Executive Functioning Theory and Autism

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Hilde M. Geurts, Marieke de Vries, and Sanne F.W.M. van den Bergh

Autism Spectrum Disorders

The triad of characteristics that defines the autistic disorder includes the following: social and communication impairments, and restricted, stereotypical patterns of behavior and interests (American Psychiatric Association [APA], 1994, 2000, for all symptoms see Table 8.1). There are different classic autism-like conditions, and these other pervasive developmental disorders (PDD), such as Asperger syndrome and PDD not otherwise specified (PDDNOS), are part of the broader phenotype of autism. In the current classification system, DSM-IV (APA, 1994, 2000), also Rett syndrome and the Disintegration disorder are considered autism-like conditions. However, in the current chapter we will focus solely on classic autism, Asperger syndrome, and PDDNOS. The combination of these three disorders is referred to as an autism spectrum disorder (ASD), which is the term we will use throughout this chapter.

H.M. Geurts (✉) • S.F.W.M. van den Bergh
Department of Psychology, Dutch Autism & ADHD Research Center (d'Arc), University of Amsterdam, Amsterdam, The Netherlands

Dr Leo Kannerhuis, Amsterdam/Doorwerth, The Netherlands
e-mail: h.m.geurts@uva.nl

M. de Vries
Department of Psychology, Dutch Autism & ADHD Research Center (d'Arc), University of Amsterdam, Amsterdam, The Netherlands

ASD is a heterogeneous, lifelong neurobiological disorder, with an enormous impact on all developmental domains of which the prevalence is estimated between 60 and 100 cases per 10,000 (Baird et al., 2006; Brugha et al., 2011; Gezondheidsraad, 2009). ASD can be diagnosed as early as 18 months of age and leads to a wide array of affective, behavioral, and cognitive problems that are waxing and waning across the lifespan (Rapin & Tuchman, 2008; Volkmar, Lord, Bailey, Schultz, & Klin, 2004). Approximately 70 % of the individuals with an ASD diagnosis have an IQ below 80, indicating an intellectual disability (Fombonne, 2005; Matson & Boisjoli, 2008). ASD also commonly co-occurs with other disorders such as attention-deficit hyperactivity disorder (ADHD), anxiety, and mood disorders (Hofvander et al., 2009; Leyfer et al., 2006; Matson & Nebel-Schwalm, 2007). In children with ASD 71 % has at least one comorbid disorder, and 41 % at least two (Simonoff et al., 2008). This high prevalence of comorbid disorders is persistent into adulthood (Geurts & Jansen, 2012; Hofvander et al., 2009) and has probably a large impact on the observed cognitive problems of individuals with ASD.

An influential cognitive theory of ASD purports that the symptoms observed in individuals with ASD arise from executive function (EF) deficits (Damasio & Maurer, 1978; Hill, 2004; Maurer & Damasio, 1982; Pennington & Ozonoff, 1996; Russell, 1997; Russo et al., 2007). As described in the previous chapters, executive functions (EFs) encompass the ability to suppress responses (inhibition), to keep and

S. Goldstein and J.A. Naglieri (eds.), *Handbook of Executive Functioning*,
DOI 10.1007/978-1-4614-8106-5_8, © Springer Science+Business Media New York 2014

Table 8.1 ASD symptoms

	DSM-IV-TR criteria autistic disorder
A	1. Qualitative impairment in social interaction
	(a) Marked impairments in the use of multiple nonverbal behaviors such as eye-to-eye gaze, facial expression, body posture, and gestures to regulate social interaction
	(b) Failure to develop peer relationships appropriate to developmental level
	(c) A lack of spontaneous seeking to share enjoyment, interests, or achievements with other people (e.g., by a lack of showing, bringing, or pointing out objects of interest to other people)
	(d) Lack of social or emotional reciprocity (e.g., not actively participating in simple social play or games, preferring solitary activities, or involving others in activities only as tools or "mechanical" aids)
	2. Qualitative impairments in communication
	(a) Delay in, or total lack of, the development of spoken language (not accompanied by an attempt to compensate through alternative modes of communication such as gesture or mime)
	(b) In individuals with adequate speech, marked impairment in the ability to initiate or sustain a conversation with others
	(c) Stereotyped and repetitive use of language or idiosyncratic language
	(d) Lack of varied, spontaneous make-believe play or social imitative play appropriate to developmental level
	3. Restricted repetitive and stereotyped patterns of behavior, interests and activities
	(a) Encompassing preoccupation with one or more stereotyped and restricted patterns of interest that is abnormal either in intensity or focus
	(b) Apparently inflexible adherence to specific, nonfunctional routines or rituals
	(c) Stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping or twisting, or complex whole body movements)
	(d) Persistent preoccupation with parts of objects
B	Delays or abnormal functioning in at least one of the following areas, with onset prior to age 3 years: in social interaction, language as used in social communication, symbolic or imaginative play
C	The disturbance is not better accounted for by Rett's disorder or childhood disintegrative disorder

Note: In the DSM-IV-TR, one needs a total of six (or more) items from (1), (2), and (3), with at least two from (1), and one each from (2) and (3) one to meet criteria for the autistic disorders, for Asperger's disorder domain (2) is not part of the criteria. Georgiades and colleagues (2007) showed that the three categorical DSM-IV ASD domains, social relationships, communication, and restrictive repetitive and stereotyped behavior are very heterogeneous. For example, communication includes behavior that regulates social interaction, but also includes flexible use of language. Also repetitive behavior consists of both repetitive stereotyped movements and inflexible behavior. They suggested three new factors (1) social communication; (2) inflexible language and behavior; and (3) repetitive sensory and motor behavior. Especially the last two might be related to inflexibility, respectively to cognitive and to motor inflexibility. In the proposal for the DSM-5 two domains are included, Persistent deficits in social communication and social interaction across contexts, not accounted for by general developmental delays and restricted, repetitive patterns of behavior, interests, or activities including Hyper- or hyporeactivity to sensory input or unusual interest in sensory aspects of environment

manipulate information online (working memory), to change strategies (cognitive flexibility), and to plan ahead (planning). Individuals with ASD seem to encounter deficits in each of these domains. Some even argued (Damasio & Maurer, 1978) that EF deficits might be at the core of ASD as individuals with ASD have problems with exerting effortful control when they need to deal with novel, complex, or ambiguous situations in everyday life. Moreover, it seems that these deficits in ASD are associated with structural and functional abnormalities in the underlying frontostriatal network (Amaral, Schumann, & Nordahl, 2008; Gilbert, Bird, Brindley, Frith, & Burgess, 2008; Kana, Keller, Minshew, & Just, 2007; Luna et al., 2002; Schmitz et al., 2006; Shafritz, Dichter, Baranek, & Belger, 2008).

In this chapter we will first address the origin of this theory, followed by a short overview of the literature focusing on the (dis)functioning of the frontostriatal network in ASD. Hereafter, we will describe how several ASD symptoms might arise from an EF deficit for the following executive functioning domains: inhibition, working memory, cognitive flexibility, and planning. For each of these four EF domains, a short overview of the most recent findings in ASD will be provided.

The Analogy with Patients with Frontal Lobe Damage

The first who postulated an executive dysfunction account of ASD were Damasio and Maurer (1978). In their influential paper they described

how individuals with frontal lobe damage show specific behavior which is also typical for people with ASD (Eslinger & Damasio, 1985). The observation that patients with frontal lobe lesions have social difficulties led to the hypothesis that ASD might be a frontal lobe disorder. This idea has inspired various research groups across the world to determine whether or not individuals with ASD indeed encounter EF deficits, whether individuals with ASD show deficits in all or just in some EF domains, and whether there is evidence for a disruption of the frontal network.

The Involvement of the Frontostriatal Network in ASD

Brain imaging studies of ASD demonstrate abnormalities in both structure and function of several brain regions including the prefrontal cortex (Agam, Huang, & Sekuler, 2010; Amaral et al., 2008; Mcalonan et al., 2005; Stanfield et al., 2008). Other studies have suggested that rather than deficits in localized activity, ASD may be better conceptualized as dysfunctions in activity of distributed brain network, or deficient synchronization within those networks (Courchesne & Pierce, 2005). According to Courchesne and Pierce (2005) the "autistic brain" is characterized by local over-connectivity and long-range under-connectivity of the *frontal* cortex. Just, Cherkassky, Keller, and Minshew (2004) postulated that ASD arises from reduced synchronization between *frontal and posterior regions* of the cortex. This reduced synchronization has been observed during performance on a broad range EF tasks (Agam, Huang, et al., 2010; Just et al., 2004; Kana, Keller, Cherkassky, Minshew, & Just, 2006; Kleinhans et al., 2008; Mason, Williams, Kana, Minshew, & Just, 2008; Solomon et al., 2009) but, for example, also during social processing (Kana, Keller, Cherkassky, Minshew, & Just, 2009; Welchew et al., 2005). Moreover, this connectivity has been related to the presence of repetitive behavior in individuals with ASD, which is one of the key aspects of the ASD diagnosis (e.g., Agam, Huang, et al., 2010; Langen, Durston, Kas, Van Engeland, & Staal, 2011). Imaging studies revealed that, while performing EF tasks, people with ASD show activation abnormalities in the frontostriatal circuitry (Gilbert et al., 2008; Kana et al., 2007; Luna et al., 2002; Schmitz et al., 2006; Shafritz et al., 2008). They often recruit *more* brain areas when performing these tasks as compared to healthy people, but both over- and under-activation have been observed in individuals with ASD as compared to controls (Gilbert et al., 2008; Kana et al., 2007; Luna et al., 2002; Schmitz et al., 2006; Shafritz et al., 2008).

With respect to the different EF domains, several ASD studies focused on the frontostriatal and frontoparietal network. For example, imaging studies focusing on inhibition reported more frontal and less parietal activation (Kana et al., 2007; Schmitz et al., 2006). In working memory studies (e.g., Belmonte & Yurgelun Todd, 2003; Gomarus, Wijers, Minderaa, & Althaus, 2009; Koshino et al., 2005; Luna et al., 2002), less task-related activation has been observed, for example, in the dorsolateral prefrontal cortex and the posterior cingulate (Luna et al., 2002), the left inferior frontal area (Koshino et al., 2008), and in the middle frontal gyrus and superior parietal lobe activation (Belmonte & Yurgelun Todd, 2003). In line with these findings, anterior-posterior coherence in brain connectivity is higher in children with ASD, which is associated with worse working memory performance (Chan et al., 2011). Moreover, reduced connectivity in the prefrontal regions is not just related to working memory but also to ASD severity (Poustka et al., 2012). In a meta-analysis (Di Martino et al., 2009) it was shown that when performing the so-called nonsocial tasks (these were mainly EF tasks), the pre-supplementary motor area and the dorsal anterior cingulate cortex (ACC) were hypo-activated in individuals with ASD, while in social tasks (including facial processing tasks and theory of mind [ToM] tasks), the perilingual wall of the ACC and right anterior insula were hypo-activated. Hence, currently ASD is seen as a brain connectivity disorder (see Schipul, Williams, Keller, Minshew, & Just, 2012; Vissers, Cohen, & Geurts, 2012; Wass, 2011), and the observed EF deficits have been related to the

increased connectivity within the prefrontal cortex and the decreased connectivity of the frontal cortex with more posterior regions of the brain.

Do People with ASD Have Specific Executive Functioning Deficits?

Even though the EF dysfunction account does have an intuitive appeal to explain the observed behavior in individuals with ASD, there are some difficulties with this idea. A complication for an executive dysfunction account of ASD is that various other disorders (e.g., ADHD, see Chap. 10) are also associated with EF deficits. Hence, the specificity of the EF hypothesis has been widely disputed (Pennington & Ozonoff, 1996; Sergeant, Geurts, & Oosterlaan, 2002) as, for example, working memory deficits seem to be present in a wide range of disorders (Willcutt, Sonuga-Barke, Nigg, & Sergeant, 2008). Even though executive dysfunctions are not specific for ASD, this does not imply that it is not worthwhile studying EF in relation to ASD as the pattern of EF deficits might gain insight in the day-to-day deficits people with ASD encounter.

Another complication is that recent reviews and meta-analyses suggest that findings regarding EF in ASD are rather inconsistent across studies (Geurts, Corbett, & Solomon, 2009; Hill, 2004; Russo et al., 2007). For example, some argue that there is a clear deficit in cognitive flexibility (Hill, 2004; Russo et al., 2007) while this is doubted by others (Geurts, Corbett, et al., 2009). To explain these different findings, various arguments have been proposed. First, it has been noted that the participants included in the ASD groups may differ in their clinical diagnosis (i.e., autism, Asperger syndrome, PDDNOS). Even though these subgroups seem to have similar patterns of EF deficits (Verté, Geurts, Roeyers, Oosterlaan, & Sergeant, 2006b), this is often used as an explanation for the different pattern of findings. Second, there are differences among studies in the IQ range of the included participants and in how IQ differences between groups are handled. However, even studies in which the IQs of the participants were similar have shown inconsistent results (e.g., Geurts, Verté, Oosterlaan, Roeyers, & Sergeant, 2004). Third, some studies focus on children (e.g., Corbett, Constantine, Hendren, Rocke, & Ozonoff, 2009; Goldberg et al., 2005; Happe, Ronald, & Plomin, 2006; Luna, Doll, Hegedus, Minshew, & Sweeney, 2007), others on adults (e.g., Bramham et al., 2009; Lopez, Lincoln, Ozonoff, & Lai, 2005; Luna et al., 2007) or even the elderly (Geurts & Vissers, 2012), and some even include individuals from a very broad age range (Ambery, Russell, Perry, Morris, & Murphy, 2006; Hill & Bird, 2006). Hence, the inconsistencies might be due to the deviant developmental trajectory of EFs in people with ASD (see Happe et al., 2006; Luna et al., 2007). Fourth, both the types of task used to measure the different EF domains and the reported dependent measures of these tasks vary widely (Sergeant et al., 2002). There is some truth in each of these four arguments, but the inconsistent findings also reflect the genuine heterogeneity in the cognitive deficits of the ASD population. A recent study by Pellicano (2010b) seems to support this argument; considerable individual differences were found in EF abilities in very young children with ASD (see also Geurts, Sinzig, Booth, & Happé, submitted [children]; Johnston, Madden, Bramham, & Russell, 2011 [adults]).

An important discussion in the EF literature regarding ASD is how the EF theory relates to other dominant ASD theories. Two other dominant cognitive theories about ASD are the central coherence theory (Frith, 1989; Frith & Happé, 1994; Happé, 1999) and the ToM (e.g., Baron Cohen, 2001; Frith, Morton, & Leslie, 1991). Central coherence refers to an information processing style in which one processes information in its specific context, and a weak central coherence would result in piecemeal processing which is often observed in individuals with ASD (Happé, 1999; Pellicano, 2007, 2010b). The relationship between EF and central coherence is hardly studied as the assumption is that these theories explain different aspects of the autism spectrum (see also Happe et al., 2006). ToM refers to the ability to attribute mental states to oneself and to others and to the ability to understand how mental states influence human behavior.

A well-developed ToM is crucial for making social inferences and guiding social behavior in everyday life communicative interactions. Importantly, people with ASD may have impaired ToM abilities (e.g., Baron Cohen, 1995; Happé, 1994), even when their performance on inference tasks that do not require understanding of mental states is unimpaired (Baron Cohen, 1995, 2001; Charman & Baron Cohen, 1992; Happé, 1994; Ozonoff, Pennington, & Rogers, 1991). The relationship between EF and ToM has received a great deal of attention (Fisher & Happé, 2005; Hughes & Ensor, 2007; Ozonoff et al., 1991; Pellicano, 2007) as these constructs seem to be highly interlinked (Frye, Zelazo, Brooks, & Samuels, 1996; Hala, Hug, & Henderson, 2003; Hughes, 1998; Perner & Lang, 1999; Sabbagh, Xu, Carlson, Moses, & Lee, 2006). For example, cognitive flexibility involves the ability to switch rapidly between multiple tasks and may be crucial to change strategies or perspective in ToM tasks or during everyday conversation. Moreover, ToM tasks require working memory (Mckinnon & Moscovitch, 2007) as intermediate steps are needed to perform well on various complex ToM tasks. The intermediate steps need to be kept in mind, evaluated, and perhaps adjusted, so more intermediate steps may require a larger involvement of working memory. In EF tasks such as classical cognitive flexibility tasks, ToM may play a role as participants have to adjust their behavior based on feedback given by the assessor of the task (Ozonoff & Miller, 1995). In most EF tasks the participants need to conceptualize (i.e., infer) what the experimenter wants them to do (see for details also Pellicano, 2007) which is an important aspect of ToM. To put it differently, to perform adequately on these EF tasks, one needs to have a representational understanding of mind (Perner & Lang, 2000). Hence, it is no surprise that EF and ToM deficits often go hand in hand.

Interestingly, the development of ToM seems intricately intertwined with the development of EF (e.g., Carlson, Mandell, & Williams, 2004; Hughes & Ensor, 2007). In fact, some researchers have argued that EF ability is necessary to perform adequately on many ToM tasks (Frye et al., 1996) and, more generally, that development of EF is a prerequisite for the development of ToM (e.g., Hughes, 1998; Russell, 1997). In contrast, some researchers have proposed a reverse relationship (e.g., Perner & Lang, 1999, 2000), namely, that the metarepresentational capacity that underlies ToM, the understanding that behavior is guided by internal states, is required for the development of EF. The results from longitudinal studies in typically developing children thus far support the notion that EF competence is important for the acquisition of ToM (Carlson et al., 2004; Flynn, O'Malley, & Wood, 2004; Hughes, 1998; Hughes & Ensor, 2007; Muller, Liebermann-Finestone, Carpendale, Hammond, & Bibok, 2012).

The results from studies that have focused on EF and ToM in ASD also underline the strong relationship between these two constructs (e.g., Pellicano, 2007, 2010a; Zelazo, Jacques, Burack, & Frye, 2002). However, the precise nature of the EF-ToM relationship remains unclear. On the one hand, the correlation analyses typical of most research studies do not allow for any causal inferences (but see Pellicano, 2007). On the other hand, however, training EF abilities in children with ASD seems to improve their performance on ToM tasks, whereas training on ToM does not result in improved EF (Fisher & Happé, 2005). These findings hint at the possibility that EF deficits are primary in ASD. Pellicano (2007) hypothesized that ToM and EF are crucially linked at an early stage of development when both abilities begin to emerge, but do not influence one another when children are older and conceptual understanding has been developed. To be able to establish whether this is indeed the case, more longitudinal studies are needed (see for an example Pellicano, 2010a). In all, the consensus thus far seems that EF and ToM abilities can interact with one another, share a developmental timetable, and are both impaired in people with ASD. In the current chapter we will focus on EF, but if relevant for interpreting the EF findings in relation to ASD, we will also discuss ToM studies.

Inhibition

Inhibition problems are often observed in day-to-day behavior in people with ASD. For example, the ability to generate appropriate responses during

social interactions involves selecting the most fitting response while inhibiting those responses deemed inappropriate. Also in language use it is necessary to inhibit one (frequently used) meaning of a word (e.g., a *bank* to sit on vs. a *bank* to withdraw money from) if you need to use the other (less frequently used) meaning of a word. Taking language literally is one of the often observed behavior characteristics in people with ASD. Repetitive behavior in individuals with ASD might also be due to difficulties in suppressing behavior even when the consequences are negative (e.g., Langen et al., 2012). In several studies (Geurts & De Wit, in press; Solomon, Ozonoff, Cummings, & Carter, 2008), the observed ASD behavior (as measured with parent reports, diagnostic interviews, or observational schedules) correlated with performance on inhibitory control tasks (but see Happé, Booth, Charlton, & Hughes, 2006). Hence, several key characteristics of ASD might be related to deficits in inhibitory control.

Since the first series of studies by Ozonoff and Russell and colleagues in the 1990s (Hughes & Russell, 1993; Hughes, Russell, & Robbins, 1994; Ozonoff & Strayer, 1997; Ozonoff, Strayer, Mcmahon, & Filloux, 1994), various research groups around the globe focused on inhibitory control in children and adolescents (Adams & Jarrold, 2009; Christ, Holt, White, & Green, 2007; Christ, White, Mandernach, & Keys, 2001; Corbett et al., 2009; Eskes, Bryson, & Mccormick, 1990; Geurts, Begeer, & Stockmann, 2009; Geurts, Luman, & Meel, 2008; Geurts et al., 2004; Goldberg et al., 2005; Happé & Frith, 2006; Johnson et al., 2007; Kilincaslan, Mukaddes, Kucukyazici, & Gurvit, 2010; Lee et al., 2009; Lemon, Gargaro, Enticott, & Rinehart, 2011; Mahone et al., 2006; Pellicano, 2007; Raymaekers, Van Der Meere, & Roeyers, 2006; Russo et al., 2007; Semrud-Clikeman, Walkowiak, Wilkinson, & Butcher, 2010; Sinzig, Morsch, Bruning, Schmidt, & Lehmkuhl, 2008) and adults (e.g., Agam, Joseph, Barton, & Manoach, 2010; Barnard, Muldoon, Hasan, O'Brien, & Stewart, 2008; Johnston et al., 2011; Kana et al., 2007; Langen et al., 2012; Mosconi et al., 2009; Nydén et al., 2010; Raymaekers, Antrop, Van Der Meere, Wiersema, & Roeyers, 2007; Schmitz et al., 2006) with ASD. The findings across these studies seem, at first sight, not very consistent as inhibitory control deficits in ASD did not come to the fore in various studies. This inconsistency of findings seems to be independent of the age of the participants. According to Luna et al. (2007), inhibitory control seems to be deficient in ASD throughout development even though there are developmental improvements in the capacity to inhibit in ASD.

Inhibition can be divided into prepotent response inhibition, resistance to distractor interference, and resistance to proactive interference (Friedman & Miyake, 2004), and a broad range of measures has been used to measure these three inhibitory control constructs. The inhibitory control impairments in people with ASD seem to be most prominent in resistance to distractor interference tasks (e.g., Adams & Jarrold, 2012; Christ, Kester, Bodner, & Miles, 2011; Geurts et al., 2008; but see Henderson et al., 2006; Johnston et al., 2011; Solomon et al., 2008), for example, called flanker (Eriksen & Eriksen, 1974) tasks, while proactive interference seems to be relatively intact (Bennetto, Pennington, & Rogers, 1996; Christ et al., 2011). On typical prepotent response inhibition tasks such as the Go-NoGo task (Casey et al., 1997) and the Stop task (Logan, 1994), findings seem to be inconsistent as various studies reported null findings (Adams & Jarrold, 2012; Christ et al., 2007, 2011; Eskes et al., 1990; Geurts, Begeer, et al., 2009; Goldberg et al., 2005; Happé & Frith, 2006; Kana et al., 2007; Kilincaslan et al., 2010; Ozonoff & Jensen, 1999; Ozonoff & Strayer, 1997; Raymaekers et al., 2007; Russell, Jarrold, & Hood, 1999; Schmitz et al., 2006; Semrud-Clikeman et al., 2010), while some others do report deficits in individuals with ASD (Adams & Jarrold, 2009; Corbett et al., 2009; Geurts et al., 2004; Johnston et al., 2011; Ozonoff et al., 1994; Raymaekers, Van Der Meere, & Roeyers, 2004). In a recent study Christ et al. (2011) suggest that the observed impairment in resistance to distractor interference might be due to a developmental delay which resolves with aging. This would suggest that adults with ASD will probably not have this type of inhibitory control impairments, but so far this has not been tested in sufficiently powered studies.

In most interference control tasks, but also in the Stop task (Logan, 1994) and the Stroop task (Macleod, 1991), the participants need to inhibit a formerly learned response to a specific stimulus. Yet, this is in contrast with a typical Go-NoGo task, in which a NoGo stimulus is typically not associated with a response. Hence, children with ASD might mainly have difficulties with inhibiting a learned response instead of having difficulties in just not responding. However, the null findings on Stroop like tasks contradict this interpretation (Christ et al., 2007; Goldberg et al., 2005; Kilincaslan et al., 2010; Semrud-Clikeman et al., 2010). An alternative explanation might lie in the role of working memory in most inhibitory control tasks. Previous studies have demonstrated that children with ASD are especially challenged by inhibitory control tasks with a heavy working memory load (Hughes & Russell, 1993; Joseph, Steele, Meyer, & Tager-Flusberg, 2005; Kana et al., 2007; Luna et al., 2007; Ozonoff & Strayer, 1997; Ozonoff et al., 1994; Russell, 1997). It could well be that working memory deficits are partly underlying the reported difficulties with inhibitory control (but see Christ et al., 2011). Nonetheless, so far the evidence suggests that people with ASD have difficulties in their ability to ignore and/or suppress irrelevant (interfering) information and there is no convincing evidence for an ASD-related impairment in prepotent response inhibition.

Working Memory

Individuals with ASD also seem to experience working memory (WM) problems, as, for example, a common complaint by parents is that their child with ASD is not able to execute instructions or commands. Even children with well-developed hearing and verbal understanding seem to demonstrate such difficulties in implementation of instructions. Especially when more than one instruction is given at once, individuals with ASD have difficulties to follow them all. This seems to be a WM problem; although information seems to be understood, and possibly stored, the transmission to actually manipulate and use the information subsequently seems to be disturbed (Baddeley, 1992). In everyday life WM is necessary in various situations, e.g., remembering directions while driving or remembering the name of someone who introduced himself. For children, WM is necessary when a teacher at school explains a future assignment or when parents instruct their children. Apart from this obvious role of WM, WM deficits might also influence social behavior as in social situations WM plays an important role. When meeting new people, it is necessary to introduce oneself, remember not just the name of the person you meet, but also the subject of the conversation. Moreover, for a smooth social interaction, it is important to remember, process, and interpret information like a person's face, facial expression, tone of voice, and body language. To be able to interact appropriately with others, new information needs to be stored and combined with familiar information and needs to be interpreted fast and accurately. These different aspects of social interaction require WM (Causton-Theoharis, Ashby, & Cosier, 2009). Hence, when individuals with ASD indeed encounter WM deficits, this is of crucial importance for their day-to-day functioning.

In the WM literature a distinction is often made between (1) the central executive, (2) the visual-spatial sketch pad, and (3) the phonological loop (Baddeley, 1992; Gathercole & Alloway, 2006; Gathercole, Pickering, Ambridge, & Wearing, 2004). However, in the ASD literature, the main distinction made is whether verbal or visual information needs to be processed. Therefore, the latter distinction will be discussed in this section. Overall, it seems that individuals with ASD do show deficits in both verbal and visual-spatial WM (Willcutt et al., 2008), but some argue that the deficits in visual-spatial WM are the most prominent (Williams, Goldstein, Carpenter, & Minshew, 2005; Williams, Goldstein, & Minshew, 2006).

Memory span tasks are often used to measure verbal WM; a list of stimuli (e.g., digits, letters, or sentences) has to be remembered and reproduced (Bennetto et al., 1996; Cui, Gao, Chen, Zou, & Wang, 2010; Gabig, 2008; Minshew &

Goldstein, 2001; Williams et al., 2005, 2006). Another verbal WM measurement, which is commonly used in ASD research, is the *n*-back task (Kana et al., 2007; Koshino et al., 2005, 2008; Williams et al., 2005). Verbal stimuli are visually displayed and participants have to alternately point out if a certain stimulus is similar to a target stimulus (0-back), the previous stimulus (1-back), or two stimuli earlier (2-back). The *n*-back task is thought to be mainly verbal as even pictures are mostly remembered in words (Williams et al., 2005). When WM load is minimal, individuals with ASD seem to have no impairment in verbal WM (Cui et al., 2010; Williams et al., 2005), but when a large amount of complex information has to be processed, individuals with ASD do show verbal WM deficits (Williams et al., 2006). More specifically, increasing WM load seems to impair children with ASD more than typically developing children (Cui et al., 2010). These deficits are reported in various age groups (Bennetto et al., 1996; Gabig, 2008; Minshew & Goldstein, 2001) and a similar pattern is seen in everyday life. Children with ASD seem particularly disabled when several complex or ambiguous tasks have to be performed consecutively, thus when WM load is high. When performing or finishing a relatively difficult task, WM seems to get overloaded. When given one task at a time, with clear instructions, or step-by-step guidance—hence low WM load—individuals with ASD are indeed able to perform one or more tasks.

Classical visual-spatial WM tasks widely used in ASD research are the Corsi Block-Tapping Task (Berch, Krikorian, & Huha, 1998; Corsi, 1972) and the highly similar CANTAB spatial WM task (Cambridge, 2002; Corbett et al., 2009; Goldberg et al., 2005; Happé et al., 2006; Landa & Goldberg, 2005; Sinzig et al., 2008; Steele, Minshew, Luna, & Sweeney, 2007) and the CANTAB spatial span task (Barnard et al., 2008; Cambridge, 2002; Corbett et al., 2009). Visual-spatial WM seems to be impaired in ASD when measured with the aforementioned tasks. Although not all studies are confirmative (Ozonoff & Strayer, 2001; Yerys, Hepburn, Pennington, & Rogers, 2007), evidence that there are actual problems in this area is increasingly convincing. Children with ASD show difficulty in storing, maintaining, and retrieving visual-spatial information (Corbett et al., 2009; Goldberg et al., 2005; Happé et al., 2006; Landa & Goldberg, 2005; Sinzig et al., 2008; Williams et al., 2005). Moreover, visual-spatial WM deficits seem to correlate with ASD symptoms (Verté, Geurts, Roeyers, Oosterlaan, & Sergeant, 2006a). Also adults with ASD do still show similar WM deficits (Barnard et al., 2008; Gomarus et al., 2009; Luna et al., 2007; Steele et al., 2007; Williams et al., 2005). In everyday life, individuals with ASD often use pictures, symbols, or icons to represent tasks that have to be performed and events that will happen during a certain day or period of time (Ganz, Davis, Lund, Goodwyn, & Simpson, 2011). It might be that this visually offered information supports the less well-developed visual-spatial WM. By displaying the pictures externally, WM load will be reduced, which might, in turn, increase self-reliance, by helping individuals with ASD to keep up with daily routines.

WM interacts with inhibitory control (see para. 2.1) but also with the other EF domains; to be able to execute a task, one needs to keep a certain rule in mind that needs to be followed (Barnard et al., 2008). WM and other EFs are thought to be mutual influential (Stoet & López, 2010). Not only does WM influence executive functioning per se, but under certain conditions, WM itself is used or triggered by other EFs. WM is influenced by, and influences, attention, inhibition, flexibility, and planning. In executive functioning, firstly, an individual has to pay attention to certain information. If information does not get proper attention, it will not be processed sufficiently and as a result, will not be stored and enter the WM process. Secondly, one can only attend to certain information, when other information will simultaneously be ignored (i.e., a response towards this information needs to be inhibited) as it is impossible to pay attention to, and process, all available information (Chun, Golomb, & Turk-Browne, 2011). Thirdly, one can only focus on one aspect of incoming information and ignore other information, when one can flexibly switch between a variety of available

information. Fourthly, to plan an action, WM is needed to trace, scan, and choose what information to use and react to. Especially when more complex tasks are used to measure EF constructs in individuals with ASD, such as cognitive flexibility and planning, it is important to determine the role of WM abilities on task performance as WM in itself is already impaired in individuals with ASD (Willcutt et al., 2008).

Cognitive Flexibility

In the diagnostic criteria of ASD (APA, 1994, 2000), stereotypical and repetitive behavior is the third domain of the ASD triad of symptoms. Also in the social and communication domains, inflexible behavior is part of the ASD criteria (see for a detailed review Geurts, Corbett, et al., 2009). This is one of the reasons why especially the EF construct of cognitive flexibility has an immediate appeal when one tries to explain ASD-related behavior. Cognitive flexibility involves the ability to rapidly switch between multiple tasks (Monsell, 2003) and may therefore be crucial for the ability to change strategies or perspective during everyday conversation. The difficulties of people with ASD to respond to unexpected events might also be related to an inability to flexibly adjust one's behavior to the changing environment. However, the face validity of this relationship between ASD symptoms and cognitive flexibility is difficult to reveal in experimental studies (see Geurts, Corbett, et al., 2009).

In a wide range of cognitive flexibility studies in ASD, the classical neuropsychological task, the Wisconsin Card Sorting Task (WCST; Berg, 1948), has been used (e.g., Bennetto et al., 1996; Griebling et al., 2010; Liss et al., 2001; Maes, Eling, Wezenberg, Vissers, & Kan, 2011; Minshew, Goldstein, Muenz, & Payton, 1992; Ozonoff et al., 1991; Prior & Hoffmann, 1990; Robinson, Goddard, Dritschel, Wisley, & Howlin, 2009; Rumsey, 1985; Sumiyoshi, Kawakubo, Suga, Sumiyoshi, & Kasai, 2011). In most of these studies, children and adults with ASD indeed seem to have cognitive flexibility deficits as they perform worse on the WCST compared to typically developing controls (Geurts, Corbett, et al., 2009). However, not just cognitive flexibility is of importance to perform well on the WCST. Difficulties with learning from feedback, keeping a goal of in mind (i.e., WM), noticing that a change in strategy is necessary, inhibiting a previous motor response, switching to another response, and sustaining responding over time can lead to a decreased WCST performance (Barcelo, 1999; Geurts, Corbett, et al., 2009; Ozonoff, 1995). As we discussed in the previous paragraphs, individuals with ASD seem to have deficits in specific aspects of, for example, inhibitory control and WM, and deficits in these EF domains might already decrease the WCST performance.

However, cognitive flexibility can be measured with a wide range of tasks and not just with the WCST. The difficulty with the ASD cognitive flexibility literature is that the findings of studies using other clinical neuropsychological tasks, or of studies using more experimental tasks, are rather inconsistent (Geurts, Corbett, et al., 2009). Some studies do report cognitive flexibility deficits (Hughes et al., 1994; Ozonoff et al., 2004; Reed & Mccarthy, 2012; Reed, Watts, & Truzoli, 2013; Yerys et al., 2007, 2009), while other do not report any deficits (Corbett et al., 2009; Goldberg et al., 2005; Happé & Frith, 2006; Poljac et al., 2009; Schmitz et al., 2006; Shafritz et al., 2008; Sinzig et al., 2008; Stahl & Pry, 2002; Whitehouse, Maybery, & Durkin, 2006). Studies differ, of course, in methodology (like choice of dependent measures, age, and diagnosis of participants), but this does not seem to be the main reason for the observed inconsistency in findings. In our earlier work (Geurts, Corbett, et al., 2009) we hypothesized that the failure to find cognitive flexibility deficits in ASD in relatively pure cognitive flexibility measurements (such as switch tasks) is due to the predictability of the switches in most of these tasks, while in day-to-day life switches are often unpredictable. In recent studies it was indeed shown that children with ASD are relatively cognitive inflexible when switches occur random and unpredictable (Maes et al., 2011; Stoet & López, 2010).

Various other alternative explanations for the inconsistency in findings have been explored resulting in a series of new studies focusing on cognitive flexibility in children and adults with ASD (e.g., Dichter et al., 2010; Geurts & Vissers, 2012; Griebling et al., 2010; Pellicano, 2010b; Poljac et al., 2009; Reed & Mccarthy, 2012; Robinson et al., 2009; Van Eylen et al., 2011; Yerys et al., 2009). For example, WM load varied largely in different studies and tasks, as a task cue can be available continuously (Schmitz et al., 2006), at the beginning of a task run (Poljac et al., 2009; Shafritz et al., 2008), or only when the task starts (Maes et al., 2011). On switch tasks with minimal WM demand, children with ASD do not show difficulties (Schmitz et al., 2006; Stoet & López, 2010), but when WM demand is higher, the results are inconclusive; some studies report difficulties in ASD (Maes et al., 2011; Shafritz et al., 2008; Stoet & López, 2010) and some do not (Poljac et al., 2009; Whitehouse et al., 2006). Moreover, performance on switch tasks is more influenced by WM demand in children with ASD than in typically developing children (Dichter et al., 2010; Stoet & López, 2010). While WM has probably a large influence on task performance (but see Russo et al., 2007), two alternative hypotheses (Maes et al., 2011; Van Eylen et al., 2011) might also shed some new light on the circumstances in which individuals with ASD do encounter cognitive flexibility deficits.

The first hypothesis is that the possibility to observe flexibility impairments is determined by the degree of explicitly provided task instructions (Van Eylen et al., 2011; see for similar ideas White, Burgess, & Hill, 2009). Van Eylen et al. (2011) classified cognitive flexibility tasks based on the explicitness of task instructions and concluded that the WCST (on which individuals with ASD generally fail) is the task with the lowest degree of explicit task instructions and typical experimental task switch paradigms (on which individuals with ASD generally succeed) have the highest degree of explicit task instructions. In a task switch paradigm where explicitness of task instructions was also low (Van Eylen et al., 2011), children with ASD indeed showed cognitive flexibility problems.

The second hypothesis is that novelty processing might be impaired in individuals with ASD (Maes et al., 2011), resulting in the perseverative behavior observed on WCST-like tasks in people with ASD. The idea is that individuals with ASD are less prone to respond to novel stimuli (see also Anckarsater et al., 2006) and, therefore, keep responding to familiar stimuli. Indeed when a paradigm was used in which the tendency to pay attention to novel or familiar stimuli could be disentangled, children with ASD seemed to favor familiar stimuli (Maes et al., 2011) suggesting reduced novelty processing in individuals with ASD.

In sum, unpredictability, high WM load, the lack of explicit task instructions, and reduced novelty processing might all contribute to the observed day-to-day difficulties in cognitive flexibility. Which account is the most plausible explanation for the inconsistent findings in past cognitive flexibility studies needs to be tested, but for now it seems that especially those cognitive flexibility tasks that are complex in various aspects are those that individuals with ASD cannot succeed on.

Planning

Besides cognitive flexibility problems, in daily life people with ASD often experience planning problems. For example, difficulties are encountered when making homework assignments, organizing morning activities in order to get to work in time, or when running a household. Impairments in communication and social interaction, key characteristics of ASD, might also partly be influenced by planning deficits. Parents or partners, for example, often organize all social appointments of their relative or partner with ASD in order to keep the social relations active. It is also known that at least in children with ASD, planning skills and ToM abilities are strongly related (Pellicano, 2007), for example, performance of children with ASD on a planning task predicts ToM abilities 1 year later, independent from age and verbal ability (Pellicano, 2010b). The fact that day-to-day difficulties in

planning are observed in individuals with ASD is not surprising as planning is a complex process of working towards a desired goal and various skills are needed, such as monitoring, reevaluating, and updating actions (Hill, 2004; Shallice, 1982). Hence, like cognitive flexibility, planning is a complex cognitive process in which both inhibitory control and WM are of importance (Newman, Carpenter, Varma, & Just, 2003; Welsh, Satterlee-Cartmell, & Stine, 1999; Zinke et al., 2010). It is simply not possible to plan and perform an action, without using information that is already stored, combining this information with new information, and ignoring irrelevant information. Planning usually consists of several steps, and each of these steps has to be stored but also adjusted to the changing context meaning that also cognitive flexibility is of importance for planning.

Planning is one of the EF domains that is most consistently found to be impaired in people with ASD as compared to typical developing groups (e.g., Bennetto et al., 1996; Booth, Charlton, Hughes, & Happé, 2003; Griebling et al., 2010; Lopez et al., 2005; Ozonoff & Jensen, 1999; Ozonoff & Mcevoy, 1994; Ozonoff et al., 1991; Pellicano, 2010a; Prior & Hoffmann, 1990). Moreover, planning seems even more impaired in individuals with ASD than in individuals with ADHD (Bramham et al., 2009; Geurts et al., 2004; Semrud-Clikeman et al., 2010), although these differences between ASD and ADHD groups are not confirmed in all studies (Booth et al., 2003). Moreover, some studies are not able to differentiate individuals with and without ASD with respect to planning (e.g., Boucher et al., 2005; Corbett et al., 2009; Happé & Frith, 2006; Liss et al., 2001). These null findings might challenge the idea of a general impairment in planning in people with ASD, but based on a meta-analysis of 21 ASD planning studies (Geurts & Bringmann, 2011), it seems that planning difficulties in ASD clearly exist, independent of which ASD diagnosis an individual has and which age. Not just children (e.g., Landa & Goldberg, 2005; Ozonoff et al., 2004; Pellicano, 2010a; Semrud-Clikeman et al., 2010; Verté et al., 2006a; Zinke et al., 2010) but also adults with ASD are impaired in planning compared to typically developed adults (e.g., Bramham et al., 2009; Hill & Bird, 2006; Just, Cherkassky, Keller, Kana, & Minshew, 2007; Lopez et al., 2005). Normally, planning performance improves across development, but this improvement does not seem to be quite evident in individuals with ASD (Keary et al., 2009; Ozonoff & Mcevoy, 1994). Although in a recent longitudinal study (across a 3 year period) young children with ASD showed a steeper increase in planning performance as compared to typically developing peers, the control group still outperformed the ASD group. This suggests that the developmental delay in planning abilities in children with ASD indeed remains present across development (Pellicano, 2010a). Future research is needed to gain insight in the developmental trajectories of planning skills in people with ASD.

As the planning tasks and reported outcomes in planning studies are often very different, it is challenging to make a comparison across studies (see for recent reviews Geurts & Bringmann, 2011; Hill, 2004). The inconsistencies are partly due to the fact that tasks used to measure planning often only correlate moderately (e.g., Tower of Hanoi and Tower of London-Revised, Welsh et al., 1999). Also, the large range of IQ levels in most studies makes it difficult to determine whether ASD or learning disabilities is the main contributing factor to the observed planning problems (Hill, 2004). For example, planning problems in one study (Mari, Castiello, Marks, Marraffa, & Prior, 2003) seem to be due to IQ level, while in another study (Hughes et al., 1994) planning impairments seem to be ASD specific as people with ASD are also impaired when compared to a group with moderate learning disabilities. Across the used tasks it seems that especially the so-called Tower tasks, except the "Stocking of Cambridge" (SoC), are the most effective in determining planning problems in people with ASD (Geurts & Bringmann, 2011). The SoC is a computerized task and might therefore be less related to planning in daily life, because for individuals with ASD it seems easier to perform on a computerized task than a task requiring more social interaction (Ozonoff, 1995).

Hence, the task choice does influence the outcome of ASD planning studies.

There is a cognitive neuroarchitecture model (called 4CAPS) of problem solving with a Tower task (Just & Varma, 2007). The basic idea of this model is that functional connectivity is crucial for efficient problem solving; multiple cortical networks perform multiple cognitive functions in specialized, as well as dynamic, ways (Just & Varma, 2007). This is of interest for understanding the ASD-related planning impairments, as ASD is more and more considered a brain connectivity disorder (Courchesne & Pierce, 2005; Just et al., 2004; Schipul et al., 2012; Vissers et al., 2012; Wass, 2011). The difficulty levels of Tower assignments are correlated with activation in the right and left dorsolateral prefrontal cortex and with the left, but not right superior parietal regions (Newman et al., 2003). In the 4CAPS model, four collaborating centers are proposed: the left and right hemisphere executive centers (in the dorsolateral prefrontal cortex) and the left and right spatial centers (in the superior parietal regions). The right hemisphere executive center is hypothesized to be important in strategic control by selecting and planning the moves, while the left hemisphere executive center is involved in controlling and executing the planning process. The left spatial center is proposed to spatially transform the Tower image by imagining moving the objects and, thereby, controlling the execution of planning. The right spatial center would only generate perceptual moves and, therefore, is not linked to planning difficulty (Newman et al., 2003). Fitting such a model to data obtained from individuals with ASD (see, e.g., Griebling et al., 2010 for a Tower related imaging study with ASD) could potentially inform us what brain network deficiencies underlie the observed difficulties with planning tasks in ASD.

More work involving mathematical models of cognitive functioning in planning tasks but also regarding other EF tasks would be of importance to unravel how and when individuals with ASD do or do not encounter EF deficits. This is especially important as not just in planning but also in other EF domains, the needed cognitive processes to perform well on the tasks are highly intertwined. It is not clear whether difficulties on a wide range of EF tasks are due to just one underlying deficient cognitive process or whether especially performing multiple cognitive processes at the same time is the reason for the observed failures on EF tasks.

Is ASD an Executive Function Disorder?

ASD cannot be described as an EF disorder as (1) many individuals with ASD do not encounter EF deficits (Geurts et al., submitted; Johnston et al., 2011; Pellicano, 2010b) and (2) EF deficits are not specific for people with ASD (Pennington & Ozonoff, 1996; Sergeant et al., 2002; Willcutt et al., 2008). However, as EF deficits are more common in individuals with ASD as compared to typical developing individuals, it is important to study EF in relation to ASD. That is, the idea that get its feet on the ground more and more is that ASD results from an interacting compound of cognitive deficits (and/or styles such as EF, ToM, and weak central coherence) and no single deficit might be sufficient or even necessary for the diagnostic symptom profile to arise (e.g., Happé & Ronald, 2008; Happe et al., 2006).

Various challenges for the field have been discussed in the different sections within this chapter as there is currently no consensus regarding the type of tasks which has the highest validity to measure EF in ASD, there is no consensus regarding the dependent variables that need to be reported, nor is there consensus regarding the variables we need to control for when choosing appropriate control group. However, the field is progressing as more and more EF studies include mathematical models (Just & Varma, 2007) and experimental paradigms (see, e.g., Christ et al., 2011; Maes et al., 2011; Solomon et al., 2009) in which different cognitive processes that might be affected in ASD can be disentangled.

We feel that there are two other major challenges for EF researchers. First of all, for people with ASD it could be helpful if we establish which individuals with ASD do encounter EF

deficits and which persons do not show deficits. This is important as this might have implications for the determining which interventions are the most suitable for a specific individual. For example, children with ASD with a cognitive flexibility deficit (as measured with the WCST) might not benefit from social skills training, while children without such a deficit do (Berger, Aerts, Spaendonck, Cools, & Teunisse, 2003). Whether one does or does not have certain EF deficits could also be of importance when one wants to train EF. Given that EF deficits are so widely studied in ASD, it is surprising that, as far as we know, only one study focused on training EF (Fisher & Happé, 2005). This is especially startling given that EF training (mainly WM training) seems an effective intervention for ADHD (Beck, Hanson, Puffenberger, Benninger, & Benninger, 2010; Holmes et al., 2009; Klingberg, Forssberg, & Westerberg, 2002; Klingberg et al., 2005; White & Shah, 2006), a neurodevelopmental disorder which is often comorbid with ASD (Rommelse, Franke, Geurts, Hartman, & Buitelaar, 2010). A first pilot study by Fisher and Happé (2005) indeed showed that training EF (focusing on cognitive flexibility) improved the performances of children with ASD on ToM tasks. In this study children with ASD received ToM training, EF training, or no intervention. Both training programs had a strategy-based approach which was adjusted for each participant. In each training session different rules were learnt and the trainer used objects and illustrative stories to explain the rules. Directly after the ToM training sessions, the children improved on ToM tasks, but not on EF tasks. In contrast, directly after the EF training sessions, the children did neither improve at the ToM nor at the EF tasks. However, at follow-up (6–12 weeks later) all children who received a ToM training or an EF training improved in their ToM performance. These results suggest that children with ASD could pass ToM tasks after both ToM and EF training. However, the EF did not improve at all. Both the ToM and the EF training failed to reduce the EF difficulties in these children with ASD. This might suggest that in ASD EF cannot be trained, while in the ADHD literature, game-like computerized training programs seem to be successful, especially when the focus is on WM (Beck et al., 2010; Holmes et al., 2009; Klingberg et al., 2002, 2005). The first preliminary findings of our study in which we compare a game-like training of WM and cognitive flexibility, with a non-EF computer training in ASD, do suggest that children with ASD improve in their day-to-day EF skills (De Vries, Prins, Schmand, & Geurts, 2011). Hence, to determine whether or not EF training will indeed be a candidate intervention for people with ASD, more research is needed. However, when studying this type of interventions, one needs to take into account that there are individual differences in EF deficits in individuals with ASD, as the profile of EF deficits and strengths might be of great importance for the failure or success of such an intervention (Berger et al., 2003).

The second challenge for ASD researchers is to incorporate a developmental perspective. ASD is a neurodevelopmental disorder, but studies often focus on one specific age range when studying ASD. As described in both childhood and adulthood, individuals with ASD show a broad range of EF deficits, but these findings are not unambiguous. The developmental pattern of EF in children and adolescents with ASD appears to be atypical (Happé et al., 2006; Luna et al., 2007; Pellicano, 2010a). For example, children with autism between 8 and 11 years of age showed several EF deficits, while these deficits did not emerge in children with autism aged 11–16 years (Happé et al., 2006). Also, in a recent longitudinal 3 year follow-up, planning capacity in children with autism improved at a faster rate than that of typically developing children (Pellicano, 2010a). Hence, these findings indicate that at least some EF deficits decline when aging. However, this idea of abating deficits might be in contrast with adult studies in which executive dysfunctions are still present in individuals with ASD above 16 years of age (e.g., Ambery et al., 2006; Bramham et al., 2009; Geurts & Vissers, 2012; Goldstein, Johnson, & Minshew, 2001; Hill & Bird, 2006; Lopez et al., 2005; Minshew, Meyer, & Goldstein, 2002). In a cross-sectional developmental study (Luna et al., 2007) executive dysfunctions were present in

people with ASD of different ages (8–12, 13–17, and 18–33 year). Across the three age groups, the autism group encountered inhibitory control deficits as well as WM deficits. However, developmental improvements in inhibitory control were similar in both groups (i.e., parallel development), while the development of WM was impaired in the autism group (Luna et al., 2007). The smaller extent of improvements in EF in children has also been reported in two longitudinal studies (Griffith, Pennington, Wehner, & Rogers, 1999; Ozonoff & Mcevoy, 1994). The combined findings from cross-sectional and longitudinal studies suggest that there are different developmental patterns for different aspects of EF. So studies focusing on both *how* and *when* EFs are disturbed are needed to fully grasp the EF impairments of individuals with ASD.

Although the aforementioned avenues for research might make it even more challenging to make clear statements about the ASD group as a whole, the large individual differences by themselves do give information as the ASD group is apparently a heterogeneous group. In sum, individuals with ASD do seem to experience EF problems, but (1) not all individuals with ASD do so, (2) not in the same areas, (3) not with similar severity, and (4) not all individuals have similar compensatory skills.

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