (17°/s), constant 5 cycles go/no-go (12 tr./condition), MGSs (1.2 s, 13 tr.) Tasks always presented in this order	Frequency of MGSs: ADHD < C Premature MGSs: ADHD > C Commission errors to no-go stimuli: ADHD > C Intrusive saccades in go/no-go: ADHD > C Spatial accuracy of MGSs: ADHD < C (trend) Anticipatory & catch-up sac in pursuit: ns Maintenance gain in pursuit: ns RMSE in pursuit: ADHD > C ( $p = .09$ )
	20 Control	9.6 (1.7)	0:100	Not rep.		
Gould et al. (2001)	24 ADHD-C	10.0 (2.0)	100:0	Not rep.	Active fixation (21 s)	Large (>4°) sac: ADHD > C Floor effects, poor test–retest reliability
	29 ADHD-C	8.8 (1.6)	0:100	Not rep.		
	26 Control	10.3 (1.5)	100:0	Not rep.		
	18 Control	9.4 (1.7)	0:100	Not rep.		
Cairney et al. (2001)	13 ADHD-C	8.2 (1.7)	77:23	99 (15)	Contextual modulation of sac (200 sac tr., 200 catch tr.)	FOE for high-probability targets: ns FOE for low-probability targets: ADHD < C Premature & inappropriate sac: ADHD > C
	15 ADHD-C	8.9 (1.9)	93:7	96 (12)		
	15 Control	8.5 (6–11)	87:13	101 (12)		
	15 Control	25.5 (21–38)	40:60	Not rep.		
Karatekin (2006)	10 ADHD-C	14.3 (12–18)	80:20	12 (2) <sup>h</sup>	Pro- and antisac, modified antisac tasks (32 tr./task) Anti always followed by pro; order of antisac tasks counterbalanced	Prosac RT: ns Antisac errors: ADHD > age-matched & younger C Antisac RT on 1st administration: ADHD > age-matched C Antisac RT on 2nd administration: ns Premature sac: ADHD > age-matched & younger C Corrective sac: ADHD < age-matched & Younger C
	15 Control	15.0 (11–19)	60:20	13 (2)		
	15 Control	10.3 (9–11)	47:53	14 (3)		
	18 Control	19.2 (18–20)	11:89	12 (2)		
Ross et al. (1994a, 1994b)	13 ADHD-III-R	11.2 (1.3)	100:0	107 (12)	MGSs (800 ms, 31.5 s)	Premature MGS: ADHD > C Sac RTs: ns Spatial accuracy of MGS: ns
	10 Control	11.5 (1.0)	50:50	116 (16)		
Aman et al. (1998)	22 ADHD <sup>i</sup>	12.1 (1.2)	100:0	111 (8)	Modified prosac (42 tr.) & antisac (42 tr.) Pro always followed by anti	Prosac errors: ns Antisac errors: ns
	22 Control	12.1 (1.2)	100:0	110 (7)		



Mostofsky et al. (2001a) <sup>j</sup>	8 ADHD 11 ADHD 25 Control	10.8 (7.2–17.9)	52:48	Not rep. Not rep. Not rep.	Prosac (60 tr.), antisac (60 tr.), MGSs (4.5–5 s, 60 tr.) Task order not rep	Antisac errors: ADHD (med = unmed) > C Premature MGS: ADHD (med = unmed) > C Prosac RTs: ns CV of prosac RTs and RT of MGSs: ADHD (unmed) > ADHD (med) = C
O'Driscoll et al. (2005)	10 ADHD-C 12 ADHD-I 10 Control	12.4 (0.6) 12.7 (0.6) 12.7 (0.6)	100:0 100:0 100:0	108 (9) 109 (11) 110 (11)	Prosac (48 tr.), antisac (48 tr.) predictive (tr not rep) task switch (48 tr.) Order counterbalanced	RT, amplitude, peak velocity of prosac: ns Predictive saccades for predictable direction: ns Predictive saccades for predictable direction & timing: ADHD-C < ADHD-I = C Antisac errors: ADHD-C > C; ADHD-I between ADHD-C and C and did not differ significantly from either Antisac RTs: ns Task switching: ns
Bylisma and Pivik (1989)	20 ADHD-III-R 20 Control	9.6 (1.7) 9.5 (1.7)	85:15 55:45	Not rep. Not rep.	Pursuit (.45 Hz or 29°/s, sinusoidal, 15–20 oscillations; attentional manipulation)	Velocity arrest scores: ADHD > C RMSE: ns
<i>Effects of medications</i>						
Ross et al. (1994a, 1994b)	13 ADHD-III-R 10 Control	11.2 (1.3) 11.5 (1.0)	100:0 50:50	107 (12) 116 (16)	MGSs (800 ms, 31.5 s) ADHD tested on placebo & methylphenidate, in random order, with 1 week in between Controls also tested twice	No effect of methylphenidate on MGSs
Aman et al. (1998)	22 ADHD <sup>i</sup> 22 Control	12.1 (1.2) 12.1 (1.2)	100:0 100:0	111 (8) 110 (7)	Modified prosac (42 tr.) & antisac (42 tr.) Pro always followed by anti ADHD tested on psychostimulant med first, off med second Controls also tested twice. Inter-session interval: 1 week	Correct prosac: close to perfect in both groups in both sessions Antisac errors: decreased in controls from 1st to 2nd session; no difference in ADHD from 1st (med) to 2nd (unmed) session

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Table 6 (continued)

Study	N	Groups	Age <sup>a</sup>	M:F	IQ (or est'd IQ)	Task <sup>b</sup>	Results <sup>c,d</sup>
Mostofsky et al. (2001a) <sup>j</sup>	8 11 25	ADHD ADHD Control	10.8 (7.2–17.9)	52:48	Not rep. Not rep. Not rep.	Prosac (60 tr.), antisac (60 tr.), MGSs (4.5–5 s, 60 tr.) Task order not rep Children were not assigned randomly to medication	No difference between med & unmed ADHD in prosac RTs, antisac errors & premature MGSs CV of prosac RTs and RT of MGSs: med ADHD > unmed ADHD
O'Driscoll et al. (2005)	10 12 10	ADHD-C ADHD-I Control	12.4 (0.6) 12.7 (0.6) 12.7 (0.6)	100:0	108 (9) 109 (11) 110 (11)	Prosac (48 tr.), antisac (48 tr.) predictive (tr not rep) task switch (48 tr.) Order counterbalanced Double-blind cross-over trial, ADHD tested once at baseline, Then on placebo & methylphenidate, in counterbalanced order, with 3 at least weeks in between Controls tested once	Methylphenidate led to faster pro- and antisac RTs, more predictive sac, fewer antisac errors, fewer task switching errors
Klein et al. (2002)	27	ADHD-C	12.6 (10–15)	100:0	Not rep.	Pro-g/o, anti-g/o(tr and order reported elsewhere) Children tested on and off methylphenidate in Counterbalanced order, with 1 week in between	Methylphenidate led to shorter pro- & antisac RTs, fewer antisac errors, more corrective antisac, shorter RTs for corrective sac, more express sac Interaction between medication & order effects: When tested on placebo first and med second, performance improved on most measures. When tested on med first, little difference between sessions Only two measures showed a main effect of medication but no medication × order interaction: children made more express prosac and corrected their antisac errors more frequently when on medication

Bylisma and Pivik (1989)	20 ADHD-III-R 20 Control	9.6 (1.7) 9.5 (1.7)	85:15 Not rep. 55:45 Not rep.	Pursuit (.45 Hz or 29°/s, sinusoidal, 15–20 oscillations; attentional manipulation) Controls tested once, ADHD tested twice, on and off methylphenidate, with 1 week in between	No effect of methylphenidate on velocity arrest scores or RMSE Velocity arrests: Med ADHD = C
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*Abbreviations:* ADHD, Attention-deficit/hyperactivity disorder, subtype not specified. ADHD-III-R: Attention-deficit/hyperactivity disorder, diagnosis based on DSM-III-R, which did not specify subtypes. Anti-g/n/o: antisaccade task with gap, null (no gap), and overlap conditions. C, control. Med, medicated or medications. MGSs, memory-guided saccades. Ns, not significant. RT, response time. Sac., saccade(s) or saccadic. Tr., trials. TS, Tourette's syndrome. Unmed, unmedicated.

<sup>a</sup> Ages refer to mean ages, numbers within parentheses refer to standard deviation or range, when available. Age was used as a covariate in Mostofsky et al. (2001a) and Klein et al. (2003).

<sup>b</sup> Information in parentheses refers to task characteristics, including number and duration of trials. In pursuit studies, the information in parentheses refers to, in order, target speed, whether it was constant or not, and total duration or cycles for the task.

<sup>c</sup> All results refer to those involving group differences.

<sup>d</sup> Studies examining the effects of medications are listed twice. In the first section, results refer to the unmedicated state to facilitate comparisons across studies. In the second section, details of the design relevant to medications are added, and only the medication effects are listed under Results.

<sup>e</sup> The control group overlapped with that used in Munoz et al. (1998).

<sup>f</sup> All of the controls in the study were included in Klein (2001).

<sup>g</sup> Children were at risk for substance use disorders by virtue of having a father with a lifetime diagnosis of alcohol abuse or dependence. ADHD was diagnosed based on questionnaires filled out by the mother and child. The ages, gender ratios, and IQs of the at-risk children with and without ADHD were not reported separately. Average age for the at-risk group was 11.0 years (range = 10–12), M:F gender ratio was 55:45, and average IQ was 108 ( $SD = 17$ ).

<sup>h</sup> IQ was estimated from Vocabulary subtest scores.

<sup>i</sup> 73% of the sample had the Combined subtype, 18% had the Inattentive subtype, and 9% had the Hyperactive/Impulsive subtype.

<sup>j</sup> The ages, gender ratios and subtypes of the medicated and unmedicated children were not reported separately. Mean age of the whole ADHD group was 11.3 years (range = 7.1–16.1), M:F ratio was 58:42, 32% were diagnoses with the Combined subtype, and 42% with the Inattentive subtype. Twenty-six percent of the sample could not be assigned a subtype due to discrepancies between parent and teacher reports.

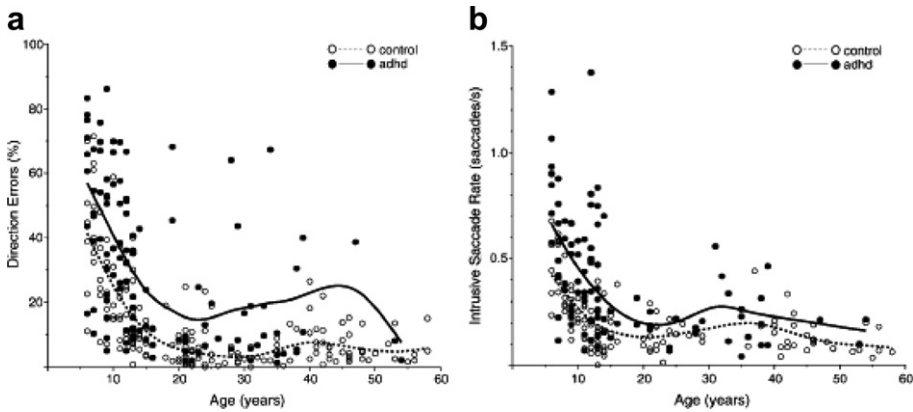


Fig. 10. (a) Antisaccade errors and (b) intrusive saccades during fixation as a function of age in individuals with ADHD and healthy controls (Munoz et al., 2003). Reprinted with permission of the American Physiological Society.

preted as indicating difficulty with suppressing inappropriate saccades and generating volitional saccades, consistent with dysfunction in the prefrontal cortex and/or basal ganglia.

In another study as well (Klein, Raschke, & Brandenbusch, 2003), children with ADHD showed impairments on most indices of oculomotor function compared to controls. The researchers also examined group differences in developmental trends. In controls, both pro- and antisaccade RTs declined with age, although the decline in antisaccade RTs was steeper. In the ADHD group, however, the functions relating age to RT had similar slopes on the two tasks. Furthermore, although the frequency of premature responses decreased with age in both groups, the rate of this reduction was slower in the ADHD group. Results pointed to a dysfunction in ADHD in the frontal eye fields, resulting in delays in release from fixation and impairments in visual attention, as well as in the prefrontal cortex.

An earlier study of pro- and antisaccades with null and overlap conditions (Rothlind, Posner, & Schaughency, 1991) found few differences between ADHD and control groups on either task, although the small number of trials (10) makes it difficult to draw firm conclusions about the negative findings.

In two other studies, ADHD was examined as a secondary condition. In one of these, boys with Tourette syndrome with or without comorbid ADHD were compared to controls on pro-, antisaccade, and memory-guided saccade tasks (Mostofsky, Lasker, Singer, Denckla, & Zee, 2001b). The performance of the boys with both Tourette's and ADHD was worse than that of boys with Tourette only on most measures. However, prosaccade RTs were elevated in boys with Tourette's, regardless of comorbidity. In addition, group differences in variability of prosaccade RTs were more apparent in boys younger than 10 than in boys older than 10. In another study of antisaccades (Habeych, Folan, Luna, & Tarter, 2006), the focus was on at-risk children of fathers with lifetime diagnoses of alcohol abuse or dependence compared to children of fathers without this diagnosis. Within the at-risk group, 18% were diagnosed with ADHD. Results showing that at-risk children with ADHD performed worse than at-risk children without ADHD were interpreted as indicating that the ADHD children had difficulties with inhibitory control of behavior.

Castellanos and Colleagues (2000) compared girls with ADHD to controls on a memory-guided saccade task, an oculomotor go/no-go task, and a pursuit task. The girls with ADHD showed impairments on most saccade measures, but few impairments on pursuit. Fifteen of the ADHD girls were retested on the go/no-go task 3–9 weeks later while they were receiving placebo during a medication trial. Results showed improvements in commission and intrusion errors. However, as the controls were not retested, these effects could not be compared across groups. Test–retest reliabilities for the measures were not reported. Results were interpreted as indicating deficits in inhibition and working memory in ADHD.

In a study of active fixation (Gould, Bastain, Israel, Hommer, & Castellanos, 2001), girls (who had also taken part in Castellanos et al., 2000) and boys with ADHD were compared to controls on an active fixation task. The ADHD group made more large-amplitude intrusive saccades away from fixation than controls. However, there was a significant floor effect in all groups, and test–retest reliability was very poor. The authors concluded that children with ADHD have difficulty maintaining fixation, which may be related to frontal-striatal dysfunction, but that the measure was not stable over time.

Another investigation (Cairney et al., 2001) focused on a task assessing ability to use contextual information to modulate oculomotor responses. In this task, a target appeared on 20% of the trials in one condition, and on 80% of the trials in another condition. Both conditions included gap and overlap trials. The ability of the participants to utilize contextual information was inferred from the magnitude of the fixation offset effect (FOE), the difference between RTs on gap and overlap trials. FOE had been found to be smaller for high- compared to low-probability targets: in the high-probability condition, participants can prepare saccades based on the contextual information, which diminishes the advantage of the gap in reducing RTs. There was no difference in the magnitude of the FOE between control children and adults and no difference in the magnitude of the FOE for high-probability targets between the control and ADHD children. However, children with ADHD failed to show a larger FOE effect for low-probability targets. Further analyses indicated that the ADHD children were able to utilize the contextual information, but that they did not modulate their FOE effectively because the overlap condition did not lead to the normal increase in saccadic RT in ADHD. In addition, the frequencies of premature and inappropriate saccades were higher in control children than in adults, and higher in ADHD than in the control children. These results were interpreted as indicating that the ADHD group “had difficulty inhibiting saccades only when they were required to use context to increase the level of tonic inhibition within their saccadic system. Thus, the presence of inhibitory deficits in ADHD depends upon the context in which the individual’s current behavioral goals are set” (p. 516). The authors further suggested that the difficulties of the ADHD group may be related to dysfunction in the frontal eye fields, which modulate the activity of fixation cells in the superior colliculus.

In a recent study (Karatekin, 2006), I examined the effects of task manipulations on improving antisaccade accuracy and RTs of adolescents with ADHD, age-matched controls, 10-year-olds and young adults. Order effects were tested by administering the task at the beginning and end of the session. Other task manipulations involved a visual landmark to reduce demands on working memory and internal generation of saccades; spatially specific and non-specific cues at three intervals (200, 600, 1000 ms); and central engagement of attention through perceptual and cognitive means at three intervals (200, 600, 1000 ms). As expected, adolescents with ADHD were impaired relative to controls

in terms of accuracy and saccadic RT on the first administration of the task. Although their accuracy improved with most of the manipulations, it did not improve disproportionately compared to controls. Nevertheless, with most of the manipulations, they achieved the same level of accuracy as unaided controls on the first administration of the task. In contrast, the saccadic RTs of the ADHD group came close to normal under several conditions, indicating that elevated antisaccade RTs in this disorder may be related to attentional factors. The ADHD group made more premature saccades and fewer corrective saccades than both the age-matched and younger groups, suggesting difficulties with impulsivity and goal neglect. The findings suggested that cognitive scaffolds can ameliorate at least some of the inhibition deficits in adolescents with ADHD.

A number of studies have examined the effects of medications on oculomotor performance in ADHD (summarized in Table 6). In a study on the effects of methylphenidate on memory-guided saccades in ADHD (Ross et al., 1994a, Ross, Radant, Young, & Homer, 1994b), the ADHD group was tested on both methylphenidate and placebo, with order assigned randomly. The control group was also tested twice, and the inter-session interval was 1 week. There were no order effects in controls and no medication effects in the ADHD group. As a result, data were collapsed across these variables. The only significant result was that children with ADHD made more premature saccades than controls, which was interpreted as indicating that the ADHD children had difficulties in inhibiting information held in working memory.

In another study on the effects of psychostimulant medications on pro- and antisaccades (Aman, Roberts, & Pennington, 1998), the performance of boys with ADHD was examined while they were on and off medication, and compared to that of controls boys who were also tested twice. The antisaccade task differed from the traditional version in that the cue was presented for 100 ms, followed by a 400-ms gap. Next, a three-sided square was presented on the opposite side of the cue for 150 ms. Participants indicated which side of the square was open. Unfortunately, medication status was confounded with testing order, as all the boys were tested on medication first and off medication second. Whereas antisaccade performance improved with time in controls, the ADHD children showed no difference between the first (on medication) and second sessions (off medication).

In another study (Mostofsky, Lasker, Cutting, Denckla, & Zee, 2001a), saccadic eye movements of eight children who were already on methylphenidate were compared to those of 11 children not taking medications and to controls. Based on the findings, the authors ruled out dysfunction in the posterior parietal cortex, and frontal and supplementary eye fields. Instead, the deficits observed on the antisaccade and memory-guided saccade tasks were interpreted as reflecting deficits in prefrontal-striatal circuitry in ADHD, particularly in the dorsolateral prefrontal and/or inferior prefrontal regions. In addition, they suggested that methylphenidate increases consistency of motor responses. As noted by the authors, however, the children were not randomly assigned to medication status, resulting in selection bias.

In another study, a double-blind cross-over trial design was used to assess the effects of methylphenidate in boys with ADHD (O'Driscoll et al., 2005). The tasks included pro- and antisaccades, predictive saccades, and task switching (where pro- and antisaccades were presented in mixed order). All participants were tested at baseline while unmedicated. The ADHD boys were then retested twice, on and off medication in counterbalanced order, with a retest interval of at least 3 weeks. Compared to placebo, the medication

condition led to better performance on all tasks. The authors further reported that although antisaccade errors declined with age in the control group ( $r = -.83$ ), who ranged in age between 11.5 and 14, correlations were not significant in either the Inattentive ( $r = -.20$ ) or the Combined subtypes ( $r = -.02$ ). The authors hypothesized that the deficits observed in the antisaccade and predictive saccade tasks could be related to dysfunction in the cerebellum and frontal eye fields.

In another study of methylphenidate designed to address questions regarding order effects (Klein, Fischer, Fischer, & Hartnegg, 2002), boys with ADHD were tested on pro- and antisaccade tasks. All boys were tested on and off medication, with an inter-session interval of 1 week, and testing order was counterbalanced. Although medication improved performance, there were interactions between test order and medication status. In general, when children were tested on placebo first and medication second, their performance improved on most of the measures on both pro- and antisaccade tasks. In contrast, when they were tested on medication first, and placebo second, there was little difference between the two sessions. Only two measures showed a main effect of medication but no medication  $\times$  order interaction: the children made more express saccades on the prosaccade task and corrected their errors more frequently on the antisaccade task when on medication than when on placebo. Results were interpreted as indicating that methylphenidate weakens the fixation system but strengthens voluntary control over saccades, and that control for order effects is essential in studies of medication effects.

Finally, an earlier study examined the effects of methylphenidate on pursuit in children with ADHD and controls (Bylsma & Pivik, 1989). To enhance attention, the children were instructed to press a button whenever the pursuit target light disappeared briefly from view. The effects of administering the task in the light versus dark were also investigated. The controls were tested once, and the ADHD group was tested twice, on and off medication. Performance on pursuit was assessed by velocity arrest scores (based on time periods when gaze velocity was less than  $2^\circ/\text{s}$ ) and RMSE. Although the unmedicated ADHD group had higher velocity arrest scores than controls, their RMSE did not differ from those of controls. Methylphenidate did not have a significant effect on either measure within the ADHD group; however, the medicated group did not differ from controls in terms of velocity arrests.

### **Critique of studies of eye tracking in normative and atypical development**

The main goals of the saccade and pursuit studies in typical development have been to chart the developmental trajectories of different indices of performance and to examine the effects of several factors (especially gap and overlap conditions in saccades and target velocity in pursuit) in children versus adults. The studies of atypical development have focused on delineating impairments in disorders and making inferences about the neural bases of these impairments.

At a global level, there are some impressive consistencies across these studies. These replications across different labs and samples clearly demonstrate the reliability of the findings. In normative development, the consistent findings include the observations that the dynamics of prosaccades (peak velocity, duration) do not change substantially after age 4, that prosaccades and antisaccades have different developmental trajectories, and that pursuit gain continues to improve through adolescence for fast targets. In atypical development, the most consistent findings are pursuit impairments in schizophrenia, elevated

antisaccade error rates in ADHD and PDDs, and premature saccades on a variety of tasks in schizophrenia, ADHD, and PDDs.

Eye tracking studies also reveal the extent to which impairments are specific to one disorder or are common across disorders. For example, an impairment in smooth pursuit is a very robust finding in youth-onset schizophrenia. In contrast, evidence for smooth-pursuit abnormalities is less strong in individuals at genetic risk for schizophrenia, and in individuals with PDD or ADHD. There is strong evidence for abnormalities in scan patterns in both schizophrenia and PDD, but not in ADHD. On the other hand, premature saccades and impairments on antisaccades do not show specificity to these three disorders. More studies directly comparing these disorders would provide valuable information on the extent of specificity.

Among the most valuable contributions of eye tracking studies are the fact that they allow researchers to examine different aspects of performance in detail on relatively simple tasks that can be completed by young children or children with intellectual or motoric limitations, and to make inferences about the neurobiological bases of their performance. For this reason, eye tracking measures are used as endophenotypes in schizophrenia in studies designed to identify the genetic bases of the disorder. Eye tracking measures could serve the same function in ADHD and PDD, although there are as yet no published studies of children and adolescents examining the utility of eye tracking measures as endophenotypes in ADHD or PDD.

Very few studies reviewed in this paper have examined the functional correlates of eye movement measures. However, the study on PDD by [Klin et al. \(2002\)](#), in which eye movements to video clips depicting social scenes were found to be related to measures of social competence, demonstrates the potential of these types of analyses.

At a more detailed level, though, there are also some discrepancies across the studies reviewed in this paper. For instance, different studies have come to different conclusions about the precise developmental trajectories of pro- and antisaccades and pursuit gain. In addition, there are discrepancies in terms of intrusive and compensatory saccades in pursuit, express saccades and the gap effect, and spatial accuracy of memory-guided saccades. Some of these discrepancies are due to idiosyncratic factors that are not of general concern. Some of the reasons, however, may be relevant to the field of developmental cognitive neuroscience in general.

(1) Charting “the” developmental trajectory of performance on a task may not be a feasible goal. As with many other tasks (e.g., [Kagan, 2003](#)), performance on eye tracking tasks is a function of not only task difficulty, but also of contextual factors, including the task parameters and state variables such as anxiety and fatigue. For example, introducing a 200-ms gap or overlap between fixation and stimulus on a prosaccade task substantially changes the nature of the resulting saccades. As a result, different developmental trajectories are obtained for the gap versus the overlap versions of prosaccade task ([Klein, 2001](#)). Different trajectories are also obtained in pursuit studies depending on the target velocity ([Accardo et al., 1995](#)) and in face perception depending on whether the stimuli are schematic drawings or photographs ([Schwarzer et al., 2005](#)). The precise wording of the instructions (“follow the lights” vs. “move your eyes in time with the lights”) affects the characteristics of predictive saccades in adults ([Isotalo, Lasker, & Zee, 2005](#)), and the saliency of task instructions affects the manifestation of antisaccade impairments in clinical populations of adults ([Nieuwenhuis, Broerse, Nielen, & de Jong, 2004](#)). The characteristics and neural bases of prosaccades differ depending on whether they are presented



in blocks or intermixed with antisaccades (Cornelissen et al., 2002). In a large sample of “healthy” adults, levels of anxiety and depression as measured on a checklist, affect antisaccade performance (Smyrnis et al., 2003).

Like other cognitive tasks, performance on eye tracking tasks is also tied to temporal factors unfolding over short periods of time. For instance, studies in adults show time-on-task (Smyrnis et al., 2002) and practice effects (Dyckman & McDowell, 2005) on antisaccades. In ADHD, elevated antisaccade RTs come down to normal levels when the task is re-administered (Karatekin, 2006).

In this context, it is important to note that one of the factors that varies quite a bit across the studies reviewed above is task length. Estimating the effect of task length on performance is not easy. Of course, an inadequate number trials reduces the reliability of the measure. In the studies reviewed above, only Malone and Iacono (2002) have addressed this issue statistically. In addition, when a task includes relatively few trials, it is likely that it is tapping not only the specific construct it is purported to tap, but also the ability of the participants to adapt to a novel situation. Shorter tasks are also less likely to induce fatigue. Longer tasks, on the other hand, assess participants’ ability to improve their performance with practice (learning to learn), which may differ depending on age and clinical status. For instance, in their study of a 100-trial antisaccade task in ADHD, Klein et al. (2003) conducted post-hoc analyses to examine the effect of task length. They report that differences in antisaccade errors between the ADHD and control groups were larger for the whole task and for the second 50 trials than for the first 50 trials. Whereas the error rate declined slightly in the control group from the first to the second half, they increased slightly in the ADHD group. Furthermore, this increase in error rate was more pronounced in the younger than in the older ADHD participants. Thus, the effect of task length may differ depending on the nature of the task as well as the age and clinical status of the participants. It is, therefore, disconcerting to see that task length does not differ randomly across these studies—in general (and for good reason), tasks tend to be shorter in studies that include younger children or clinical populations of children. It is also disconcerting that order effects were not controlled for and that task order was not even reported in a number of the studies reviewed in this paper.

It is clear that what is being measured on these eye tracking tasks cannot be viewed as unitary, static constructs that exist independently of context and time. Not only do contextual and temporal factors affect performance, but they also interact with age and clinical status. Thus, it seems like an illusory goal to try to chart “the” developmental trajectory of a “pure” process and to pinpoint the age at which performance reaches maturity. Instead, it might be more informative to incorporate contextual and dynamic factors into the definition of the constructs and the design of the studies and to collect more empirical evidence on the factors that facilitate or hinder performance as a function of age and clinical status (cf. Karatekin, 2006; Klein, 2001; Klein et al., 2003; Nieuwenhuis et al., 2004). This approach could also make it easier to compare results across studies and allow for stronger inferences.

(2) Another reason for the discrepancies across studies has to do with the method of analyzing age-related changes in normative studies and the method of addressing developmental issues in clinical studies. In some normative studies, participants are grouped into ages (sometimes spanning 1–2 years, sometimes 5–6 years), and ANOVAs are used to compare groups. Lack of differences between age groups is interpreted as indicating lack of developmental change, although these negative findings could also be attributed to lack

of statistical power due to small and/or unequal sample sizes across age groups. On the other hand, positive findings could, in some cases, be due to the fact that researchers based their conclusions on visual inspection of the data or did not correct for multiple comparisons. In other cases, researchers use a correlational approach, which assumes a linear relationship between age and the dependent variable. With a correlational approach, the focus tends to be on whether the process is related to age across the whole age range under study. In other cases, investigators test different developmental functions against each other to determine which function provides the best fit with the data and at which age performance asymptotes. A regression approach is more suitable than an ANOVA or a simple correlational approach when the underlying process is assumed to change in a continuous, quantitative and non-linear manner with age (Klein, Foerster, Hartnegg, & Fischer, 2005). Although these approaches are more appropriate for examining developmental questions, they lose sight of the information regarding age-related changes in variability. Thus, the conclusion that is drawn is that children are initially poor at a process and gradually improve in a linear or non-linear fashion up to a point, whereas visual inspection of the scatterplots indicates that there is initially a great deal of variability, with some children performing at a high level even at young ages. Statistical methods that provide confidence intervals for the shape of the developmental trajectory or age ranges for adult-like performance (cf. Van Geert, 2002) rather than “the” age at which performance reaches maturity would be more informative than methods that only focus on the shape of the distribution. These methods might also make it easier to distinguish between normal and abnormal variability in studies of atypical development.

Thus, for the same data set, it is possible to conclude that performance matures earlier if an ANOVA is used or that it continues to develop through that age range if a correlational or regression approach is used. This discrepancy can be clearly seen in the study by Scherf et al. (2006) in which both an ANOVA and inverse-curve fit were used to analyze saccadic data, and different conclusions emerged concerning age-related changes from the different statistical methods.

There are also inconsistencies in clinical studies regarding methods of addressing age differences across groups. First, the width of the age range varies substantially across studies, as can be seen easily in Tables 4–6. Second, there are age differences across groups in some studies. However, when the sample sizes are small, these differences do not reach significance. Thus, the groups are reported to not differ in age, and age is not considered further in the analyses. The developmental studies, however, indicate that depending on the age range and task, seemingly small age differences can, in fact, affect the results. For instance, Klein et al. (2003) note that in a previous study (Rothlind et al., 1991) in which an ADHD sample was found to make more antisaccade errors than controls, “patients were 8 months younger than controls (125 months old). In our sample of 199 participants aged 6–28 years, we found that this age difference alone can explain [antisaccade] errors” (p. 26).

Third, some studies use age as a covariate when comparing clinical to control groups. This approach assumes that age and the dependent variable are linearly related in both groups, and that the strength of the relationship does not differ across groups. However, in only two of the studies reviewed above (Klein et al., 2003; Ross et al., 2005) were these assumptions explicitly tested.

(3) As with most other measures, the shape of the developmental trajectory and the extent of differences between clinical and control groups depend on the psychometric char-

acteristics of the measures, including their variability, reliability, discriminating power, and whether they are subject to ceiling or floor effects (e.g., Knight & Silverstein, 2001; Meier & Perrig, 2000; Miller, Chapman, Chapman, & Collins, 1995). Given two measures purported to assess the same construct, the measure with greater reliability and discriminating power is more likely to show age-related or clinical differences. The problem becomes thornier when different age or clinical groups are compared on tasks with different psychometric properties assessing different constructs (e.g., antisaccades and memory-guided saccades). In these cases, psychometric properties are confounded with what is being measured, and interpretations of differential developmental trajectories and differential deficit become suspect. The reliability of the measures also affects the extent to which they are inter-correlated, complicating interpretations of factor analyses and correlational and regression analyses to examine the interdependence of cognitive processes.

Thus, to interpret normative and clinical data, it is important to be aware of the psychometric properties of these measures and to test if they differ as a function of age or clinical status. Reliability of eye tracking measures was examined in several of the studies reviewed above. Internal consistency (measured with Cronbach's  $\alpha$ ) of antisaccade errors on a 20-trial task in a large sample of healthy children was found to be .81 for 11-year-olds and .82 for 17-year-olds (Malone & Iacono, 2002). Intra-class correlations (ICCs) for split-half reliability of RMSE in a pursuit task in adolescents with schizophrenia-spectrum disorders and controls ranged from .75 to .96 (Kumra et al., 2001). Test-retest reliability (measured with ICCs) over 3–6 weeks in a sample of 22 children with ADHD was .79 for antisaccade errors and .62 for predictive saccades for which both the direction and timing of the target were predictable (O'Driscoll et al., 2005). On the other hand, test-retest reliability of intrusive saccades during active fixation (measured through Pearson  $r$ ) was only .16 over 3–9 weeks in 23 girls with ADHD, which probably reflects the floor effects on this measure (Gould et al., 2001).

In the largest developmental study of reliability of saccadic measures in children, internal consistency and split-half reliability of pro- and antisaccades were computed with the Pearson  $r$  in 327 healthy 9- to 88-year-olds (Klein & Fischer, 2005b). With age partialled out, instrumental reliabilities were high for pro- and antisaccade RTs (.91–.96 for odd–even reliability, .81–.90 for split-half reliability), and antisaccade errors (.95 for odd–even, .83 for split-half). Test-retest reliability over 19 months was computed only for 6- to 18-year-olds. Reliability estimates were moderately high for pro- and antisaccade RTs (.65–.66), but low for antisaccade errors (.43), perhaps due to individual differences in the rate of maturation of the neural substrates of antisaccades. The authors further noted that the instrumental (within-session) and test-retest reliability estimates were not affected by age.

These issues regarding the method of data analysis, lack of statistical power, and the psychometric properties of the tasks matter for making inferences about the integrity and maturation of brain-behavior relations. These inferences are based not only on the positive findings, but on the total pattern of positive and negative findings considered jointly. If enough confidence cannot be placed in the negative findings, one cannot interpret the positive findings with much confidence, either.

In addition to the reasons for discrepancies across studies, it is also important to note that all of the developmental studies reviewed in this paper have used a cross-sectional approach. The risks of drawing inferences about development from cross-sectional data are well-documented (e.g., Kramer, Yesavage, Taylor, & Kupfer, 2000; Schneider, Kron,

Hünnerkopf, & Krajewski, 2004; Siegler, 1998). For instance, the data reviewed above point to the conclusion that development of pro- and antisaccades occurs in a gradual, non-linear manner through middle childhood and adolescence, whereas a different picture could well emerge in a longitudinal study.

The problems associated with making inferences about development from cross-sectional data are compounded in clinical samples, where age can easily be confounded with other variables, such as severity, gender, comorbidity, confidence in the diagnosis, and duration of pharmacological treatment. Some of these problems could be overcome if researchers provide more information on these variables when comparing different age groups. Otherwise, caution needs to be exercised when making inferences about differences in developmental trajectories between clinical and control groups.

Finally, a weakness in some of the normative and clinical studies reviewed above is that insufficient consideration has been given to the role of attention in performance. There is a close relationship between saccades and visual–spatial attention, although the precise nature of the relationship between orienting of visual–spatial attention and programming of saccades is still being debated (e.g., Doré-Mazars, Pouget, & Beauvillain, 2004; Godjin & Theeuwes, 2003; Juan, Shorter-Jacobi, & Schall, 2004). On the one hand, this relationship provides an advantage for researchers interested in attention because eye movements provide an excellent means of investigating attention. On the other hand, it also means that eye movement data cannot be interpreted without taking attentional factors into account. Although this may be true for most cognitive tasks, it is especially true for eye movement tasks. An eye movement to a stimulus implies overt orienting of visual–spatial attention to that location (e.g., Hoffman & Subramaniam, 1995; Peterson, Kramer, & Irwin, 2004; but also see Mokler & Fischer, 1999), and regions involved in the control of eye movements, such as the frontal eye fields, lateral intraparietal area, and superior colliculus, are also involved in covert visual–spatial attention (e.g., Moore & Fallah, 2001; Müller, Philiastides, & Newsome, 2004; Murthy, Thompson, & Schall, 2001; Powell & Goldberg, 2000; Wardak, Ibos, Duhamel, & Olivier, 2006). Indeed, some researchers have questioned the meaningfulness of the distinction between visual–spatial attention and visual–spatial working memory (e.g., Awh & Jonides, 2001; Medendorp, Goltz, & Vilis, 2006). Thus, for instance, it is likely that the improvements seen in prosaccade RTs through at least adolescence are in part related to attentional factors, and not just improvements in general processing speed or oculomotor programming (Hainline, 1988; Ross & Ross, 1983). Similarly, immaturity or impairments on tasks assessing antisaccades, predictive saccades, or memory-guided saccades cannot be interpreted solely in terms of inhibition, anticipation, or working memory, respectively, without considering the role of visual–spatial attention in performance.

### **Limitations of eye tracking as a tool**

To use a tool effectively, it is necessary to be aware of its limitations as well as strengths. One limitation of eye tracking is the extent to which inferences can be drawn from the oculomotor system about other motor systems. The neural bases of eye movements on laboratory tasks are established at a higher level of detail than the neural bases of other kinds of movements (Schall, Hanes, & Taylor, 2000). In addition, oculomotor and skeletomotor processes have been hypothesized to be organized in parallel frontostriatal loops (Alexander, DeLong, & Strick, 1986). Furthermore, certain experimental manipulations (e.g., the

number of sequential movements to be made, the effect of foreperiod length) have similar effects on oculomotor and skeletomotor systems (summarized in Schall et al., 2000), and eye and hand movements share a common reference frame when reaching for a target (Scherberger, Goodale, & Andersen, 2003). Thus, there is the temptation to use eye movements as a simple model of motor control. However, other evidence suggests that the two systems may not have a parallel organization. For example, pointing away and looking away from visual targets recruit overlapping but separable regions and may be accomplished through somewhat different mechanisms (Connolly, Goodale, Goltz, & Munoz, 2005). In addition, phenomena observed in the manual modality are not always replicated in the oculomotor modality. For instance, the effects of stimulus intensity are similar to the effect on manual RTs for externally, but not internally guided saccades (Jaśkowski & Sobieralska, 2004). Similarly, saccades do not show the one target advantage observed in manual movements (Pratt, Shen, & Adam, 2004). Therefore, it is important to be careful about making generalizations across systems.

Second, eye movements in the lab may not necessarily behave like eye movements in the real world. Many saccade tasks require a single saccade to be made to a single stimulus. In a naturalistic context, however, we make sequences of saccades in a more complex environment. The characteristics and neural bases of saccades completed during a sequence (e.g., Caspi, Beutter, & Eckstein, 2004; Shima & Tanji, 1998; Van Loon, Hooge, & Van den Berg, 2002) or in perceptually complex environments (e.g., Deubel & Frank, 1991; Ilg, Jin, Schumann, & Schwarz, 2006; Schiller & Kendall, 2004) differ from those elicited during typical laboratory tasks. The characteristics of monkeys' eye movements during free-viewing visual search also differ from fixations during traditional search tasks, in which practice trials are provided and accurate performance is rewarded (Shen & Paré, 2006). Furthermore, naturalistic contexts tax to a greater extent the decision making processes involved in where and when to look and elicit more proactive and anticipatory eye movements (Land & Furneaux, 1997).

The brain bases of eye movements under typical laboratory conditions were compared to those in a more naturalistic task in a recent study (Ipata, Gee, Goldberg, & Bisley, 2006). Previous studies had yielded conflicting results regarding the role of the lateral intraparietal area in saccade generation. In this study, monkeys were trained to perform a visual search task. Unlike typical laboratory tasks, their eye movements were not restricted, and the monkeys did not receive any rewards or punishments for looking at the distractors. Behavioral results indicated that saccadic RTs were shorter than RTs obtained on tasks on which the rewards were contingent on the monkeys' making certain eye movements, suggesting that the monkeys were being more cautious when a reward depended on the eye movement. In contrast to the findings of other studies, there were very strong relationships between activity in the lateral intraparietal area and the goal and latency of saccades. To explain the discrepancy between their findings and those of previous studies, the researchers suggested that neurons in the lateral intraparietal area represent a salience map that guides attention. This map is normally activated in conjunction with both attention and eye movements. In artificial situations in which monkeys are punished for making an eye movement to an attended location, activity in this area may be tightly related to attention but not to eye movements. In other words, brain-behavior relationships may differ between typical laboratory tasks and more naturalistic settings. More direct comparisons between laboratory and naturalistic contexts, perhaps using head-mounted eye monitors, can shed more

light on the degree to which results obtained in the lab are applicable to the real world.

Third, because even the simplest prosaccades and smooth-pursuit eye movements are mediated by distributed neural networks, eye tracking by itself is of limited use when the researchers' goal is to make inferences about specific brain regions in typically developing children or in psychiatric disorders that do not involve focal lesions. This limitation can be overcome to some extent if researchers have a priori and "risky" (Meehl, 1978) hypotheses that can constrain the interpretation of the findings, and rely on more than one or two global measures of performance. With this approach, it might be possible to conduct studies that lead to accumulating and more detailed knowledge about the development or impairment of specific regions or circuits.

Finally, there are limitations in making inferences about the sources of developmental and clinical differences. Especially when only one or two outcome variables are used to measure performance, it cannot be assumed that the task is being performed in the same way—with the same cognitive and neural substrates—across ages or clinical groups. Many of the tasks used in eye tracking, though usually simpler than more traditional tasks, still involve a multiplicity of cognitive processes. Therefore, it is possible that the lower level of performance observed in children compared to adults is due to one reason at one stage of development and another reason at another stage. For instance, young children may have difficulty performing the antisaccade task due to difficulties in attention, preparatory processes, and inhibition, whereas older children may have difficulties only with the inhibitory component. Nevertheless, if only one outcome measure is used, the data could still look as if there is a gradual improvement in performance, implying gradual maturation of a coherent set of neural substrates underlying a unitary process. Thus, a multimethod approach is necessary to identify sources of failure at different developmental stages. Indeed, in the only developmental study of eye tracking that also used electrophysiological measures, Klein and Feige (2005) demonstrated that 7- to 11-year-olds were recruiting different regions than adults prior to target onset on the antisaccade task, and that the neural substrates shifted with age. Similarly, in clinical populations, it cannot be assumed without evidence that the lower performance evidenced by the clinical groups is attributable to the main construct that is purported to be assessed by the task.

### **Strengths and potentials of eye tracking as a tool**

Despite these limitations, eye tracking measures have much to offer to developmentalists. One important benefit of eye tracking measures is that they open up alternative ways of examining development. They are easily amenable to a process-oriented approach, through examination of the dynamics of saccades, the course of pursuit performance, the shapes of pupillary waveforms, and the evolving pattern of fixations during scene and face perception. By emphasizing the active, goal-directed nature of eye movements, eye tracking studies also highlight the fact that perception involves control over action. By emphasizing the dynamic nature of resource recruitment as a function of task demands, organismic priorities, and motivational factors, pupillary dilation studies highlight the top-down, active, and flexible nature of attentional control (e.g., Meyer & Kieras, 1997), as opposed to a boxology approach in which attention is viewed as more of a static construct (e.g., Baddeley, 1996).

Eye tracking is a non-invasive technique that can be tolerated by young children. In addition, many of the tasks used in eye tracking studies are relatively simple and require no reading skills or complex motor skills. Thus, eye tracking measures provide a powerful means to compare performance across a wide range of ages and clinical groups without these confounding factors.

The fundamental characteristics of basic eye tracking measures (saccades, pursuit, fixations during scene perception, pupillary dilation) are well delineated, there is consensus on the operational definitions of the key measures, and the values of most of these measures can reasonably be expected to fall within a relatively narrow range. For pro- and antisaccade tasks, an attempt has been made to ensure that task parameters and definitions of the dependent variables are identical across laboratories (Fischer et al., 1997a, 1997b). Indeed, to standardize data collection across clinical and research settings for fixation and pro- and antisaccade tasks, Hartnegg and Fischer (2002) have developed a head-mounted eye-tracker that not only records eye movements but also presents stimuli. The data collected on this device can be compared to normative data collected by the authors for ages 7–70 years.

In addition, there is a large body of excellent research on saccades and smooth-pursuit eye movements in adults and non-human primates. This research has detailed which kinds of factors affect which aspects of eye movements and how they are related to specific cognitive and motor processes. This work provides a strong foundation for further research. So far, however, it has been vastly under-utilized in studies with children and adolescents. Much more work can be done examining age-related and clinical differences in the effects of contextual and temporal factors on eye movements as well processes such as visual-spatial attention and its guidance by bottom-up and top-down factors (e.g., Gagnon, O'Driscoll, Petrides, & Pike, 2002; Iba & Sawaguchi, 2003; Shen & Paré, 2006; Thompson, Bichot, & Sato, 2005), and motor control (e.g., Gold & Shadlen, 2000; Sinha, Brown, & Carpenter, 2006; Vaziri, Diedrichsen, & Shadmehr, 2006). There is also, of course, a very large literature on saccade tasks assessing inhibition and working memory.

The neural bases of different types of eye movements have been examined in many studies with human adults and non-human primates, but only four studies so far have used electrophysiological (Klein & Feige, 2005) or brain imaging methods (Dalton et al., 2005; Luna et al., 2001; Scherf et al., 2006) to examine the maturation or integrity of the neural bases of eye movements in children and adolescents. Thus, the potentials of eye tracking measures to probe the maturation and integrity of the oculomotor circuitry in these populations have barely been tapped. In studies with specific a priori hypotheses and careful attention to contextual and temporal factors, eye tracking can be very useful in investigating this circuitry. In addition, despite the vast amount of research using single-cell recording and lesion methods to probe neural substrates of eye movements in non-human primates, very little research has been conducted on young animals. Extensions of primate research on saccades to developing animals can be invaluable in addressing questions of interest to developmental psychologists.

There is a smaller but equally interesting body of research in human adults and non-human primates on eye movements during face and scene perception. The advantage of these measures is that they can be used flexibly to address a variety of questions regarding scene perception (e.g., Henderson, 2003), attention (Bekkering & Neggens, 2002; Houtkamp & Roelfsema, 2005; McCarley, Wang, Kramer, Irwin, & Peterson, 2003), emergent literacy and language (e.g., Knoeflerle & Crocker, 2006), short- and long-term memory

(e.g., Chua, Boland, & Nisbett, 2005), eyewitness testimony, observational learning, skill learning (e.g., Land & McLeod, 2000; Lee & Anderson, 2001; Underwood, Chapman, Brocklehurst, Underwood, & Crundall, 2003), the development of expertise (Reingold, Charness, Pomplun, & Stampe, 2001), and the relations between perception and action. Eye movements can be especially useful for addressing questions about implicit cognition (e.g., Dragoi & Sur, 2006; Tseng & Li, 2004) and problem solving (e.g., Grant & Spivey, 2003; Hodgson, Tiesman, Owen, & Kennard, 2002).

Pupillary dilation provides a direct psychophysiological measure of resource recruitment and effort that can be time-locked to stimuli and responses in an exquisitely sensitive manner. Thus, in research on normative development, pupillary dilation can play a key role in providing an empirical foundation for resource theories of development (e.g., Case, 1991; Pascual-Leone, 2000; Swanson, 1999).

For the most part, the tasks in the studies reviewed above have been taken from the cognitive science or neuroscience literatures and used to investigate cognitive processes in children and adolescents. However, eye tracking is a tool that can also be used effectively in the service of elucidating emotional processes and social information processing. The studies on eye movements to faces and social stimuli in typically developing and autistic samples are excellent examples of this approach. One could imagine many more studies examining eye movements during face and scene perception that could yield valuable information regarding socioemotional development and the influence of different factors on how typical and atypical populations of children process social information.

There is a growing number of eye movement studies in adults in cognition, emotion, and motivation (e.g., Platt, 2002; Polli et al., 2005; Roesch & Olson, 2005), including the effects of rewards on saccades and the neural bases of these effects (e.g., Campos, Breznen, Bernheim, & Andersen, 2005; Hikosaka, Nakamura, & Nakahara, 2006; Hodgson, Golding, Molyva, Rosenthal, & Kennard, 2000). In a recent study extending this line of research to adolescents, investigators showed that monetary rewards and punishments had greater effects on antisaccade parameters in adolescents than in adults (Jazbec et al., 2006). Pupillary dilation measures also provide an excellent means of testing the effects of rewards and difficulty on effort (Steinhauer & Hakerem, 1992), and examining processing of emotionally laden stimuli (Bitsios, Szabadi, & Bradshaw, 2004; Partala & Surakka, 2003).

Finally, eye tracking measures can be useful in different kinds of translational research with typical and atypical populations. As reviewed above, for instance, eye tracking measures have been used to examine the effects of medications in ADHD. They can also be used to examine the effects of cognitive and psychosocial interventions in educational and clinical settings. For instance, eye movements were used to examine the effects of monetary rewards and punishments on antisaccades in adolescents with depression or anxiety (Jazbec et al., 2005), and how adults with spider phobia process pictures that include spiders (Rinck & Becker, 2006). Pupillary dilations were used to demonstrate that depression in adults is associated with ruminatory tendencies for negative or personally relevant stimuli (Siegle et al., 2003a). This research can easily be extended to examine the effects of interventions. In a study on dyslexia, where abnormalities in eye movements themselves are believed to contribute to reading difficulties, children with dyslexia were trained to exert greater control over internally guided eye movements (Fischer & Hartnegg, 2000). Finally, advances in technology have made it feasible to use head-mounted cameras to



determine where participants are fixating in a scene. It is especially easy to imagine studies where this technology could be used to translate research from the lab to the real world (e.g., Hayhoe & Ballard, 2005).

In conclusion, studies of eye tracking in normative and atypical populations of children and adolescents have already yielded reliable and intriguing findings regarding a wide variety of cognitive processes and their neural substrates. Eye tracking offers researchers many more possibilities for studying cognitive and socio-emotional development.

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