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A review on eye movement studies in childhood and adolescent psychiatry

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ABSTRACT

The neural substrates of eye movement measures are largely known. Therefore, measurement of eye movements in psychiatric disorders may provide insight into the underlying neuropathology of these disorders. Visually guided saccades, antisaccades, memory guided saccades, and smooth pursuit eye movements will be reviewed in various childhood psychiatric disorders. The four aims of this review are (1) to give a thorough overview of eye movement studies in a wide array of psychiatric disorders occurring during childhood and adolescence (attention-deficit/hyperactivity disorder, oppositional deviant disorder and conduct disorder, autism spectrum disorders, reading disorder, childhood-onset schizophrenia, Tourette's syndrome, obsessive compulsive disorder, and anxiety and depression), (2) to discuss the specificity and overlap of eye movement findings across disorders and paradigms, (3) to discuss the developmental aspects of eye movement abnormalities in childhood and adolescence psychiatric disorders, and (4) to present suggestions for future research. In order to make this review of interest to a broad audience, attention will be given to the clinical manifestation of the disorders and the theoretical background of the eye movement paradigms.

1. Aims and structure of this review

1.1. Aims

Eye movements are perhaps the most thoroughly studied domain in the field of cognitive neuroscience. Numerous ingenious paradigms have been developed to unravel their underlying neurological and cognitive substrates. Current knowledge of these processes has been obtained by measuring eye movements in monkeys and healthy adults. These studies have provided detailed knowledge of the basic characteristics of eye movements. This, in turn, has prompted researchers to assess eye movements in various psychiatric disorders and has facilitated the understanding of the complex underlying neuropathophysiology of these disorders. Most of these studies have focused on eye movements in adult patients. These findings may not necessarily be extrapolated to child and adolescent patients, since the clinical manifestation and aetiology of psychiatric disorders may differ between childhood-onset and adult-onset (Carlson, Bromet, & Sievers, 2000; Jaffee et al., 2002; Matsumoto et al., 2001). Moreover, eye movements themselves may differ substantially between children and adults (Karatekin, 2007; Mezzalira, Coelho Neves, Queiroz Maudonnet, o Bilécki, & Gobbi de Ávila, 2005). It seems important, therefore, to review separately eye movement studies in childhood and adolescent psychiatric disorders to determine which eye movement dysfunctions are present in these patients.

Several reviews are available on eye movement studies in adult patients (often focused on a particular disorder or eye movement paradigm; Hutton & Ettinger, 2006; Lee & Williams, 2000; Reuter & Kathmann, 2004; Trillenberg, Lencer, & Heide, 2004), whereas only two reviews are available on child and adolescent patients (Karatekin, 2007; Sweeney, Takarae, Macmillan, Luna, & Minshew, 2004). Both reviews were instructive in reviewing the literature on attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorders (ASD) American Psychiatric Association [APA] (1994), but eye movement literature on Tourette's syndrome (TS) and childhood-onset schizophrenia was reviewed in only one of these papers, and neither review focused on childhood-onset obsessive compulsive disorder (OCD), reading disorder (RD), anxiety and depression, or oppositional deviant disorder (ODD) and conduct disorder (CD). Therefore, the main aims of the current review are (1) to give a thorough overview of eye movement studies in a wide array of psychiatric disorders occurring during childhood and adolescence, (2) to discuss the specificity and overlap of eye movement findings across disorders, (3) to discuss the developmental aspects of eye movement abnormalities in childhood and adolescence psychiatric disorders, and (4) to present suggestions for future research.

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1.2. Structure

Eye movements in psychiatric disorders are of interest to various professions: cognitive neuroscientists, psychologists, and psychiatrists. In order to perform this review and make it of interest to a broad audience, we will briefly discuss both the background of eye movement paradigms as well as the clinical manifestation of each disorder. For a more thorough discussion of eye movement paradigms, readers are referred to Barnes, this issue (for SPEM) and Hutton, this issue (for saccades). Furthermore, we will try to summarize the most important neurological and cognitive deficits associated with each disorder, since these findings have often guided eye movement studies to focus on a particular topic/direction. This review will focus on antisaccades (AS; see glossary), visually guided saccades (VGS; see glossary), memory guided saccades (MGS; see glossary), and smooth pursuit eye movements (SPEM; see glossary), in order to increase comparability with other articles appearing in this issue. Therefore, eye movement studies on visual search (Theeuwes, Kramer, Hahn, & Irwin, 1998; Williams, Reingold, Moscovitch, & Behrmann, 1997), picture scanning (Peters, Iyer, Itti, & Koch, 2005; Torralba, Oliva, Castelhano, & Henderson, 2006), and eye movements during reading (Laubrock, Kliegl, & Engbert, 2006; Rayner, 1998), are not reviewed here. Only those studies are reviewed here that have included children and adolescents (participants of 18 years and younger). In some studies, the age range included both adolescents and adults. Those studies were only included, if the medium age of the participants was 18 years or younger. With regard to adult psychiatric populations, saccadic abnormalities are reviewed by Gooding and Basso in this special issue, and SPEM distortions by O'Driscoll.

2. Utility of eye movement recordings in psychiatric disorders

As will become evident in this review, there is a growing body of the literature investigating eye movements in children with psychiatric disorders. Recording eye movement paradigms provide various benefits over standard procedures. First, relative to reaction time data, eye movements provide a much richer data set which allows better understanding of the underlying neurophysiological mechanisms. Eye movements, while performing a task, not only provide information regarding the process measured by the task, but also regarding the metrics and dynamics of oculomotor control, such as velocity, duration and trajectories of saccades (see, e.g., Van der Stigchel, Meeter, & Theeuwes, 2006; Smyrnis, this issue).

Second, eye movement tasks are designed to have simple visuomotor baseline (or control) conditions, e.g., prosaccades (see glossary) for antisaccades. This is important from an a priori experimental point of view and has an important (empirical) advantage over non-eye movement tasks in that certain patient populations (e.g., schizophrenia) have robust slowing of basic, reflexive manual reaction times but no (or inconsistent) deficits in reflexive saccadic reaction times.

Third, because the execution of an eye movement is the result of a complex interaction between various cognitive processes, oculomotor behavior tells us something about these processes and deficits in these processes. Making an eye movement is an important way of exploring our environment. Therefore, eye movements give some indications on how children experience their daily environment and can increase our knowledge about the various complex behavioral processes underlying psychiatric disorders in children.

Fourth, recent technological advances have made it increasingly easier to record eye movements with a high spatial and temporal resolution. Non-invasive techniques enable eye movement systems to be relatively easily accessible while the precision of measurement of modern eye trackers is high. In line with this, remote eye tracking systems are also becoming more accurate. These systems have the advantage that the participant does not have to carry a head-mounted system, which can be heavy for children, especially if they are also restricted in their head movements.

Fifth, as will be revealed in the next section, eye movement tasks are generally very simple and not difficult to perform by children with psychiatric disabilities. The paradigms discussed in the current review require no advanced cognitive skills such as language, reading, or complex motor responding. This makes the paradigms relatively straightforward and easy to explain to participants even at an early age.

Box 1. Utility of eye movement recordings in childhood and adolescence psychiatric disorders

Benefits of eye movement paradigms over non-eye movement paradigms:

1. Relative to reaction time data, eye movements provide a much richer data set which allows better understanding of the underlying neurophysiological mechanisms.
2. Eye movement tasks are designed to have simple visuomotor baseline (or control) conditions, e.g., prosaccades for antisaccades, which is important from an a priori experimental point of view and has an important (empirical) advantage over non-eye movement tasks in that certain patient populations (e.g., schizophrenia) have robust slowing of basic, reflexive manual reaction times but no (or inconsistent) deficits in reflexive saccadic reaction times.
3. Eye movements give some indications on how participants experience their daily environment and can increase our knowledge about the various complex behavioral processes underlying psychiatric disorders.
4. Recent technological advances have made it increasingly easier to record eye movements. Techniques are becoming increasingly accurate and non-invasive, making them increasingly suitable for recording of eye movements in children.
5. Eye movement tasks are generally very simple and not difficult to perform by children (with psychiatric disabilities) and often require no advanced cognitive skills such as language, reading, or complex motor responding.

Conditions under which eye movement paradigms would further benefit over non-eye movement paradigms:

1. The underlying brain mechanisms of eye movement paradigms are well documented in adults. However, much less is known about the underlying brain mechanisms in children and adolescents; increasing knowledge in this area would allow for affected oculomotor behavior to more reliably pinpoint to specific neurological problems in children and adolescents compared to non-eye movement paradigms.
2. The normal developmental trajectories in oculomotor behavior have largely been studied using ad hoc samples and only few studies have modeled the developmental trajectories appropriately. Well-designed longitudinal studies would allow for a better understanding of the developmental trajectories of eye movements. This knowledge would allow for a more reliable investigation of continuation and discontinuation in neurobiological parameters of psychiatric disorders, which hypothetically may prove to be more stable traits than non-eye movement parameters.

There are two conditions under which eye movement paradigms would further benefit over non-eye movement paradigms. First, the underlying brain mechanisms of eye movement paradigms are well documented in adults (Hall & Moschovakis, 2003; Leigh & Kennard, 2004; Munoz, 2002; Schall, 1991). However, much less is known about the underlying brain mechanisms in children and adolescents. Increasing knowledge in this area would allow for affected oculomotor behavior to more reliably pinpoint to specific neurological problems in children and adolescents compared to non-eye movement paradigms and may even be used as a diagnostic tool.

Second, the normal developmental trajectories in oculomotor behavior have largely been studied using ad hoc samples and only few studies have modeled the developmental trajectories appropriately. Well-designed longitudinal studies would allow for a better understanding of the developmental trajectories of eye movements. This knowledge would allow for a more reliable investigation of continuation and discontinuation in neurobiological parameters of psychiatric disorders, which hypothetically may prove to be more stable traits than non-eye movement parameters.

3. Eye movement paradigms: Measurement and proposed neurophysiological underpinnings

Various paradigms have been used to investigate eye movements in children with psychiatric disorders. As mentioned earlier, the present review discusses four types of saccadic paradigms, namely, AS, VGS, MGS, and SPEM. These paradigms are designed to assess basic oculomotor behavior or to measure executive functions, such as inhibition (see glossary) and working memory. For a detailed review on the neurophysiology and neuroanatomy of these paradigms, please see the papers in this special issue (SPEM, Ilg & Thier; Lencer & Trillenberger; Sharpe; Saccades, Johnston & Everling; McDowell and colleagues; Mueri & Nyffeler).

3.1. Antisaccades (AS)

In AS a visual onset is presented and observers are required to make an eye movement away from the onset location to its mirror position (Everling & Fischer, 1998; Hallet, 1978; Munoz & Everling, 2004). AS have longer latencies than saccades towards the onset and observers frequently make erroneous saccades towards the onset location. Successful performance on AS requires two processes: the top-down inhibition of a reflexive saccade (see glossary) to the onset location, and the execution of a voluntary eye movement to the mirror location of the onset. Typical measures in AS are the number of directional errors (reflecting inhibitory problems: a failure to suppress an inappropriate response due to problems with top-down regulation) and saccade latency.

Neuropsychological research has revealed an important role for frontal areas in this task. For instance, imaging studies have identified various frontal areas that are more active during AS than during VGS, like the frontal eye fields (see glossary), dorsolateral prefrontal cortex (DLPFC; see glossary), and the supplementary eye fields (Everling & Munoz, 2000; Funahashi, Chafee, & Goldman-Rakic, 1993). Directional errors are, therefore, generally linked to frontal dysfunctions.

3.2. Visually guided saccades (VGS)

In VGS, participants are instructed to make an eye movement to a visual stimulus presented in the periphery. This task is generally included to investigate the basic dynamics of an eye movement and is in most cases included as a baseline condition in AS or MGS. Typical measures are saccade latency, variability in latency, amplitude, under-versus overshoot, peak velocity, and duration.

Deviations in these measures of saccade metrics can pinpoint to deficits in the neurological substrate of eye movements, such as the cerebellum (see glossary) or the frontal eye fields (Leigh & Zee, 1999). Saccade latency is, for instance, regarded as a measure of the speed of visual processing, whereas variability in latency indexes the regulating processes of saccade initiation.

3.3. Memory guided saccades (MGS)

In MGS, participants are instructed to look at a central fixation (see glossary) point. During this fixation, a target appears at a location in the peripheral visual field. The participant is not allowed to make a saccade towards the target but has to remember the location of the target. When the fixation point disappears, the participant is required to make a saccade towards the memorized location. The time between the disappearance of the target and the moment the participant is allowed to make a saccade (delay period) can be varied in order to vary the visuo-spatial working memory (see glossary) load. Typical measures in MGS are accuracy (reflecting how well the participant has remembered the location of the target), the number of anticipatory errors (reflecting inhibitory problems), and saccade latency.

The neurological substrates underlying performance on the memory guided saccade task are well established: in addition to the brain areas active in basic oculomotor control, memory guided saccades activate the DLPFC, anterior cingulate (see glossary), and supplementary eye field (see glossary; Ettinger et al., 2005; Sawaguchi & Iba, 2001; Sweeney et al., 1996). Based on these findings, deficits in the MGS are related to frontal functioning.

3.4. Smooth pursuit eye movements (SPEM)

SPEM involve the smooth oculomotor tracking of a small object in motion with a constant (slow) speed. In order to execute correctly the task, the velocity of this non-ballistic movement has to be continuously adapted to the velocity of the object. The most widely measured variable in SPEM is gain (see glossary), calculated as the ratio of eye velocity to target velocity, which can indicate difficulties in matching gaze to target velocity. Other measures exist, such as root mean square error (the difference between target and gaze position) and the number of compensatory and intrusive saccades. If the eyes do not reproduce the target motion, the saccadic system has to compensate for this error which can be indexed by the number of saccades necessary to catch-up with the moving object.

SPEM have a strong emphasis on basic oculomotor control and less on cognitive processes. The neurophysiological correlates of the pursuit system overlap with that of saccadic movements and both systems work in an integrative way (Fukushima, 2003). If deficits in SPEM are observed, it is difficult to pinpoint specific brain areas that are causing the effect, because a widespread network is responsible for correct SPEM performance. Therefore, problems in SPEM are generally not linked to specific neurological and/or cognitive deficits but to fundamental deficiencies in the functioning of the pursuit system.

4. Overview of eye movement studies in childhood and adolescent psychiatric disorders

A summary of all the reviewed studies concerning patient characteristics (diagnosis, age, and gender), eye movement paradigm, analyzed dependent measures, and results is provided in Table 1. All clinical descriptions are according to APA (1994), unless specified otherwise.

4.1. Attention-deficit/hyperactivity disorder (ADHD)

4.1.1. Clinical manifestation of ADHD

ADHD is one of the most common psychiatric conditions of childhood, characterized by symptoms of inattention, hyperactivity, and impulsivity (APA, 1994). Inattention may manifest itself as failing to give close attention to details, making careless mistakes in (school)work, difficulty persisting in tasks that require sustained attention, and not listening to what is being said. Hyperactivity is often characterized by fidgetiness, unableness to remain seated when expected to do so, excessive talking, and appearing as 'driven by a motor'. Impulsivity is often expressed as lack of patience, difficulty awaiting turn, and frequently interrupting others (APA, 1994). Most individuals have symptoms in all three domains (combined type), although a predominantly inattentive and a predominantly hyperactive-impulsive subtypes also exist. In most cases, the severity of the symptoms become less conspicuous with age, especially signs of excessive gross motor activity (APA, 1994), although the core symptoms of ADHD are believed to persist into adulthood for some of the patients (Tannock, 1998). ADHD is estimated to affect around 5% of children with an overrepresentation of boys by approximately 5:1–9:1 (APA, 1994). Genetic factors strongly contribute to the disorder, but adverse environmental factors have also been identified. The disorder is frequently accompanied by developmental problems, including dyslexia, specific and general learning difficulties and causes significant impairments in social relationships and school performance. Many patients also suffer from comorbid conditions such as antisocial, mood, anxiety, and substance use disorders (Biederman and Faraone, 2005).

4.1.2. Neurological and cognitive problems in ADHD

Because problems with impulsivity and inattention suggest deficits in the voluntary control of behavior, the deficits observed in ADHD have generally been related to the neurological dysfunctions in the fronto-striatal circuitry (Castellanos, 2001; Mattes, 1980; Tannock, 1998). Indeed, anatomical neuroimaging studies have revealed altered architecture of this circuitry (Castellanos et al., 1996; Yeo et al., 2003) and reduced activity in frontal and cingulate regions in subjects with ADHD compared to controls (Aman & Carmichael, 1997; Rubia et al., 1999; Zametkin et al., 1990). Moreover, the dopaminergic system plays an important role in the pathology of ADHD (Levy, 1991; Levy & Swanson, 2001) which may also account for the problems in executive functioning. With respect to the cognitive dysfunctions, children with ADHD are known to have problems in a variety of tasks related to executive control. Overall, children with ADHD perform worse than controls on inhibition tasks (Hervey, Epstein, & Curry, 2004; Lijffijt, Kenemans, Verbaten, & Van Engeland, 2005; Nigg, 1999; Oosterlaan, Logan, & Sergeant, 1998), working memory tasks (Karatekin & Asarnow, 1998b; Kempton et al., 1999; Martinussen, Hayden, Hogg-Johnson, & Tannock, 2005; Oie, Sunde, & Rund, 1999) and show an overall lower speed of responding (Hooks, Milich, & Lorch, 1994; Karatekin & Asarnow, 1998a; Mason, Humphreys, & Kent, 2003; Schachar, Tannock, Marriot, & Logan, 1995; Van der Meere & Sergeant, 1988; Van der Stigchel et al., 2007). Current cognitive theories of ADHD have focused on response inhibition as forming the primary deficit, although these models differ in the formulation of the fundamental impairment (for an overview see Tannock, 1998). Importantly, one of the most robust findings in the ADHD literature is an increased variability of responding in ADHD patients compared to controls (Kuntsi, McLoughin, and Asherson, 2006; Leth-Steensen, Elbaz, & Douglas, 2000), which may be related to a defective effort control mechanism (Leth-Steensen et al., 2000).

4.1.3. Eye movement studies in ADHD

The hypothesis that children with ADHD have problems with response inhibition is partly revealed through research using AS, MGS, and intrusive saccades. These measures have in common that inappropriate/irrelevant motor responses need to be inhibited and are therefore suitable tests to investigate whether motor inhibition is indeed affected in ADHD.

4.1.4. Antisaccades

The results on antisaccade performance in ADHD are somewhat inconsistent, although the studies that found an elevated number of antisaccade errors are in the majority. In seven of 10 studies, more directional errors on antisaccade tasks were found in children with ADHD (Habeych, Folan, Luna, & Tarter, 2006; Karatekin, 2006; Klein, Raschke, & Brandenbusch, 2003; Mostofsky, Lasker, Cutting, Denckla, & Zee, 2001a; Mostofsky, Lasker, Singer, Denckla, & Zee, 2001b; Munoz, Armstrong, Hampton, & Moore, 2003; O'Driscoll et al., 2005). No effects on directional errors were observed in the other three studies (Aman, Roberts, & Pennington, 1998; Hanisch, Radach, Holtkamp, Herpertz-Dahlmann, & Konrad, 2006; Rothlind, Posner, & Schaughency, 1991). However, the lack of effect on antisaccade errors observed in these three studies could be explained in terms of a possible lack of statistical power and task differences. For instance, one study reported a statistical trend of $p = 0.16$ towards more directional errors (Hanisch et al., 2006). In the antisaccade task conducted by Rothlind and colleagues (1991), each participant performed only 10 trials in each condition, possibly causing low statistical power. In the third study that did not report an elevated number of antisaccade errors, a different version of the antisaccade task was used in which an additional visual event occurred on the antisaccade location and a detection task was associated with the presentation of the target (Aman et al., 1998). This could account for the lack of effect, although it must be noted that patients still committed 10% more direction errors than controls in that study. It thus appears that the majority of studies reporting on antisaccade performance in children with ADHD find an elevated number of directional errors, indicating that children with ADHD are less able than controls to suppress inappropriate responses.

There are a number of variables that influence antisaccade performance in ADHD, namely developmental effects, subtype of ADHD, cognitive manipulations, and medication. Children with ADHD did not show the normal age-related decrease in latency for correct antisaccade responses (Klein et al., 2003), suggesting an abnormal pattern of development of antisaccade performance in affected children. Another factor that seems to play a role is the specific subtype of ADHD. Recently, ADHD-combined subtype was compared with ADHD-inattentive subtype on antisaccade performance (O'Driscoll et al., 2005). Results showed that participants with ADHD-combined subtype were significantly more impaired than participants with ADHD-inattentive subtype, who did not show any impairments, indicating that the deficits in inhibiting eye movements might be mediated by brain structures implicated specifically in the hyperactive/impulsive symptoms of ADHD (O'Driscoll et al., 2005). Karatekin (2006) showed that reducing working memory demands or training of working memory improved the accuracy of adolescents with ADHD up to the level of controls on their first administration of the antisaccade task, which shows that cognitive manipulations can help to overcome some of the inhibitory deficits in children with ADHD. Further, it has been investigated whether performance on the antisaccade task improves with administration of the stimulant methylphenidate. One of the possible effects of methylphenidate is an increase in inhibitory control through its effect on fronto-subcortical pathways (Faraone & Biederman, 1998). A repeated measures experiment was conducted by Klein and colleagues (Klein, Fischer,

Table 1
Overview of eye movement studies in childhood and adolescence psychiatric disorders

Authors	*N*	Gender	Age (years)	Eye movement tasks	Dependent variables	Results
Attention-deficit/hyperactivity disorder (ADHD)						
Aman et al. (1998)	22 ADHD	22 m/0f	10–14	AS	*N* Errors	ns
	22 NC	22 m/0f	10–14			
Castellanos et al. (2000)	32 ADHD	0 m/32 f	6–13	SPEM	Gain, root mean square error, *N* saccades, catch-up saccades and anticipatory saccades	ns
	20 NC	0 m/20 f	6–13	MGS (delay 1.2 s)	*N* Anticipatory saccades, latency, accuracy	*N* Anticipatory saccades: ADHD > NC ($p < .001$). Other DVs ns
Habeych et al. (2006)	13 High risk for alcohol use disorder + ADHD	Unknown	10–12	AS	*N* Errors, latency, peak velocity, gain	*N* Errors: ADHD > NC ($p = .02$). Other DVs ns
Hanisch et al. (2006)	22 ADHD	15 m/7 f	10–14	VGS	Latency	ns
	22 NC	14 m/8 f	10–14	AS	*N* Errors	ns
Jacobsen et al. (1996)	18 ADHD	17 m/1 f	9–15	SPEM	Gain, root mean square error,% time tracking	Root mean square error: ADHD > NC ($p < .001$).
	22 NC	16 m/6 f	9–18		*N* catch-up, anticipatory and back-up saccades, *N* square wave jerks, amplitude catch-up, anticipatory and back-up saccades	Other DVs ns
Karatekin and Asarnow (1998a)	28 ADHD	19 m/9f	*M* = 13.9	VGS	Latency	ns
	38 NC	18 m/20 f	*M* = 14.1			
Karatekin (2006)	10 ADHD	8 m/2 f	12.4–18.5	VGS	Latency	ns
	15 NC	9 m/6 f	11.5–19.0	AS	*N* Errors, latency	*N* Errors: ADHD > NC ($p < .001$). Latency: ADHD > NC ($p < .001$)
Klein et al. (2002)	27 ADHD	27 m/0f	10–15	VGS	Latency	ADHD unmedicated > ADHD medicated
				AS	*N* Errors, latency	*N* Error: ADHD unmedicated > ADHD medicated Latency: ADHD unmedicated > ADHD medicated
Klein et al. (2003)	46 ADHD	41 m/5 f	7–15	VGS	Latency	ADHD > NC ($p < .05$).
	46 NC	38 m/8 f	7–15	AS	*N* Errors, latency	*N* errors: ADHD > NC ($p < .0001$). Latency: ADHD > NC ($p < .05$)
Mostofsky et al. (2001a)	19 ADHD	11 m/8 f	7.1–16.1	VGS	Latency, coefficient of variation	Latency: ns Coefficient of variation: ADHD > NC ($p < .05$)
	25 NC	13 m/12 f	7.2–17.9			
				AS	*N* errors	ADHD > NC ($p < .001$)
				MGS (delay 4.5–5 s)	*N* Anticipatory saccades, latency, accuracy Coefficient of variation	*N* Anticipatory saccades: ADHD > NC ($p < .01$) Latency: ADHD > NC ($p < .01$). Other DVs ns
Mostofsky et al. (2001b)	14 ADHD + TS	14 m/0 f	7.8–14.3	VGS	Latency, coefficient of variation	Latency: ADHD + TS > NC ($p < .01$). Coefficient of variation: ADHD + TS > NC ($p < .001$)
	10 NC	10 m/0 f	8.1–12.6	AS	*N* Errors	*N* Errors: ADHD + TS > TS ($p = .07$)
				MGS (delay 4.5–5 s)	*N* Anticipatory saccades, accuracy	*N* anticipatory saccades: ADHD + TS > TS ($p < .05$). Other DVs ns
Munoz et al. (2003)	76 ADHD	61 m/15 f	6–16	VGS	Latency, coefficient of variation, amplitude, duration, peak velocity, gap effect	Latency: ADHD > NC ($p < .05$). Coefficient of variation: ADHD > NC ($p < .001$). Duration: ADHD > NC ($p < .001$). Peak velocity: ADHD < NC ($p < .05$). Gap effect: ns
	75 NC	40 m/35 f	6–16	AS	Latency, coefficient of variation, *N* errors	Latency: ADHD > NC ($p < .01$). Coefficient of variation: ADHD > NC ($p < .001$). *N* errors: ADHD > NC ($p < .001$)
O'Driscoll et al. 2005	10 ADHD-C	10 m/0 f	11–14	VGS	Latency, amplitude, peak velocity	ns
	12 ADHD	12 m/0 f	11–14	AS	*N* errors, latency	*N* errors: ADHD–C > NC (p < .05). Latency: ns
	10 NC	10 m/0 f	11–14			

(*continued on next page*)

Table 1 (*continued*)

Authors	*N*	Gender	Age (years)	Eye movement tasks	Dependent variables	Results
Rommelse et al. (2008)	14 ADHD 15 NC	14 m/0 f 15 m/0 f	7–14 7–14	MGS (delay 3 and 7 s)	Accuracy, latency, *N* anticipatory saccades, *N* intrusive saccades, peak velocity, under/overshoot, duration, coefficient of variation	Accuracy: ADHD < NC ($p = .06$). *N* anticipatory saccades: ADHD > NC ($p < .05$). *N* intrusive saccades: ADHD > NC ($p < .05$). Overshoot: ADHD > NC ($p < .001$). Duration: ADHD > NC $p < .05$). Coefficient of variation: ADHD > NC ($p < .01$). Other DVs ns
Ross et al. (1994)	13 ADHD 10 NC	13 m/0 f 5 m/5 f	9–12 9–12	MGS (delay 0.8 s)	*N* Anticipatory saccades, latency, accuracy	*N* Anticipatory saccades: ADHD > NC ($p = .04$) Other DVs ns
Rothlind et al. 1991	20 ADHD 21 NC	20 m/0 f 21 m/0 f	6.9–13.9 6.8–14.4	AS	*N* Anticipatory saccades, latency	ns
Autism spectrum disorders (ASD)						
Goldberg et al. (2002)	11 Autism	8 m/3 f	12–18	VGS (overlap and gap)	Latency, gap effect	Latency: autism > NC ($p < .05$). Gap effect: ns
	11 NC	8 m/3 f	12–18	AS	*N* Errors, latency, velocity, accuracy	*N* Errors: autism > NC ($p < .001$). Other DVs ns
				MGS (1.5–3.0 s)	*N* Anticipatory saccades, latency, velocity, accuracy	*N* Anticipatory saccades: autism > NC ($p < .01$) Latency: autism > NC ($p < .01$). Other DVs ns
Kemner et al. (2004)	16 PDD	Unknown	*M* = 10.9	SPEM	Position gain, *N* saccade intrusions	ns
	18 NC	Unknown	*M* = 10.0			
Landry and Bryson (2004)	15 Autism	Unknown	3.8–7.6	VGS (disengage and shift)	Latency	Latency on shift: PDD > NC ($p < .01$)
	13 NC	Unknown	2.1–6.2			Latency on disengage: ns
Luna et al. (2007)	61 Autism	Unknown	8–33	VGS	Latency, accuracy, duration, peak velocity	Accuracy: autism < NC ($p < .05$). Other DVs ns
	61 NC	Unknown	8–33	AS	*N* Errors, latency	*N* Errors: autism > NC ($p < .05$). Latency: autism < NC ($p < .05$)
				MGS (delays 1.0–8.0 s)	Accuracy, latency	Accuracy: autism < NC ($p < .001$). Latency: ns
Rosenhall et al. (1988)	11 Autism 26 NC	6 m/5 f Unknown	9–16 7–13	VGS SPEM	Latency	ns Autism: unable to finish experiment
Scharre and Creedon (1992)	34 Autism	32 m/2 f	2–11	SPEM		Autism: 85% unable to finish experiment
Van der Geest et al. (2001)	16 Autism	16 m/0f	*M* = 10.9	VGS (overlap and gap)	Accuracy, latency, gap effect	Gap effect: autism < NC ($p < .05$). Other DVs ns
	15 NC	15 m/0f	*M* = 10.3			
Reading disorder (RD)						
Adler-Grinberg and Stark (1978)	25 RD	20 m/5 f	8.0–12.3	VGS	Latency, peak velocity, amplitude	ns
	19 NC	15 m/4 f	7.1–12.3	SPEM	Smoothness, accuracy, maximum velocity	RD poorer SPEM (more saccades) than NC
Bednarek et al. (2006)	16 RD	9 m/10 f	9.2–10.5	VGS with and without cues	Latency	RD < NC ($p = .02$) without cue. No group differences with cue
	25 NC	13 m/12 f	9.2–10.5			
Biscaldi et al. (1994)	12 RD	Unknown	9–11	VGS standard (overlap and gap) and sequential (overlap, gap, and simultaneous)	Fixation quality, velocity, position, corrective time, *N* express, fast and slow regular saccades	Standard: RD poorer fixation quality, difficulty hitting the target at once, smaller primary sac-cades, shorter RT to the left than NC. Sequential: RD made fewer and larger saccades and had shorter fixations than NC
	12 NC	Unknown	9–11			
Biscaldi et al. (2000)	506 RD 114 NC	Unknown 62 m/52 f	7–17 7–17	VGS AS	Latency *N* (corrected) errors, *N* misses, latency	RD > NC ($p = .01$) *N* errors: RD > NC ($p < .001$). *N* corrections: NC > RD ($p < .001$). *N* misses: RD > NC ($p < .001$). Latency: ns
Biscaldi et al. (1998)	93 RD 92 NC	76 m/17 f 51 m/41 f	8–25 8–25	VGS (single and sequential target)	Single: (SD) latency, *N* express, fast, slow, (too) late, anticipatory saccades, *N* corrective saccades in the same and opposite direction. sequential: *N* target saccades, *N* and amplitude regression saccades	SD latency, *N* fast, too late anticipatory saccades: RD > NC ($p < .05$–$< .01$). *N* slow saccades: RD < NC ($p < .05$). Other DVs ns

Table 1 (*continued*)

Authors	*N*	Gender	Age (years)	Eye movement tasks	Dependent variables	Results
Black et al. (1984a)	28 RD	27 m/1 f	7.8–16.9	VGS	Latency, accuracy, velocity, acceleration, Δ latency abduct and adduct movements	ns
	31 NC	Unknown	6.0–12.2			
Black et al. (1984b)	26 RD	22 m/4 f	8–13	SPEM	*N* (big) saccades, angle covered by saccades *M* angle per saccade, peak velocity	*N* big saccades: RD > NC (p = .02). Angle covered by saccades and M angle: RD > NC (p = .05). Peak velocity ns
	34 NC	19 m/15 f	8–13			
Black et al. (1984c)	35 RD	30 m/5 f	8–13	VGS	*N* (big) saccades, progressions, and regressions by saccades, *M* angle per saccade, peak velocity	ns
	35 NC	20 m/15 f	8–13			
Bogacz et al. (1974)	40 RD	Unknown	6–23	VGS	Step-like intrusions	Normal VGS in RD.
	24 NC	Unknown	7–15	SPEM	Uni- and bi-directional saccades, saccadic jerks	More abnormal saccades in RD than NC
Brown et al. (1983a)	34 RD	34 m/0 f	10–12	VGS	*N* (non-) predictive saccades	Overall no differences between NC and RD,
	33 NC	33 m/0 f	10–12			some indication total *N* saccades NC>RD
Brown et al. (1983b)	34 RD	34 m/0 f	10–12	VGS (saccadic, predictive and unpredictive ramp tracking)	Latency, *N* and amplitude saccades, gain, velocity, *N* saccades, regressions	ns
	35 NC	35 m/0 f	10–12			
De Luca et al. (1999)	10 RD	8 m/2 f	10.1–17.1	VGS	Amplitude, *N* forward and backward corrective saccades	ns
	11 NC	Unknown	*M* = 11.8			
Dossetor and Papaioannou (1975)	10 RD	Unknown	6–15	VGS	Latency	RD > NC (p < .01), RD children faster when saccades are directed to the left, opposite pattern for control children
	10 NC	Unknown	6–15			
Eden et al. (1994)	26 RD	Unknown	10.9–12.2	VGS	Accuracy	RD < NC (p < .05)
	39 NC	Unknown	10.2–12.1	SPEM	Qualitative assessment of smoothness	RD poorer performance than NC (p < .002)
Fischer and Hartnegg (2000a)	262 RD	Unknown	7–17	VGS	*N* intrusive saccades	RD > NC
	99 NC	Unknown	7–17	AS	*N* intrusive saccades	RD > NC
Fischer and Hartnegg (2000b)	85 RD	60 m/25 f	8–15	VGS	Latency	RD better after training, comparable to NC
				AS	*N* (uncorrected) errors, latency antisaccades, correction times, *N* misses	RD better after training on all DVs, comparable to NC
Fischer and Weber (1990)	20 RD	Unknown	9–17	VGS	*N* express saccades, *N* fast regular saccades	*N* express saccades: RD > NC. *N* fast regular: RD < NC
	17 NC	Unknown	9–17			
Jeřábek and Krejčová (1991)	52 RD	46 m/6 f	*M* = 10.5	VGS	Dysmetric saccades, sponteneous nystagmus, gaze direction nystagmus	61% of RD abnormal. Dysmetric saccades in 29% of RD. Sponteneous nystagmus in 11%. Gaze direction nystagmus in 12%
	41 NC	Unknown	M = 10	SPEM	Irregularity	RD more irregular than NC
Leisman et al. (1978)	20 RD	19 m/1 f	7–11	VGS	Latency	ns
	20 NC	16 m/4 f	7–11			
Leisman and Schwartz (1978)	20 RD	19 m/1 f	7–11	VGS	Amplitude, duration, mean angular velocity	ns
	20 NC	16 m/4 f	7–11	SPEM	Amplitude, duration, mean angular velocity	ns
Olson et al. (1983)	34 RD	27 m/7 f	8.3–13.8	VGS	*N* small and large regressive and progressive saccades	ns
	36 NC	31 m/5 f	8.3–13.8			
Pavlidis (1985)	13 RD	12 m/1 f	8–13	VGS	*N* total, regressive and progressive saccades	*N* total and regressive saccades: RD > NC (p < .001) *N* progressive saccades: NC > RD
	10 NC	6 m/4 f	7–10			
Pavlidis (1981)	12 RD	Unknown	10–16	VGS	*N* total, regressive and progressive saccades	All DVs: RD > NC (p < .001)
	12 NC	Unknown	10–16			

(*continued on next page*)

Table 1 (*continued*)

Authors	*N*	Gender	Age (years)	Eye movement tasks	Dependent variables	Results
Petri and Anderson (1980)	16 RD	13 m/3 f	6–11	VGS	Saccade duration-amplitude relationship	ns
	18 NC	12 m/6 f	6–11			
Raymond et al. (1988)	6 RD	4 m/2 f	8.8–13.3	VGS	*N* and amplitude undershoot, latency	ns
	6 NC	0 m/6 f	9.9–10.1			
Stanley et al. (1983)	15 RD	12 m/3 f	11–13	VGS	*N* regressions and corrections	ns
	15 NC	9 m/6 f	11–13			
Ygge et al. (1993)	86 RD	68 m/16 f	9	VGS	Dysmetria, latency, velocity	ns
	86 NC	68 m/16 f	9	SPEM	Tracking ability, *N* saccadic intrusions	ns
Childhood-onset schizophrenia						
Friedman et al. (1993)	6 psychosis	Unknown	Adolescents	SPEM	Gain, *N* corrective catch-up saccades,% time tracking, corrective catch-up saccade amplitude	Corrective catch-up saccade amplitude: psychosis > NC ($p = .02$). Other DVs ns
	11 NC	Unknown	Adolescents			
Gordon et al. (1994)	10 Schizophrenia	Unknown	10–18	SPEM	Gain, covered distance of total, catch-up, and intrusive saccades, *N* intrusive saccades	Gain: schizophrenia < NC ($p < .01$). Covered distance: schizophrenia > NC ($p < .01$). *N* intrusive saccades: schizophrenia > NC
	12 NC	Unknown	10–18			
Jacobsen et al. (1996)	17 Schizophrenia	10m/7 f	10–18	SPEM	Gain, root mean square error,% time tracking, *N* catch-up, anticipatory and back-up saccades, *N* square wave jerks, amplitude catch-up, anticipatory and back-up saccades	Gain and % time tracking: schizophrenia < NC ($p < .05$ and $< .0001$). *N* catch-up saccades and *N* square wave jerks: ns. All other DVs schizophrenia > NC ($p < .01$)
	22 NC	16 m/6 f	9–18			
Karatekin and Asarnow (1998a)	13 schizophrenia	7 m/6 f	*M* = 14.4	VGS	Latency	ns
	38 NC	18 m/20 f	M = 14.1			
Kumra et al. (2001)	29 Schizophrenia	16 m/13 f	6–18	SPEM	Qualitative impairment score, gain, root mean square error, *N* total, catch-up, anticipatory and large anticipatory saccades	Qualitative impairment score and gain: schizophrenia and psychosis > NC ($p = .001$) Root mean square error: schizophrenia and psychosis > NC ($p = .003$). *N* total and catch-up saccades: schizophrenia > NC ($p = .03$). Other DVs ns
	26 Psychosis	22 m/4 f	6–18			
	38 NC	24 m/14 f	6–18			
Mather (1985)	9 at-risk	Unknown	12–19	VGS	Latency, amplitude, accuracy, *N* double-jump saccades	*N* double-jump saccades: at-risk > NC ($p = .03$).Other DVsns
	8 NC	Unknown	15–20	SPEM	Latency, *N* and amplitude of intrusive saccades	*N* intrusive saccades: at-risk > NC ($p = .03$). Other DVs ns
Pollack and Krieger (1958)	15 Schizophrenia	Unknown	7–9	SPEM	Abnormality	No differences between schizophrenic and NC
	9 NC	Unknown	3–10			
Ross (2003)	49 Schizophrenia	35 m/14 f	6–15	SPEM	Gain, *N* catch-up, leading and large anticipatory saccades	Gain: schizophrenia < NC. *N* leading and anticipatory saccades: schizophrenia > NC. *N* leading saccades: at-risk > NC. *N* catch-up saccades: ns
	60 at-risk	29 m/31 f	6–15			
	80 NC	38 m/42 f	6–15			
Ross et al. (2005)	45 Schizophrenia	33 m/12 f	5.8–16.0	MGS (delay 1–3 s)	*N* anticipatory saccades, latency, accuracy	*N* anticipatory saccades: schizophrenia > NC ($p < .001$). Accuracy: schizophrenia < NC ($p = .04$).
	64 at-risk	35 m/29 f	5.8–16.0			
	84 NC	39 m/45 f	5.8–16.0			Latency: ns
Ross et al. (1996)	13 at-risk	5 m/8 f	6–15	SPEM	Gain, root mean square error, *N* and amplitude	Gain: at-risk < NC ($p = .04$). Root mean square
	19 NC	9 m/10 f	6–15	Catch-up and anticipatory saccades	Error: at-risk > NC ($p = .04$). *N* anticipatory saccades: at-risk > NC ($p = .002$). Other DVs ns	
Ross et al. (1999)	10 Schizophrenia	7 m/3 f	7–15	SPEM	% of total eye movement due to anticipatory saccades	Anticipatory saccades: schizophrenia > NC ($p < .001$), at-risk > NC ($p = .003$)
	13 at-risk	5 m/8 f	6–15			
	19 NC	9 m/10 f	6–15			

Table 1 (*continued*)

Authors	*N*	Gender	Age (years)	Eye movement tasks	Dependent variables	Results
Schiffman et al. 2006	90 at-risk	Unknown	11–13	Visual pursuit movements (fast, smooth, accurate, and	Jerky movements, fixation losses, inaccurate speed (combined as normal versus abnormal) parallel motion)	ns
	82 NC	Unknown	11–13			
Schreiber et al. (1997)	21 at-risk	10 m/11 f	10–18	VGS	*N* hypometric and hypermetric saccades, *N* non-fixations, *N* omissions	*N* hypometric saccades: at-risk > NC ($p < .05$)
	21 NC	10 m/11 f	10–18			Other DVs ns.
Tourette's syndrome (TS)						
Bollen et al. (1988)	28 TS	21 m/7 f	8–15	VGS towards stationary and newly appearing targets	Latency, peak velocity	ns
	norm values			Fixation during distraction	*N* errors, square wave jerks	ns
				SPEM	Gain	ns
Jackson et al. (2007)	7 TS	5 m/2 f	11–17	VGS and AS (mixed) with and without cues	*N* errors, latency	ns main effects of group, but TS had less difficulty switching between tasks
	12 NC	Unknown	11–17			
Mostofsky et al. (2001b)	14 TS	14 m/0 f	8.4–14.6	VGS	Latency, coefficient of variation of latency	Latency: TS ⩾ NC ($p = .07$). Other DV ns
	10 NC	10 m/0 f	8.1–12.6	AS	*N* errors	ns
				MGS (delay 4.5–5 s)	*N* anticipatory saccades, accuracy (gain)	ns
Mueller et al. (2006)	9 TS	8 m/1 f	9–16	VGS and AS (mixed) with cues	*N* errors, latency	*N* errors and latency: TS = NC on nonswitch trials, but TS perform better ($p < .05$) on switch trials for *N* errors and latency
	19 NC	Unknown	9–16			
Narita et al. (1997)	1 TS	1 m	13	VGS	Gain	Saccades were hypometric
				AS	*N* errors	10 of 10 trials erroneous saccades.
				SPEM	Gain, *N* intrusive saccades	Gain was normal, frequent intrusive saccades
Nomura et al. (2003)	79 TS	79 m/0 f	6–12	VGS	Latency, amplitude, peak velocity	ns
	19 NC	19 m/0 f	6–12	MGS (delay unknown)	Latency, amplitude, peak velocity *N* distracted saccades	TS made less distracted saccades than NC ($p < .05$), but TS were further removed from target ($p < .05$). Other DVs ns
Childhood-onset-obsessive compulsive disorder (OCD)						
Rosenberg et al. (1997a)	18 OCD	9 m/9 f	8.8–16.9	VGS	Accuracy, latency, peak velocity	ns
	18 NC	9 m/9 f	8.7–16.8	AS	*N* errors, latency, accuracy	*N* errors: OCD > NC ($p = .003$). Oher DVs ns
				MGS (delay 1–8 s)	*N* anticipatory saccades, accuracy, latency	*N* anticipatory errors: OCD ⩾ NC ($p = .07$). Other DVs ns
				Predictive saccadic task	accuracy, latency	ns
Anxiety and depression						
Hardin et al. (2007)	16 Anxiety	9 m/7 f	9–17	AS under reward, punishment, and neutral conditions with low to high saliences	*N* errors, latency	*N* errors: depression > NC ($p = .005$), anxiety in between depression and NC, not influenced by condition. Latency: no main effect, but anxious less improvement under reward and punish-ment than NC with depression in between
	11 Depression	5 m/6 f	9–17			
	30 NC	15 m/15 f	9–17			
Jazbec et al. (2005)	11 Anxiety	8 m/3 f	9–17	AS under reward, punishment, and neutral conditions	*N* errors, latency, peak velocity	*N* errors: ns. Latency: NC improved with reward or punishment ($p = .05$), no effect of condition on depression ($p = .52$), anxiety worsened with punishment ($p = .03$). Peak velocity: NC improve with reward or punishment ($p = .01$), but anxious and depression do not ($p = .84$ and .89)
	12 Depression	4 m/8 f	9–17			
	28 NC	13 m/15 f	9–17			

Note. NC, normal controls; N, number; ADHD, attention-deficit/hyperactivity disorder; OCD, obsessive compulsive disorder; DVs, dependent variables; ns, not significant; AS, antisaccades; VGS, visually guided saccades; MGS, memory guided saccades; SPEM, smooth pursuit eye movement.

Fischer, & Hartnegg, 2002) who tested boys with ADHD on medication first, then later without medication, or the reverse. Participants showed beneficial effects of methylphenidate on response inhibition, which was later confirmed for both subtypes of ADHD (O'Driscoll et al., 2005). This indicates that the mechanisms responsible for the deficits in antisaccade performance are related to the areas influenced by methylphenidate.

4.1.5. Visually guided saccades

Based on the prosaccade/visually guided saccade data collected during antisaccade experiments, it is clear that VGS latencies are consistently more variable in ADHD compared to controls (Mostofsky et al., 2001a, 2001b; Munoz et al., 2003). The study with the largest sample of ADHD patients ($N = 76$) found longer and more variable latencies, reduced peak velocity and increased durations of VGS compared to controls (Munoz et al., 2003). With respect to augmented prosaccade latencies, this was found to be non-significant in studies with a smaller sample (Hanisch et al., 2006; Karatekin & Asarnow, 1998a; Mostofsky et al., 2001a; O'Driscoll et al., 2005), but significant in another study (Klein et al., 2003). Taken together, latency of VGS is more variable and possibly slower in children with ADHD, presumably indicating that children with ADHD have difficulty in regulating processes of saccade initiation.

4.1.5.1. Memory guided saccades. MGS in ADHD have been reported in five studies (Castellanos et al., 2000; Mostofsky et al., 2001a, 2001b; Rommelse et al., 2008; Ross, Hommer, Breiger, Varley, & Radant, 1994). These studies have revealed inconsistent results with respect to the accuracy of saccades towards the memorized target. Normal accuracy was observed in three studies (Mostofsky et al., 2001a, 2001b; Ross et al., 1994), while deficits in accuracy were reported in two studies (Castellanos et al., 2000; Rommelse et al., 2008). A possible explanation for these inconsistent findings might be differences in the paradigms used, mainly the large range in delay periods used in various studies, which varied between 0.8 and 7 s. Consistently, however, all five studies reported more anticipatory saccades in patients with ADHD compared to controls (Castellanos et al., 2000; Mostofsky et al., 2001a, 2001b; Rommelse et al., 2008; Ross et al., 1994), indicating problems with inhibitory control in children with ADHD in MGS. Latencies of MGS were reported in four studies, and were found to be normal in ADHD in three of four studies (Castellanos et al., 2000; Rommelse et al., 2008; Ross et al., 1994), suggesting response initiation to be normal. Only one study reported saccade metrics during MGS, in which boys with ADHD tended to overshoot their saccades relative to controls (Rommelse et al., 2008). This was hypothesized to be caused by dysfunctions of the cerebellum and/or related to changes in the dopaminergic transmission in ADHD. Further research is needed to establish the prevalence of eye movement deviancies on the memory guided saccade paradigm in ADHD.

4.1.5.2. Smooth pursuit eye movements. There have been only two studies investigating SPEM in children with ADHD. Therefore, there seems to be no general agreement whether children with ADHD have fundamental problems in SPEM tasks. Although earlier studies have described difficulties in suppressing intrusive saccades during SPEM (Bala et al., 1981; Bylsma & Pivik, 1989; Shapira, Jones, & Sherman, 1980), these studies adopted inclusion criteria that predated the publication of the DSM-IV. In a more recent study, it was found that children with ADHD were affected in a global measure of SPEM, the root mean square error (Jacobsen et al., 1996). This measure is an index of the extent to which the eyes do not reproduce target motion. The effect on the global measure was not observed in a SPEM experiment investigating only girls with ADHD, although observed was a non-significant tendency toward a greater root mean square error in ADHD girls compared to control girls (Castellanos et al., 2000).

4.1.5.3. Variability in ADHD. As noted earlier, an increased variability of responding in ADHD patients compared to controls is one of the most robust findings in the ADHD literature (Kuntsi et al., 2006; Leth-Steensen et al., 2000). Results on variability measures have been included in four of the discussed studies in three different paradigms. All four of these studies have found an increased variability in children with ADHD compared to controls. Increased variability has been observed in antisaccades (Munoz et al., 2003), visually guided saccades (Mostofsky et al., 2001a, 2001b; Munoz et al., 2003), and memory guided saccades (Rommelse et al., 2008).

4.1.5.4. Conclusions on ADHD. Overall, there are several studies that have investigated eye movements in children with ADHD. They have primarily focused on testing executive functions to verify the hypothesis that intentional motor systems as mediated by the prefrontal areas are affected in ADHD. Indeed, a failure of top-down regulation has been found in AS, in which elevated numbers of directional errors are observed and in MGS, in which more anticipatory errors are observed. Furthermore, a wide variety of paradigms have found more intrusive saccades in ADHD. Intrusive saccades are defined as inappropriate eye movements and may also be regarded as tapping response inhibition, although it is not a failure to inhibit a response to an external stimulus, but rather a primary failure of visual fixation. More intrusive saccades are reported in a variety of tasks, such as the go-nogo tasks (Castellanos et al., 2000), an ocular fixation task (Gould, Bastain, Israel, Hommer, & Castellanos, 2001), a visual search task (Van der Stigchel et al., 2007), memory saccade tasks (Rommelse et al., 2008), a task requiring prolonged fixation (Munoz et al., 2003) and during pro- and antisaccade tasks (Klein et al., 2003). In line with results from other modalities, an increased variability is consistently observed in children with ADHD. These results indicate a consistent reduction in ability to suppress unwanted saccades and to control voluntary behavior in ADHD. These findings lend support for the presence of inhibition deficits in children with ADHD and suggest functioning of frontal areas like the DLPFC is impaired in ADHD. Since basic eye movement control may also be deviant, dysfunctions of the frontal eye fields and cerebellum may be implicated.

4.2. Oppositional deviant disorder and conduct disorder (ODD and CD)

4.2.1. Clinical manifestation of ODD and CD

ODD is characterized by a recurrent pattern of negativistic, defiant, disobedient, and hostile behaviors toward authority figures persisting for at least six months. A proportion of children with ODD later develop CD (Loeber, Burke, Lahey, Winters, & Zera, 2000), characterized by a repetitive and persistent pattern of behaviors in which the basic rights of others and major age-appropriate societal norms or rules are violated (APA, 1994; Loeber et al., 2000). ODD and CD frequently co-occur with ADHD (Loeber et al., 2000).

4.2.2. Neurological and cognitive problems in ODD and CD

Frontal lobe dysfunctioning and damage have been frequently associated with violence and aggressive behavior, especially when located in the orbitofrontal cortex (Burke, Loeber, & Birmaher, 2002). Furthermore, impairments in the functioning of the amygdala have been associated with diminished interpretation of social cues (like facial expressions). Abnormalities in the connections between the amygdala and prefrontal lobes may be associated with a negative affective style in children with disruptive behavior disorders (Burke et al., 2002). Cognitively, impairments in inhibition appear important in disruptive behavior disorders (Burke et al.,

2002), although impairments in working memory have also been found independent of possible confounding ADHD in one study (Séguin, Boulerice, Harden, Tremblay, & Pihl, 1999), but not in another study (Oosterlaan, Scheres, and Sergeant, 2005).

4.2.3. Eye movement studies in ODD and CD

We were unable to locate any eye movement studies specifically targeting children with disruptive behavior disorders. It is highly likely, however, that a (substantial) proportion of the children with ADHD participating in eye movement studies had a comorbid disruptive behavior disorder which may have contributed to the findings of eye movement deficits found in these studies. Given that disruptive behavior disorders and ADHD frequently co-occur, common underlying pathology (such as frontal lobe dysfunctions, inhibitory problems, and/or generalized executive functioning problems) may be expected. In line with this, it is reasonable to hypothesize that eye movement studies in children with disruptive behavior disorders will show deficits similar to those found in ADHD (i.e., more directional errors on antisaccade tasks, slower and more variable latencies on VGS tasks, more anticipatory saccades on memory guided saccade tasks, and more intrusive saccades in general). Clearly, work needs to be done in this area of child and adolescent pathology.

4.3. Autism spectrum disorders (ASD)

4.3.1. Clinical manifestation of ASD

ASD entails a spectrum of disorders, namely autism, Asperger's disorder, and pervasive developmental disorder-not otherwise specified (PDD-NOS), characterized by significant social deficits, repetitive behaviors, and restricted interests (Akshoomoff, 2005; DiCicco-Bloom et al., 2006). Social deficits include a marked impairment in the use of non-verbal behaviors (eye contact and facial expressions), a failure to develop age-appropriate peer relationships, a lack of spontaneous enjoyment and interest sharing with other people, and a lack of social or emotional reciprocity (APA, 1994). Repetitive behaviors and restricted interests may include a preoccupation with one or more stereotyped and restricted patterns of interests that is abnormal in intensity or focus, inflexible and non-functional routines, stereotyped and repetitive motor mannerisms (like hand flapping), and persistent preoccupation with parts of objects (APA, 1994). Onset of ASD is by definition before the age of three (APA, 1994). Unlike Asperger's syndrome, autism is also characterized by qualitative impairments in verbal communication, reflected by delay in (or total lack of) the development of spoken language, inability to initiate or sustain a conversation with others, stereotyped or repetitive use of language, and lack of social imitative or make-believe play appropriate for developmental level (APA, 1994; DiCicco-Bloom et al., 2006). PDD-NOS is often diagnosed, when a child does not meet the full criteria for autism or displays subthreshold symptomatology (APA, 1994). Once considered as a rare disorder affecting only 0.05% of children (APA, 1994), current estimates indicate a prevalence around 0.5–0.6% (DiCicco-Bloom et al., 2006; Rutter, 2005). ASD is more common in boys than girls (APA, 1994) and is strongly genetically determined. In most cases of autism, mental retardation is present, as well as a range of behavioral symptoms like hyperactivity, a short attention span, impulsivity, and aggressiveness (APA, 1994).

4.3.2. Neurological and cognitive problems in ASD

ASD is associated with an abnormal enlargement of brain volume, mainly during early childhood (DiCicco-Bloom et al., 2006). Growth abnormalities have been reported for the cerebellum, cerebrum, amygdala, and possibly hippocampus (Akshoomoff, 2005; DiCicco-Bloom et al., 2006). Brain enlargement reflects both cerebral and cerebellar, and both white and gray matter (DiCicco-Bloom et al., 2006). The most prominent cognitive problems in ASD are impairments in social skills and language, but impairments in specific aspects of executive functioning, such as abstracting rules, shifting attention, learning from feedback, and maintaining focus on multiple aspects of information processing during decision making, have also been reported (Akshoomhoff, 2005).

4.3.3. Eye movement studies

Given that the major impairments of autistic children lie in social skills and communication, the majority of eye movement studies in autism have focused on scan patterns of social scenes and facial expressions. Only seven studies have investigated the paradigms discussed in the present review, aiming to assess basic oculomotor behavior and frontal circuitry in children with autism.

4.3.3.1. Antisaccades. Two studies have focused on antisaccade performance in children with autism (Goldberg et al., 2002; Luna, Doll, Hegedus, Minshew, & Sweeney, 2007). Both studies observed more directional errors in autistic participants compared to controls. Furthermore, this basic deficit in response inhibition was present throughout development (Luna et al., 2007). The results were inconsistent as to whether children with autism are slower to execute a correct antisaccade: one study failed to find an effect (Goldberg et al., 2002), whereas the other study found that young autistic participants were even faster to initiate a correct antisaccade than controls (Luna et al., 2007).

4.3.3.2. Visually guided saccades. Children with autism do not seem to respond slower than age-matched controls on simple VGS. Three of four studies did not find an effect of latency on VGS (Luna et al., 2007; Rosenhall, Johansson, & Gillberg, 1988; Van der Geest, Kemner, Camfferman, Verbaten, & Van Engeland, 2001), although one study did observe children with autism to be slower than normals (Goldberg et al., 2002). Therefore, it seems that the dynamics of saccades are normal, although more studies are needed to confirm this. Whether children with autism have difficulties with attentional engagement is unclear. By computing the gap effect, the disengagement of attention and fixation can be measured. Two studies reported this and have found conflicting results: one study found a significant gap effect (Van der Geest et al., 2001) and one study did not (Goldberg et al., 2002). A third study found latency differences in a group of young children with autism on shift trials in which the fixation point remained on screen, but no latency differences on disengage trials, when the fixation point was removed before the peripheral target was presented (Landry & Bryson, 2004), suggesting difficulties disengaging attention from the fixation point. It should be noted that in this study there was no explicit task instruction to look at the peripheral stimulus. Overall, VGS appear normal in autism and there is insufficient evidence to claim difficulties with attentional engagement within the oculomotor domain for children with autism.

4.3.3.3. Memory guided saccades. The two studies that focused on AS are the only ones that investigated MGS in children with autism (Goldberg et al., 2002; Luna et al., 2007). The results of these studies are inconsistent, however. On the one hand, Luna and colleagues (2007) found deficits in accuracy and no effect of saccade latency in the young group (8–17 years) of their clinical population. On the other hand, Goldberg and colleagues (2002) found no effect of accuracy of MGS but slower saccade latency compared to controls. They also reported more response suppression saccades, a variable not reported by Luna and colleagues. These findings suggest that impairments in MGS may be present in children with autism, although the specificity of deficits is unclear.

4.3.3.4. Smooth pursuit eye movements. One study reported SPEM in children with PDD and observed no deficits (Kemner, Van der Geest, Verbaten, & Van Engeland, 2004). Only two relatively dated studies have focused on SPEM in children with autism (Rosenhall et al., 1988; Scharre & Creedon, 1992). Both studies reported impairments in SPEM in autism, since the vast majority of autistic children were unable to perform the experiment, making saccadic instead of smooth movements. Fundamental problems with pursuit tasks were later confirmed in a study that included participants with autism of different age groups (Takarae, Minshew, Luna, Krisky, & Sweeney, 2004). The authors analyzed which deficits in SPEM occurred in the group younger than 16 years old. They found lower gain and less accurate initial catch-up saccades (see glossary; saccades to catch-up with the moving target when gain was too low), but only for targets moving into the right visual field. Deficits in the closed-loop were specific to adults with autism. More studies on SPEM are necessary in order to draw firm conclusions on SPEM performance in children with autism, although it seems that some impairments are present.

4.3.3.5. Conclusions for ASD. It is difficult to draw firm conclusions based on the above eye movement studies in ASD, because a large body of the findings is inconsistent. However, an elevated number of antisaccade errors has been consistently reported. Because successful performance on the antisaccade task relies heavily on frontal structures, one possible interpretation is that the social difficulties in autism result primarily from disturbances in the frontal cortical areas. Since smooth pursuit also appears abnormal, various non-cortical areas may also be involved in the neuropathology of ASD.

4.4. Reading disorder (RD)

4.4.1. Clinical manifestation of RD

RD, also know as dyslexia, manifest itself as a level of reading achievement (i.e., accuracy, speed, and/or comprehension) that falls significantly below that expected given the child's age, intelligence, and education (APA, 1994). Symptoms of RD include an inability to distinguish among common letters and to associate common phonemes with letter symbols. RD is seldom diagnosed before the end of kindergarten because formal reading instruction usually does not begin until this point (APA, 1994). RD is heterogeneous and various attempts have been made to identify more homogeneous subtypes of RD (Bakker, 1979; Stanovich, Siegel, & Gottardo, 1997). Often the division between phonological and surface RD is made, with the first representing a true developmental deviancy and the latter representing a form of developmental delay (Stanovich et al., 1997). Phonological RD is a persistent, chronic condition (Shaywitz & Shaywitz, 2005). Nevertheless, adolescents and adults with RD may learn to read words accurately, although not fluently or automatically (Shaywitz & Shaywitz, 2005). Prevalence rates of RD vary between 4% (APA, 1994) up to 17% (Shaywitz & Shaywitz, 2005), depending on the criteria applied. RD is more common in boys than in girls (60–80% of patients is male) and runs in families (APA, 1994). Children with RD often have associated deficits in related domains, such as problems in oral language, writing, and mathematics. Comorbid ADHD and motor coordination problems are also frequently observed (Habib, 2000).

4.4.2. Neurological and cognitive problems in RD

Functional brain imaging studies have shown that the posterior brain systems in the left hemisphere fail to function properly during reading and non-reading tasks requiring visual processing (Shaywitz & Shaywitz, 2005). This results in the development of compensatory brain mechanisms, such as an increased activity in anterior sites (like the inferior frontal gyrus) and in analog posterior sites in the right hemisphere (Shaywitz & Shaywitz, 2005). Naturally, research in the cognitive field has focused on language processes. The phonological theory is most widely supported. This theory entails that speech is natural and inherent, but reading is acquired and must be taught (Shaywitz & Shaywitz, 2005). Children have to develop the insight that spoken words can be dissected into phonemes and that letters in a written word represent these sounds. Apparently, such awareness is largely missing in children with RD (Shaywitz & Shaywitz, 2005). Consequently, the child has difficulty decoding the word and then identifying it. Higher order cognitive and linguistic functions are generally intact (Shaywitz & Shaywitz, 2005). However, some evidence exists regarding problems in executive functions, such as verbal working memory and inhibition, in children with RD (Brambati et al., 2006; Van der Schoot, Licht, Horsley, Sergeant, 2000; Willcutt, Pennington, Olson, Chhabildas, Hulslander, 2005).

4.4.3. Eye movement studies in RD

Since RD is by definition a selective impairment of reading related processes in the absence of deficits in non-reading higher order cognitive processes (Shaywitz & Shaywitz, 2005), the vast majority of eye movement studies in RD has focused on examining and manipulating eye movements during reading tasks. These studies lie outside the scope of this review and will not be described here. Furthermore, many studies have focused on measuring ophthalmological problems in children with RD. Overall up to 75% of children with RD have some sort of ophthalmological problem, such as problems with tracking or motion perception, causing disturbed binocular vision (Habib, 2000). Elementary perceptual abnormalities have been reported in RD children, such as a reduced processing speed of visual information (longer visual persistence at low spatial frequency and a slower flicker fusion rate) and an altered contrast sensitivity (Habib, 2000). Seen in this context, it brings no surprise that the studies relevant to this review have almost all focused on measuring VGS or SPEM. Only two studies measured AS and to our knowledge no studies have used MGS.

4.4.4. Antisaccades

As far as the authors are aware, only two studies compared the performance of RD children to that of controls on an antisaccade task (Biscaldi, Fischer, & Hartnegg, 2000; Fischer & Hartnegg, 2000a). Both studies reported an impaired performance of RD children, reflected by an increased number of directional errors and misses, and a lower number of corrections (Biscaldi et al., 2000) and more intrusive saccades (Fischer & Hartnegg, 2000a). However, Fischer and Hartnegg (2000b) also showed that the performance of RD children on an antisaccade task could be improved by training, even to the extent that performance was comparable to that of controls. Thus, some evidence exists indicating impaired inhibitory processing in RD, but these problems can be overcome by training.

4.4.5. Visually guided saccades

Saccades are essential during reading. While reading, the eyes do not move continuously from left to right along the printed line, but proceed by successive saccades and fixations (Pavlidis, 1980). Even though VGS are not similar as saccades during reading (i.e., VGS are mainly elicited by the onset of a peripheral stimulus), the majority (26 of 27 studies) traced on eye movements on non-linguistic tasks in RD children have focused on measuring VGS. However, even though this is a widely studied topic in RD, no consensus has been reached as to whether or not VGS are impaired in RD. This is largely due to differences in task paradigms and methods applied, which is probably related to the wide time span in

which the studies were conducted (from 1975 to 2006). This has also consequences for the kind of dependent variables studied, which vary considerably between studies. Nevertheless, some consistent variables and results are present.

Ten studies reported latency. Seven of these did not find any latency differences between RD children and controls (Adler-Grinberg & Stark, 1978; Biscaldi, Gezeck, & Stuhr, 1998; Black, Collins, De Roach, & Zubrick, 1984a; Brown et al., 1983b; Leisman, Ashkenazi, Sprung, & Schwartz, 1978; Raymond, Ogden, Fagan, & Kaplan, 1988; Ygge, Lennerstrand, Rydberg, Wijecoon, & Pettersson, 1993). Three studies did find differences in latency, of which two reported RD children to have slower saccade latencies than controls (Biscaldi et al., 2000; Dossetor & Papaioannou, 1975) and one study reported the reverse finding (Bednarek, Tarnowski, & Grabowska, 2006). Enhanced variability in latency has been found in one study (Biscaldi et al., 1998). However, the overall pattern of findings suggests saccade latency to be normal in RD children. The same conclusion can be drawn for velocity, a variable related to latency. Six studies reported (peak) velocity. Five studies found no differences in peak velocity between RD children and controls (Adler-Grinberg & Stark, 1978; Black, Collins, De Roach, & Zubrick, 1984c; Black et al., 1984a; Leisman & Schwartz, 1978; Ygge et al., 1993) and one study reported RD children to be faster, when directing their saccades to the left (Biscaldi, Fischer, & Aiple, 1994). The relation between duration and amplitude (which can be seen as an index of velocity) has been reported to be normal (Petri & Anderson, 1980). Taken together, the findings indicate that the speed of VGS is normal in RD children.

Six studies that have reported amplitude of VGS, all reported this to be normal in RD (Adler-Grinberg & Stark, 1978; Biscaldi et al., 1998; Brown et al., 1983b; De Luca, Di Pace, Judica, Spinelli, & Zoccolotti, 1999; Leisman & Schwartz, 1978; Raymond et al., 1988). It thus does not appear that RD children under- or overshoot the target stronger compared to controls, when directing their saccades.

A variety of saccades has been studied in RD. No consistent terminology has been used in describing the kind of saccades studied, which is probably related to the wide time span in which the studies were conducted. We will describe here the most frequently studied ones. Six studies have reported a comparable type of saccade, namely directed at the target before allowed to do so. However, this type of saccade has been variously labeled 'predictive', 'forward', 'anticipatory', or 'progressive' saccade. The studies reporting on a deviant high frequency of this type of saccades in RD are in the minority (Biscaldi et al., 1998; Pavlidis, 1981, 1985) compared to the studies reporting on no difference between children with RD and controls (Black et al., 1984c; Brown et al.,1983a, 1983b; De Luca et al., 1999; Olson, Kliegl, & Davidson, 1983; Stanley, Smith, & Howell, 1983). The frequency of a saccade directed backwards to the target when overshooting the target (variously labeled corrective, backward, or regressive saccade) was generally found to be normal in most (Biscaldi et al., 1998; Black et al., 1984c; Brown et al.,1983a, 1983b; De Luca et al., 1999; Olson et al., 1983; Stanley et al., 1983) though higher in some studies (Pavlidis, 1981, 1985). Higher frequencies of fast and slow saccades (Biscaldi et al., 1998), Express saccades (Fischer & Weber, 1990), big saccades (Black, Collins, De Roach, & Zubrick, 1984b), dysmetric saccades (Jeřábek & Krejčová, 1991), and intrusive saccades (Fischer & Hartnegg, 2000a) have been found in RD. However, these saccades were either only reported in one study or findings of deviancy were disconfirmed in another study, as is the case for big saccades (Black et al., 1984c) and intrusions (Bogacz, Mendilaharsu, & De Mendilaharsu, 1974). Probably due to inconsistent terminology, no conclusion can be drawn from these findings. It appears, however, that RD may be associated with an increased frequency of saccades in general made during VGS (Brown et al., 1983a; Pavlidis, 1981, 1985).

4.4.6. Smooth pursuit eye movements

SPEM are not used in reading (Adler-Grinberg & Stark, 1978). This would only occur if the text was moving, while in fact, the reading material must be held stationary to read efficiently (Adler-Grinberg & Stark, 1978). Nevertheless, quite a few studies have investigated SPEM in RD. From the seven studies traced that have targeted SPEM in RD, five have found dysfunctions (Adler-Grinberg & Stark, 1978; Black et al., 1984b; Bogacz et al., 1974; Eden, Stein, Wood, & Wood, 1994; Jeřábek & Krejčová, 1991) while two have not (Leisman & Schwartz, 1978; Ygge et al., 1993). Abnormalities in RD include an increased frequency of (big) saccades (Adler-Grinberg & Stark, 1978; Black et al., 1984b), more abnormal saccades (Bogacz et al., 1974), reduced smoothness (Eden et al., 1994), and increased irregularity (Jeřábek & Krejčová, 1991). Velocity of pursuit, when reported, appears normal in RD (Black et al., 1984b; Leisman & Schwartz, 1978; Ygge et al., 1993). Overall, SPEM in children with RD appear to be characterized by an increased frequency of (abnormal) saccades, resulting in a decreased smoothness, but velocity appears intact in RD.

4.4.7. Conclusions for RD

Literature on non-linguistic eye movement paradigms in RD is dated, resulting in considerable discrepancies between studies in applied terminology, paradigms, and technology. The majority of studies have been conducted in the seventies and eighties when the advanced eye movement technology used currently was not available. This makes drawing of firm conclusions concerning eye movement dysfunctions in RD hazardous. Nevertheless, some results appear consistent. RD appears associated with a somewhat increased number of saccades during VGS tasks and SPEM, though exactly what type of saccades is unclear. These findings may pinpoint problems in the integration of the saccadic and pursuit networks. Antisaccade task performance may also be slightly deficient in RD, as evidenced by an increase number of errors and misses, reduced number of corrections, and more intrusive saccades, but these findings await replication, since only two studies have reported these findings. It is safe to say that eye movement research in RD is in urgent need of modern studies, using the latest technology to analyze eye movement performance. Standardized terminology would allow for a better comparison between studies with RD children and other types of pathology. The reason why non-linguistic eye movements in RD have not been investigated in recent studies, may be because deficits in cognitive processes that are directly related to reading are easier to translate to an etiological explanation for RD. The direct impact of deficits in non-linguistic eye movement on reading is more complicated.

4.5. Childhood-onset schizophrenia

4.5.1. Clinical manifestation of childhood-onset schizophrenia

The essential features of schizophrenia are a mixture of positive and negative symptoms that have been present for a significant portion of time during a 1-month period, with some symptoms persisting for at least 6 months and significantly interfering with social and/or occupational functioning (APA, 1994). Positive symptoms reflect an excess or distortion of normal functions and include delusions, hallucinations, disorganized thinking and/or speech, and severely disorganized or catatonic behavior (APA, 1994). Negative symptoms reflect a diminution or loss of normal functions and include affective flattening, alogia (diminution of thoughts reflected in decreased fluency and productivity of speech), and avolition (inability to initiate or persist in goal-directed activities) (APA, 1994). Schizophrenia usually has its onset in late adolescence and early adulthood and is often preceded by a variety of signs and symptoms (such as social withdrawal and loss of interest in school or work). Prevalence rates vary between 0.2%

and 2%. Schizophrenia is somewhat more common in males than females and strongly related to genetic factors, although discordance between monozygotic twins suggest environmental factors also to play a role (APA, 1994). Schizophrenia with childhood onset is rare: 50 times less frequent than adult-onset (Asarnow, Tompson, & McGrath, 2004; Remschmidt, 2002; Ross, Heinlein, & Tregellas, 2006), though does exist and appears broadly similar in presentation to the adult-onset form (Asarnow et al., 2004). However, some specific diagnostic issues are related to childhood-onset schizophrenia. Disorganized speech may be difficult to diagnose before the age of seven, since the speech of young children is frequently less logical and coherent (Asarnow et al., 2004), as well as delusions and hallucinations, which may be less elaborate in children compared to adults (APA, 1994). Furthermore, a deterioration of functioning may be difficult to establish. Hence, comparing the child to his/her non-affected siblings may aid in determining a failure to achieve what would have been expected for the child (APA, 1994). The vast majority of children with schizophrenia suffer from comorbid disorders, such as ADHD, ODD, depression, and separation anxiety disorder (Ross et al., 2006). Compared to adulthood-onset, schizophrenia with childhood onset is more common among boys, more strongly associated with structural brain abnormalities, and has a poorer prognosis (APA, 1994).

4.5.2. Neurological and cognitive problems in childhood-onset schizophrenia

The most common structural brain abnormalities documented in schizophrenic adult patients are enlargement of the ventricular system and prominent sulci in the cortex. Other abnormalities have also been reported, such as a decreased temporal and hippocampal size, increased size of the basal ganglia (see glossary), and decreased cerebral size (APA, 1994). These abnormalities have also been found in childhood-onset schizophrenia. It appears that the reduction of total cerebral volume in children with schizophrenia is caused by a reduction of gray matter (Remschmidt, 2002). A variety of soft neurological signs have been associated with adult schizophrenia, such as poor motor development and coordination, and left/right confusion (APA, 1994; Remschmidt, 2002). A wealth of literature has been published on cognitive deficits in schizophrenic adult patients. Mainly problems in changing response set, focused attention, and formulation of abstract concepts have been found. Disorientation and memory impairment may also be present (APA, 1994). Studies specifically targeting cognitive functioning in children with schizophrenia have found an overall lower cognitive functioning (lower IQ), increased distractibility, and decreased verbal comprehension and perceptual organization. Significant delays in motor development and coordination have been reported in children with schizophrenia (Remschmidt, 2002).

4.5.3. Eye movement studies in childhood-onset schizophrenia

Eye movement studies in childhood-onset schizophrenia have targeted two groups of subjects: children having schizophrenia themselves and children at-risk for developing schizophrenia because their parent(s) and/or sibling(s) have schizophrenia. Even though this at-risk group may not portray the symptoms of schizophrenia, this group of children is informative, since they might develop schizophrenia at a later age. Eye movement dysfunctions assessed in this group might thus be informative precursors for schizophrenia.

4.5.4. Visually guided saccades

To our knowledge, only one study reported VGS in schizophrenic children and found no dysfunctions (Karatekin & Asarnow, 1998a). Two studies reported VGS in at-risk children. Mather (1985) analyzed the VGS of nine at-risk children, and found that at-risk children made more double-jump saccades. The latency, amplitude and accuracy of saccades appeared normal. Schreiber et al. (1997) analyzed the VGS performance of 21 at-risk children and reported an increased frequency of hypometric saccades in at-risks. The frequency of hypermetric saccades, non-fixations and omissions was normal. Thus, some slight deviations in VGS appear to be present in at-risk children but VGS studies in childhood-onset schizophrenia are clearly lacking and future research is needed.

4.5.5. Smooth pursuit eye movements

The vast majority of eye movement studies with schizophrenic patients (both children and adults) have focused on the assessment of SPEM. SPEM has a strong emphasis on oculomotor control and less on cognitive processes and is frequently used in schizophrenia given the motor development and coordination problems associated with the disorder. All eight studies that have administered a SPEM task in schizophrenic children (or those at-risk for developing the disorder) have found abnormalities on various measures on the task (Friedman, Schulz, & Jesberger, 1993; Gordon et al., 1994; Jacobsen et al., 1996; Kumra et al., 2001; Mather, 1985; Ross, 2003; Ross, Hommer, Radant, Roath, & Freedman, 1996; Ross et al., 1999). Six of the eight studies reported abnormalities in visual pursuit movements; two studies did not find pursuit abnormalities in 15 schizophrenic children (Pollack & Krieger, 1958) and 90 at-risk children (Schiffman et al., 2006). As the authors of the latter study indicated, their eye movement measures were probably not the same as assessed in SPEM tasks. SPEM tasks appear, therefore, sensitive in detecting eye movement abnormalities related to childhood-onset schizophrenia.

With respect to individual task measures, the most widely measured variable in SPEM is gain (ratio of eye velocity to the target velocity). All four studies on gain in children or adolescents with schizophrenia report a reduced gain (Gordon et al., 1994; Jacobsen et al., 1996; Kumra et al., 2001; Ross, 2003. Reduced gain has also been reported in one (Kumra et al., 2001) of two studies in children with psychosis (Friedman et al., 1993; Kumra et al., 2001) and in one (Ross et al., 1996) of two studies in at-risk children (Ross, 2003; Ross et al., 1996). These findings concur with findings of reduced gain in adult-onset schizophrenia (Gordon et al., 1994; Ross et al., 2002) and suggest a reduced pursuit velocity is a characteristic sign of pathology in (childhood-onset) schizophrenia.

Another variable in SPEM is the root mean square error. Two studies reported this measure in children with schizophrenia (and psychosis) and one study reported this measure in at-risk children, all reported an increased root mean square error (Jacobsen et al., 1996; Kumra et al., 2001; Ross et al., 1996. These findings are in line with reports in adult-onset schizophrenia (Gordon et al., 1994) and suggest that children with (a risk for developing) schizophrenia have difficulty in accurately pursuing a moving target.

Other task variables include various forms of saccades, such as corrective catch-up and back-up saccades, intrusive saccades (not directed at the moving target), and anticipatory saccades (leading) saccades. Studies vary in which type of saccades are reported. Five studies have reported the frequency of catch-up saccades (Friedman et al., 1993; Jacobsen et al., 1996; Kumra et al., 2001; Ross, 2003; Ross et al., 1996). Only one of these studies observed that children with schizophrenia make more catch-up saccades than controls (Kumra et al., 2001). Four studies did not find catch-up saccade differences between (at-risk) schizophrenics and controls. However, the amplitude of catch-up saccades appears to be larger in children with schizophrenia (or psychosis) in three of four studies (Friedman et al., 1993; Gordon et al., 1994; Jacobsen et al., 1996; Ross et al., 1996). The one study that reported back-up saccades documented an increased frequency and amplitude of these types of saccades in schizo-

phrenic children (Jacobsen et al., 1996). With respect to intrusive saccades, two studies that reported on this measure found children with schizophrenia and children at-risk had a larger number of intrusive saccades than controls (Gordon et al., 1994; Mather, 1985). Results are clear for anticipatory (leading) saccades, with five of six studies reporting an increased number and/or amplitude of these saccades in (at-risk) schizophrenics (Jacobsen et al., 1996; Kumra et al., 2001; Ross, 2003; Ross, Heinlein, Zerbe, & Radant, 2005; Ross et al., 1996, 1999). Taken together, frequent abnormalities in various types of saccades during SPEM are observed in children with schizophrenia and at-risk children. The most consistent finding is an increased frequency of anticipatory (leading) and intrusive saccades. Frequency of catch-up saccades appears to be normal. Insufficient data is available regarding the frequency of back-up saccades in childhood-onset schizophrenia.

4.5.6. Memory guided saccades

One large study reported MGS in children with schizophrenia and at-risk children (Ross et al., 2005). Children with schizophrenia made more anticipatory saccades and were less accurate in their saccades than controls. Latency appeared to be normal. No MGS deficits were found in at-risk children.

4.5.7. Conclusions for childhood-onset schizophrenia

Eye movement studies in childhood-onset schizophrenia have focused on measuring SPEM, probably to allow for comparison with adult-onset schizophrenia, since SPEM is the most frequently applied eye movement paradigm in adult patients (Ross et al., 2002). Overall, clear SPEM abnormalities are evident in gain (decrease), anticipatory saccades (increase), root mean square error (increase), and intrusive saccades (increase) in childhood-onset schizophrenia, supporting the hypothesis that dysfunctions are present in the brain networks involved in smooth pursuit (cortical eye fields, cerebellum, striatum, and/or brainstem). No deficits have been reported for catch-up saccades in childhood-onset schizophrenia. Preliminary results suggest problems may be present in VGS and MGS, however, the number of studies available precludes drawing conclusions.

4.6. Tourette's syndrome (TS)

4.6.1. Clinical manifestation of TS

TS is characterized by multiple motor tics (stereotype movements, like eye blinking, grimacing, and jaw, neck, shoulder or limb movements) and one or more vocal tics (like clicks, grunts, and coughs) that may appear simultaneously or at different periods during the illness (Albin & Mink, 2006). Tics occur frequently during a day, recurrently throughout a period of more than one year without a tic-free period of more than three months (APA, 1994). Tics can vary in type and frequency over time and are often preceded by urges that are very difficult to resist. TS usually begins during childhood or early adolescence and by definition before the age of 18 (APA, 1994). In most cases, the severity of the disorder reaches its peak during the second decade of life and diminishes by the end of adolescence (Swain, Scahill, Lombroso, King, & Leckman, 2007). The estimated prevalence rates of TS vary: once considered as a rare disorder (0.04–0.05%), recent studies using comprehensive ascertainment techniques indicate a larger incidence of TS (up to 1%) (Albin & Mink, 2006; APA, 1994). Males are 1.5–3 times more often affected than females. TS is strongly influenced by genetic factors, often causing multiple family members to portray tics (Albin & Mink, 2006; APA, 1994; Swain et al., 2007), although environmental factors such as stress are known to influence the expression of tics (Swain et al., 2007). TS is frequently associated with OCD and it may be difficult to distinguish complex tics from compulsions (Albin & Mink, 2006). ADHD is also a common comorbid disorder of TS (APA, 1994).

4.6.2. Neurological and cognitive problems in TS

The underlying complex neural circuitry associated with TS is described in detail in Swain et al. (2007). Considerable knowledge of the underlying neural circuitry has been gathered from studying procedural learning, habit formation, and internally and externally guided motor control in TS. The most important brain structures involved in TS are the basal ganglia, of which the striatal organization and function seems to be abnormal (Albin & Mink, 2006). The direct and indirect basal ganglia pathways provide a balance of excitation and inhibition that may be disrupted in TS. The frontal lobes may play an important role in the suppression of tics (Swain et al., 2007). It appears that TS patients have increased levels of dopaminergic innervation of the striatum and blockage of the dopaminergic receptors can suppress tics (Swain et al., 2007). From a cognitive point of view, inhibitory deficits are the most frequently studied. TS patients appear to have deficits in prepulse inhibition, manifested in the inability to filter out unnecessary sensory information and resulting in a diminished ability to manage sensory inputs to motor programs (Swain et al., 2007). However, several executive functions (inhibition, visual working memory, planning, cognitive flexibility, and verbal fluency) have found to be normal in TS children (Verté, Geurts, Roeyers, Oosterlaan, & Sergeant, 2005).

4.6.3. Eye movement studies in TS

Recently well-designed eye movement studies reviewed below investigated eye movements in pediatric TS patients. These studies have mainly focused upon targeting inhibition by measuring AS, which fits within the theoretical framework of TS being characterized as a disinhibitory disorder related to dysfunctions of the basal ganglia. VGS have been measured frequently and in some cases MGS and SPEM in TS.

4.6.4. Antisaccades

Three studies and one case report have measured AS in pediatric TS patients (Jackson, Mueller, Hambleton, & Hollis, 2007; Mostofsky et al., 2001b; Mueller, Jackson, Dhalla, Datsopoulos, & Hollis, 2006; Narita, Shawkat, Lask, Taylor, & Harris, 1997). In all three studies, TS patients did not commit more erroneous saccades (i.e., directional errors) than controls, suggesting that children with TS were not more prone than controls to making a directional error towards the target. Similar normal results were found in TS patients, when they had to maintain their fixation during the presentation of distracting stimuli (Bollen et al., 1988). Moreover, two studies reported that TS patients had a paradoxically greater level of cognitive control than controls, since TS patients had less difficulty than controls switching between prosaccades and AS within the same task (Jackson et al., 2007; Mueller et al., 2006). The case report by Narita and colleagues (1997), however, reported a complete failure of AS (10 errors in 10 trials) in their 13-year-old TS patient, but this may be explained by severe general oculomotor abnormalities in this patient. It appears that TS is not associated with inhibitory problems in antisaccade tasks.

4.6.5. Visually guided saccades

Five studies have reported VGS in children and adolescents with TS and all reported normal latency in TS children (Bollen et al., 1988; Jackson et al., 2007; Mostofsky et al., 2001b; Mueller et al., 2006; Nomura, Fukuda, Terao, Hikosaka, & Segawa, 2003). Peak velocity (Bollen et al., 1988; Nomura et al., 2003), variation in latency (Mostofsky et al., 2001b) and amplitude (Nomura et al., 2003) were normal when analyzed. These findings suggest VGS to be normal in pediatric TS patients.

4.6.6. Memory guided saccades

MGS have been reported in two studies (Mostofsky et al., 2001b; Nomura et al., 2003). Mostofsky and colleagues applied delays of 4.5–5 s and found a normal accuracy of saccades towards the memorized target in TS patients compared to controls. Nomura and colleagues did not report on deviations in the number of correct saccades towards the memorized target in TS children, although it appeared that TS patients had reduced amplitude of saccades towards the memorized target. In line with the findings of normal inhibitory performance on the antisaccade tasks, both studies found no greater degree of anticipatory saccades towards the memorized target in TS patients. These two studies suggest that visuo-spatial working memory and inhibitory control are intact in TS children, in a memory guided saccade paradigm, though replication of these findings is required.

4.6.7. Smooth pursuit eye movements

SPEM have not been studied extensively in TS children. Bollen et al. (1988) reported normal gain during pursuit in 28 TS children, although their performance was compared to norm values and not to gender- and age-matched control group, so no firm conclusions can be drawn. One case report is available (Narita et al., 1997) reporting on normal pursuit in a 13-year-old TS patient.

4.6.8. Conclusions for TS

Taken together, pediatric TS does not seem to be associated with dysfunctions in eye movements in the paradigms studied, suggesting the brain areas and cognitive processes involved in the various eye movement paradigms appear intact in TS.

4.7. Childhood-onset obsessive compulsive disorder (OCD)

4.7.1. Clinical manifestation of childhood-onset OCD

OCD is an impairing disorder characterized by recurrent obsessions and compulsions that are time consuming and/or cause marked distress or significant impairment. Early onset OCD might be associated with an increased genetic susceptibility for the disorder (Chabane et al., 2005). Obsessions are persistent ideas, thoughts, impulses, or images that cause marked anxiety or distress. Compulsions are repetitive behaviors, such as checking and ordering, or mental acts, like counting or repeating words silently (APA, 1994). The goal of the compulsions is to prevent or reduce the anxiety triggered by the obsessions. OCD usually begins gradually during adolescence or young adulthood, is (partially) determined by genetic factors and is equally common in both sexes (about 2.5%), although the symptoms appear somewhat earlier in males than in females (APA, 1994, Chabane et al., 2005; Mataix-Cols, Conceição do Rosario-Campos, & Leckman, 2005). The disorder is heterogeneous, even to the extent that two patients with the same diagnosis can display non-overlapping symptom patterns (Mataix-Cols et al., 2005), and co-occurs frequently with TS (APA, 1994). Presentation of OCD in children is generally similar to that in adults, although children may fail to realize the excessiveness or unreasonableness of their obsessions and compulsions and generally do not request help (APA, 1994).

4.7.2. Neurological and cognitive problems in childhood-onset OCD

Obsessive compulsive symptoms have mainly been linked to activation in the orbitofrontal cortex, with less consistent results for involvement of the anterior cingulate gyrus, striatum, thalamus (see glossary), lateral frontal and temporal cortices, amygdala, and insula (Mataix-Cols et al., 2005). In animal models and studies of patients with lesions in the orbitofrontal areas and its ventral striatal target fields, a selective disturbance in the ability to suppress responses to irrelevant stimuli has been found (O'Hearn, Rosenberg, Dick, & Sweeney, 1996). In line with this, an MRI study on pediatric patients with OCD showed these patients to have volumetric abnormalities in the ventral prefrontal cortical and striatal regions (Rosenberg & Keshavan, 1998). Interestingly, no abnormalities were found in the dorsolateral prefrontal cortex, a brain area strongly involved in working memory (Blumenfeld & Ranganath, 2006), which suggests that OCD is selectively associated with inhibition problems but not with working memory (i.e., forgetting whether an action has been completed and therefore checking it) or generalized executive dysfunctions, as has been previously suggested (Frampton, 2004).

4.7.3. Eye movement studies in childhood-onset OCD

Studies using eye movement paradigms are scarce in OCD and focus almost exclusively on adult patients. Only one study has reported on children (Rosenberg et al., 1997a). Four tasks were administered, all hypothesized as tapping into prefrontal cortical functions: an AS task, a MGS task, a VGS task with unpredictable targets, and a predictive saccade task. Children with OCD committed significantly more erroneous saccades compared to age-matched controls on the AS task as well as marginally significantly more anticipatory saccades on the MGS task, supporting the hypothesis that OCD is associated with inhibitory problems possibly related to (orbito)frontal dysfunctions. No other dysfunctions in oculomotor performance were found, suggesting working memory and general oculomotor control mechanisms are intact in pediatric OCD patients. Thus, preliminary results suggest that a disturbance in inhibition may underlie the repetitive behavior characterizing the illness. However, no firm conclusions can be drawn until these findings are replicated in other studies comprising larger samples.

4.8. Anxiety and depression

4.8.1. Clinical manifestation of anxiety and depression

Anxiety in children may manifest itself in several distinct syndromes, of which separation anxiety disorder, overanxious disorder, and specific phobias are the most common (Bernstein, Borchardt, & Perwien, 1996). Separation anxiety is characterized by excessive anxiety concerning separation from home or attachment figures, which causes clinically significant distress or impairment in social and/or academic functioning (APA, 1994). Children suffering from this disorder often refuse to go to school, have sleeping difficulties, and repeated nightmares. Overanxious disorder encompasses excessive anxiety and worries, which are out of proportion to the actual likelihood of the feared event and are difficult to control for the child. Children with this disorder may be overly conforming, perfectionist, and unsure of themselves and tend to redo tasks because of excessive dissatisfaction with less-than-perfect performance (APA, 1994). Specific phobias are marked and persistent fears cued by the presence or anticipation (see glossary) of a specific object or situation (such as animals, seeing blood, receiving an injection). These fears may be expressed by crying, tantrums, or clinging in children and they may be unaware of the unreasonableness of their fears. Anxiety disorders are quite common in childhood and adolescence, with estimates varying between 4% and 15%, and tend to be familial (APA, 1994; Bernstein et al., 1996). Slightly more girls than boys are affected. The most frequent comorbid disorder of an anxiety disorder is another anxiety disorder, followed by comorbid depression (APA, 1994).

It has been suggested that anxiety and depression are variants of a single mood disorder, but the more widely held view is that despite their considerable overlap, the two constructs are separate entities (Brady & Kendall, 1992). Both disorders are characterized by negative affectivity, but depression involves low positive affect, whereas anxiety is not related to positive affect (Brady & Kendall, 1992). Nevertheless, common genetic and environmental factors

relate to both disorders and symptoms of anxiety often precede symptoms of depression (Brady & Kendall, 1992). Depression is characterized by a marked depressed mood, though it may be expressed as irritableness in children, and/or the loss of interest in activities (APA, 1994). It is often accompanied by a reduced appetite, sleeping problems, feelings of worthlessness or guilt, and concentration problems. In children, somatic complaints, irritability, and social withdrawal are particularly common. Like anxiety, depression is more common in females than males and is familial. Typical age of onset is (early) adulthood, but it can occur in children (0.4–2.5%) and adolescents (0.4–8.3%) (APA, 1994; Birmaher et al., 1996).

4.8.2. Neurological and cognitive problems in anxiety and depression

Anxiety disorders have been associated with an increased activity of the amygdala, as well as an increased activity of the anterior cingulate and decreased activity of the medial prefrontal cortex (Anand & Shekhar, 2003). In depression, a decreased activation of the anterior cingulate cortex may lead to a dysregulation of certain limbic areas, such as the amygdala (Anand & Shekhar, 2003). The dorsolateral prefrontal cortex appears to be under activated in depression (Anand & Shekhar, 2003). Little research is available on cognitive problems in children and adolescents with anxiety and depression. It appears that anxious children have an overall lower level of cognitive functioning and academic achievement and problems have been found both on verbal, non-verbal, and executive (flexibility and inhibition) tasks (Kusché, Cook, & Greenberg, 1993). Comparable problems in executive functions were found in anxious-depressed boys (Emerson, Mollet, & Harrison, 2005).

4.8.3. Eye movement studies in anxiety and depression

To the best of our knowledge, only two studies are available documenting on eye movements in children with anxiety disorders and depression. Jazbec, McClure, Hardin, Pine, and Ernst (2005) administered an AS task under neutral, reward and punishment conditions to anxious and depressed adolescents. No group differences were found regarding accuracy and no group by condition interaction was found thus indicating that anxiety and depression in adolescents were not associated with an increased number of erroneous prosaccades. Latency and peak velocity differed between the groups and group differences were modulated by condition. Normal adolescents showed an improvement in latency and peak velocity, when rewarded or punished for their performance. This was not found in anxious and depressed adolescents. Indeed, the opposite effect was found in anxious adolescents for latency (i.e. an increase), particularly when punished. These findings suggest that performance on the AS task can be influenced by motivation in normally developing adolescents, but a motivation manipulation has no effect or a deteriorating effect in anxious and depressed adolescents. The AS task was used two years later by Hardin, Schroth, Pine, and Ernst (2007), who manipulated the frequency of reward and punishment, although this did not appear to influence results. In contrast to the previous study, group differences were present for accuracy: depressed adolescents made more errors than healthy adolescents; anxious adolescents performed intermediately between both groups. These group differences were not influenced by condition. In agreement with the previous study, groups were differently affected by condition with respect to latency. Again, the latency of healthy adolescents improved more strongly when rewarded or punished than the latency of anxious or depressed adolescents. Taken together the results of both studies suggest that there is preliminary evidence for deviations in the performance of anxious and depressed adolescents on an AS task. Mainly the insensitivity of the latency of saccades for reward or punishment appears to be a sensitive indicator of pathology relating to negative affectivity, possibly reflecting deficits in the speed of inhibiting a response (Jazbec et al., 2005).

5. Overlap and specificity of findings across eye movement paradigms and across psychiatric disorders

Table 2 provides an overall summary of findings of (ab)normality of eye movements in psychiatric disorders. AS have been investigated in the majority of disorders reviewed here. An elevated number of directional errors appears present in ADHD, ASD, RD, OCD, and anxiety and depression. No AS have been investigated in schizophrenia or ODD/CD. Only TS does not appear to be associated with an increased frequency of directional errors. These findings suggest that AS are sensitive in detecting abnormal psychological functioning in general, but cannot be used for identifying specific types of pathology. Rather, most psychiatric disorders in childhood and adolescence appear related to dysfunctional inhibitory control, as indicated by the inability to suppress reflexive saccades towards the target (prosaccades), suggesting impaired frontal functioning is common in most childhood psychiatric disorders.

VGS have also been frequently investigated, but appear less sensitive to general psychiatric deviancy than AS. ASD, schizophrenia, TD, and OCD do not appear to be associated with dysfunctional VGS. The majority of studies on VGS in RD have documented normal performance, although a general elevated number of saccades appears related to RD. Only in ADHD have problems in VGS been consistently reported: children with ADHD have overall more variable and possibly somewhat slower saccade latencies than control children. This suggests VGS to be an interesting research tool to explore the uniqueness of underlying neurological pathology of ADHD and also suggests that most childhood disorders are not characterized by deficits in the basic neurological substrate of eye movements, such as the cerebellum and frontal eye fields.

MGS appear deficient in various psychiatric disorders. A decreased accuracy of MGS has been found in ADHD, ASD, and schizophrenia, suggesting these disorders may share a weakness in visuo-spatial memory abilities. An elevated number of anticipatory saccades during MGS has been documented in ADHD, ASD, schizophrenia, and OCD, making this measure sensitive to inhibitory problems occurring in most psychiatric disorders, but insensitive in detecting specific pathology. Like the findings on AS, the findings on MGS suggest impaired frontal functioning is common in childhood psychiatric disorders.

Although strongly related to schizophrenia, abnormalities in SPEM are also found in other disorders. Frequently found abnormalities in schizophrenia, such as reduced gain, increased root mean square error, and increased saccade frequency, have been found in relation to ADHD, ASD, and RD. Only TS does not seem to be related to problems in SPEM. Since SPEM involve a complex interaction between various cortical and subcortical brain areas, it is difficult to interpret these findings with respect to specificity and overlap of cognitive and neurological dysfunctions across the various disorders.

Overall, the AS, MGS, and SPEM paradigms were able to discriminate between psychologically normal and abnormal subjects, making them useful tools in experimental and clinical practice. However, they lack specificity, making it difficult to distinguish between eye movement dysfunctions amongst the various disorders. The reverse appears to be true for VGS, which appear generally normal in most disorders, but deficient in ADHD.

6. Developmental aspects of eye movement abnormalities in childhood and adolescence psychiatric disorders

All eye movement tasks discussed in this review are heavily influenced by developmental changes in brain maturation (Luna and colleagues, this issue). Mainly AS and MGS, and to a lesser ex-

Table 2
General conclusions regarding findings of (ab)normal eye movements across various paradigms in psychiatric disorders in childhood and adolescence

Disorders	Eye movement paradigms				
	AS	VGS	MGS	SPEM	Conclusion
ADHD	- - ↑ errors	- - ↑ SD latency, possibly ↑ latency	- ↑ anticipatory saccades, ↓ accuracy	- ↑ root mean square error, ↑ saccades	ADHD characterized by inhibitory problems across paradigms, possibly reflecting dysfunctions in frontal areas like DLPFC. Basic eye movement control may also be impaired, reflecting frontal eye field and cerebellar deviations
ODD/CD	?	?	?	?	ODD and CD may be associated with similar eye movement findings as ADHD given the high degree of comorbidity between ADHD and ODD/CD
ASD	- ↑ errors	+	- ↑ anticipatory saccades, ↓ accuracy, ↑ latency	- ↑ saccades, ↓ gain	ASD associated with inhibitory problems, possibly reflecting frontal cortex dysfunctions. Saccadic control appears normal, but smooth pursuit not, which may suggest various non-cortical areas may be involved in ASD
RD	- ↑ errors, ↑ misses	- ↑ saccades	?	- - ↑ saccades, ↓ smoothness	RD appears related to an increased number of saccades across paradigms studied and with decreased smooth persuit, possibly reflecting problems in the integration of the saccadic and pursuit networks
Schizophrenia	?	+	- ↑ anticipatory saccades, ↓ accuracy	- - - ↑ root mean square error, ↓ gain, ↑ saccades	Schizophrenia characterized by abnormal smooth pursuit, reflecting abnormal functioning of cortical eye fields, cerebellum, striatum, and brainstem. Frontal functioning may also be abnormal.
TS	+	+	+	+	TS appears not associated with eye movement abnormalities, inhi bitory or working memory problems, suggesting most cortical and non-cortical areas involved in eye movements function normally
OCD	- ↑ errors	+	- ↑ anticipatory saccades	?	OCD may be associated with inhibitory deficits related to problems in the (orbito)frontal areas. Basic saccadic control appears normal
AD/ depression	- ↑ errors, ↑ latency	?	?	?	AD and depression associated with insensitivity of saccade latency for reward or punishment, possibly reflecting abnormalities in inhibition related to frontal areas
Conclusion	AS sensitive in detecting general abnormal functioning, but not in discriminating between disorders	VGS not sensitive in detecting general abnormal functioning, but appears discriminative for ADHD	MGS sensitive in detecting general abnormal functioning, but not in discriminating between disorders	SPEM sensitive in detecting general abnormal functioning, but not in discrim	

Note. +, (probably) normal; -, possibly abnormal; - -, probably abnormal; - - -, abnormal; ?, not investigated.
AS, antisaccades; VGS, visually guided saccades; MGS, memory guided saccades; SPEM, smooth pursuit eye movements; ADHD, attention deficit hyperactivity disorder; ASD, autism spectrum disorders; RD, reading disorder; TS, Tourette's syndrome; OCD, obsessive-compulsive disorder; AD,Anxiety disorder; ODD, oppositional deviant disorder; CD, conduct disorder.

tent VGS and SPEM, continue to develop through adolescence (Klein, Foerster, Hartnegg, & Fischer, 2005). It may thus be feasible that the eye movement deficits associated with childhood and adolescent psychiatric disorders described in this review, correct themselves by the end of adolescence. A brief review of adult eye movement literature may indicate whether characteristic eye movement difficulties present in children and adolescents with a disorder are also present (or not) in adults with the disorder. Importantly, however, psychiatric disorders occurring during childhood and adolescence may not necessarily be comparable to psychiatric disorders occurring during adulthood. Other genetic and environmental factors may influence psychiatric functioning during childhood/adolescence and adulthood. Certain disorders typically become apparent during childhood and remit during late adolescence/adulthood (ADHD, ASD, and TS), whereas others typically become apparent in late adolescence/adulthood (schizophrenia, OCD, and depression). Adults having a disorder that usually improves during late adolescence/adulthood and children having a disorder that usually does not become apparent until late adolescence/adulthood, may be atypical groups. Thus, even though reviewing adult eye movement literature may shed light on the developmental continuity of eye movement deficits reported in children and adolescents with psychiatric disorders, comparing results is somewhat hazardous.

6.1. Attention-deficit/hyperactivity disorder

As described earlier and in Table 2, ADHD in childhood and adolescence is characterized by an increase of AS errors, an increase in VGS latency, and also with more anticipatory saccades and decreased accuracy on MGS, and with some SPEM abnormalities. One study assessed eye movements in both children and adults with ADHD and found similar deficits in VGS and AS in both groups (Munoz et al., 2003), which was in line with findings from another study on abnormalities during VGS and AS in adults with ADHD (Feifel, Farber, Clementz, Perry, & Anllo-Vento, 2004). In addition,

a further study reported an increase in the percentage of premature saccades during MGS in young adults with ADHD (Ross, Harris, Olincy, & Radant, 2000), which has also been reported in children and adolescents with ADHD. However, accuracy of MGS appeared normal in ADHD adults in this study and no SPEM impairment were present in a group of adults with ADHD (Ross, Olincy, Harris, Sullivan, & Radant, 2000). Overall, eye movement deficits on paradigms most strongly associated with ADHD in childhood and adolescence (AS and VGS) appear also present in adults with ADHD, with some mixed/negative results found for eye movement deficits on paradigms less robustly associated with ADHD (MGS and SPEM).

6.2. Oppositional deviant disorder and conduct disorder

We were not able to locate any studies documenting eye movements in adults with conduct disorder or criminal behavior, as was the case in children with ODD or CD.

6.3. Autism spectrum disorder

With respect to ASD, several studies have documented on AS, VGS, en MGS in autistic adults. Mixed findings have been reported for VGS, although most studies reported no abnormalities (Minshew, Luna, & Sweeney, 1999; Takarae, Minshew, Luna, & Sweeney, 2007), but some found some evidence for abnormal accuracy during VGS (Luna et al., 2007). However, a functional magnetic resonance imaging (fMRI (see glossary)) study and an ERP (event-related potential) study found atypical brain activation (Takarae et al., 2007) and ERP potentials (Kawakubo et al., 2007) during VGS in adults with ASD even when behavioral data for VGS was normal (Takarae et al., 2007), suggesting the underlying neural substrate for VGS may be abnormal in ASD in adults. Normal behavioral data on VGS are in line with findings in children and adolescents with ASD. An increased number of errors during AS and MGS in adults with ASD has been documented in two studies (Luna et al., 2007; Minshew et al., 1999), and is in line with findings for children and adolescents with ASD. Furthermore, abnormalities in SPEM have been found in adults with ASD, as well as children and adolescents (Takarae et al., 2004). It thus seems that most findings on abnormalities in eye movements documented in children and adolescents with ASD appear also to be present in adults with ASD.

6.4. Reading disorder

In adults with RD, increases in frequency of saccades during different VGS paradigms have been documented (Biscaldi et al., 1998; Fischer, Biscaldi, & Otto, 1993), with additional evidence for abnormal saccade latencies (Biscaldi et al., 1998; Fischer et al., 1993; Ram-Tsur, Faust, Caspi, Gordon, & Zivotofsky, 2006). The findings for an increase in saccades during VGS are in line with the findings reported in children and adolescents, although abnormal latencies have not been reported consistently in childhood and adolescent literature.

6.5. Schizophrenia

The number of studies on eye movements in adults with schizophrenia is far greater than the number of studies on eye movements in children and adolescents with schizophrenia, since schizophrenia in the former group is more common than schizophrenia in the latter group. Abnormalities in SPEM performance (reduced gain and an increased number of saccades) and reduced brain activation during SPEM have been consistently found in adults with schizophrenia (Boudet et al., 2005; Keedy, Ebens, Keshavan, & Sweeney, 2006; Smyrnis et al., 2007; Zanelli et al., 2005), and is in line with findings of children and adolescents with the disorder (Ross et al., 2002). In addition, abnormal AS, VGS, and MGS performance and abnormal brain activation during these types of saccades have also been found in adults with schizophrenia (Boudet et al., 2005; Keedy et al., 2006; Zanelli et al., 2005), suggesting abnormal eye movements in adult schizophrenia are generalized across paradigms and even correlated across paradigms (Zanelli et al., 2005). Findings for AS, VGS, and MGS performance in children and adolescents with schizophrenia are scarce, but suggest some abnormalities may be present in MGS. No abnormalities have been reported for VGS in children and adolescents with schizophrenia and no studies have investigated AS in this group. All in all, SPEM abnormalities are evident in both children, adolescents and adults with schizophrenia, but findings are not yet conclusive regarding eye movement abnormalities in other paradigms.

6.6. Tourette's syndrome

Pediatric TS does not seem to be associated with dysfunctions in eye movements in the paradigms studied, suggesting the brain areas and cognitive processes involved in the various eye movement paradigms appear intact in TS. This is remarkable, given that studies on adult TS generally report on longer latencies, more anticipatory and intrusive saccades, and abnormal amplitudes in adult TS patients (Dursun, Burke, & Reveley, 2000; Farber, Swerdlow, & Clementz, 1999; LeVasseur, Flanagan, Riopelle, & Munoz, 2001; Munoz, LeVasseur, & Flanagan, 2002; Straube, Mennicken, Riedel, Eggert, & Müller, 1997). The abnormal eye movement findings in adults with TS might be explained by the fact that individuals with tics persisting into adulthood may be an atypical group (Albin & Mink, 2006), since the symptoms of TS diminish substantially during adolescence and only 20% or fewer of children with TS continue to experience a moderate level of impairment by the age of 20 (Swain et al., 2007).

6.7. Obsessive compulsive disorder

In contrast to childhood and adolescents literature, studies on eye movement in OCD in adults are numerous. As performance appears normal in adults with OCD, except for a somewhat longer latency (Maruff, Purcell, Tyler, Pantelis, & Currie, 1999; Rosenberg, Dick, O'Hearn, & Sweeney, 1997b; Van der Wee et al., 2006). This contrasts with the one study in childhood-onset OCD, where an increase of the number of errors was found (Rosenberg et al., 1997a), although an increase in errors has also been reported in adults with OCD when the peripheral visual targets were presented close to central fixation (Rosenberg, Dick, O'Hearn, & Sweeney, 1997b). Some abnormalities have been reported in VGS in adults with OCD, such as reduced peak velocity (Rosenberg, Dick, O'Hearn, & Sweeney, 1997b), decreased accuracy (Gambini, Abbruzzese, & Scarone, 1993) and an increased number of predictive and corrective saccades (Spengler et al., 2006). This contrasts with normal VGS in the one OCD child study (Rosenberg et al., 1997a). However, normal VGS has also been reported in adults with OCD (Maruff et al., 1999; Nickoloff, Radant, Reichler, & Hommer, 1991; Van der Wee et al., 2006), as was normal latency and accuracy (Gambini et al., 1993; Rosenberg, Dick, O'Hearn, & Sweeney, 1997b). It thus remains inconclusive as to whether or not OCD in childhood and/or adulthood is associated with impairments in VGS. MGS, though not heavily investigated in adults or children/adolescents with OCD, appeared normal in one study on adults (Rosenberg, Dick, O'Hearn, & Sweeney, 1997b), but characterized by an increased number of anticipatory saccades in children (Rosenberg et al.,

1997a). Clearly, more studies are needed to draw firm conclusions for MGS in OCD. SPEM has been examined only in adults with OCD, but not children or adolescents, and findings are inconsistent. Normal SPEM has been reported (Nickoloff et al., 1991; Spengler et al., 2006), slightly abnormal SPEM has been reported (normal pursuit except for smaller catch-up saccades) (Clementz, Farber, Lam, & Swerdlow, 1996), as well as abnormal SPEM (reduced gain and/or increased number of anticipatory saccades or square wave jerk intrusions) (Gambini et al., 1993; Pallanti et al., 1996; Sweeney, Palumbo, Halper, & Shear, 1992). These inconsistent findings may be related to differences in velocity of the targets in the different studies, because it has been suggested that patients with OCD may have only a modest SPEM deficit that is elicited only while following faster velocity targets (Clementz et al., 1996). In conclusion, findings for eye movement impairments in adult OCD are evident, partly overlap with findings of the one study in children and adolescents with OCD, and suggest more studies are needed that examine eye movements in childhood-onset OCD.

6.8. Anxiety and depression

Studies reporting on eye movements in relation to anxiety and depression in adults are not numerous, but some consistent findings do emerge. Errors on AS have been found to be related to anxiety, though not related to depression (Shafiq-Antonacci et al., 1999; Smyrnis et al., 2003). Depression was found to be related to poorer saccade latency (Shafiq-Antonacci et al., 1999), though not consistently (Smyrnis et al., 2003). An increase in AS errors and latency has also been documented in anxious and depressed children and adolescents. VGS and MGS have hardly been studied in adults with anxiety and depression, as was the case in the childhood and adolescent literature. One study documented on normal peak velocity and accuracy on VGS, but slower latency in adults with depression (Mahlberg, Steinacher, Mackert, & Flechtner, 2001). SPEM is a more frequently studied task, and pursuit gain has been found abnormal in several studies in relation to depression in adults (Flechtner, Steinacher, & Mackert, 1997; Kathmann, Hochrein, Uwer, & Bondy, 2003; Malaspina et al., 1994), though not in all studies (Smyrnis et al., 2007). Because only AS have been studied in children and adolescents with anxiety and depression, comparison between eye movement results in children/adolescents and adults is limited, but results for AS appear to agree between children/adolescents and adults with anxiety and depression.

7. Recommendations for future research

It has become clear from reviewing the literature on eye movement studies in childhood and adolescence psychiatric disorders that for each disorder, a line of research has evolved relatively independently of the lines of research for the other disorders. This is largely driven by hypotheses regarding the nature of the underlying neurological and cognitive problems of each disorder. For example, the vast majority of studies in RD has focused on VGS, since it has been hypothesized that reading problems might be partly related to saccadic dysfunctioning (Pavlidis, 1980, 1981, 1985). Since SPEM abnormalities have been consistently found in adults with schizophrenia, the most obvious and dominant area of focus in childhood-onset schizophrenia has been SPEM. This is, of course, a sensible approach when comprehensively unravelling the eye movement impairments associated with that disorder. Different illnesses raise different questions and, thus, different methodologies can be of value. However, increasing knowledge is gained on the shared genetic, neurobiological and neuropsychological impairments of psychiatric illnesses. Eye movement research may contribute to this line of research by standardizing methodologies and/or by including several groups of psychiatric patients and comparing those with controls in a study, instead of one group of patients comparing to controls. We do not advocate that studying eye movements in one psychiatric disorder is less sensible that studying eye movements in several psychiatric illnesses in order to compare eye movement deficits across disorders, but rather want to emphasize that the latter approach (examining eye movements in several groups of psychiatric illnesses instead of just one) has not received enough attention in the eye movements literature, but should be given more consideration in light of the increasing knowledge on shared genetic, neurobiological, and neuropsychological factors across disorders. Therefore, our first recommendation for future research is to use similar eye movement paradigms in various disorders in order to examine the specificity and overlap of eye movement deficits for these disorders. For example, ADHD frequently co-occurs with ODD/CD and RD. It would facilitate the understanding of the nature of the individual disorders and their overlap if eye movements would be measured in children suffering from one or a combination of these disorders in a so-called double dissociation design (Cantwell, 1995; De Jong, Oosterlaan, & Sergeant, 2006). The same is true for OCD and TS. In addition to this, it is likely that the available studies have included patients with multiple disorders, while reporting the effect of one disorder on eye movements. That is, the vast majority of children suffering from a psychiatric disorder suffer from at least one other psychiatric disorder (Caron & Rutter, 1991). Failure to take these comorbid conditions into account when analyzing eye movement performance, as has been done in almost all eye movement studies, makes it difficult to determine the communality and specificity of findings for these childhood disorders.

Some disorders have received considerable attention from eye movement researchers, whereas other disorders have not. We were unable to locate any studies investigating eye movements in children with behavioral problems (ODD/CD), hardly any studies in children with anxiety/depression and OCD, and only a few studies in children with TS. Clearly, more work is needed in investigating eye movements in these pediatric psychiatric groups. It may prove valuable to expand the focus of research in RD and schizophrenia outside respectively the areas of SPEM and VGS, since the few studies that have done that, have found deficits.

A final recommendation for future research relates to the developmental aspect of disorders in childhood and adolescence. Many children suffering from a psychiatric disorder become psychiatrically impaired adults. However, this is not a 100% continuity, since some children do not suffer from psychiatric problems, when grown up or suffer from another disorder than that diagnosed in childhood (Hollis, 2000; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993; Zoccolillo, Pickles, Quinton, & Rutter, 1992). Thus, there is both continuity and discontinuity in psychiatric development. A recommendation for future research is to longitudinally study eye movement parameters in children with psychiatric disorders growing up. Like for certain neurobiological and cognitive measures, some eye movement parameters measured early in life may prove to be related to psychiatric functioning later in life. This eye movement parameter may than be used to form a more homogeneous subgroup of patients having the eye movement deficit in common, for whom the longitudinal outcome of their disease may be more similar than for the whole group of patients with that disorder. In addition, the eye movement deficit may also shed light on the involvement of brain areas/systems in causing the disorder at an early stage for this subgroup of patients.

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