
CRITICAL REVIEW

Puppets, robots, critics, and actors within a taxonomy of attention for developmental disorders

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Abstract

This review proposes a new taxonomy of automatic and controlled attention. The taxonomy distinguishes among the role of the attendee (puppet and robot, critic and actor), the attention process (stimulus orienting vs. response control), and the attention operation (activation vs. inhibition vs. adjustment), and identifies cognitive phenotypes by which attention is overtly expressed. We apply the taxonomy to four childhood attention disorders: attention deficit hyperactivity disorder, spina bifida meningocele, traumatic brain injury, and acute lymphoblastic leukemia. Variations in attention are related to specific brain regions that support normal attention processes when intact, and produce disordered attention when impaired. The taxonomy explains group differences in behavioral inattention, hyperactivity, and impulsiveness, as well as medication response. We also discuss issues relevant to theories of the cognitive and neural architecture of attention: functional dissociations within and between automatic and controlled attention; the relative importance of type of brain damage and developmental timing to attention profile; cognitive-energetic models of attention and white matter damage; temporal processing deficits, attention deficits and cerebellar damage; and the issue of cognitive phenotypes as candidate endophenotypes. (*JINS*, 2008, *14*, 673–690.)

Keywords: Attention deficit hyperactivity disorder, Spina bifida, Traumatic brain injuries, Acute lymphoblastic leukemia, Mesencephalon, Frontal lobe

INTRODUCTION

Attention is unobservable, so models of attention and its components are based on inferences about how an individual perceives, thinks, and acts. This article presents a new, three-dimensional functional taxonomy that organizes contingent relationships among perception, cognition, and movement into a framework for understanding attention and its disorders in children. Herein, we:

- a. Describe the architecture of the taxonomy in terms of three unobservable constructs and an observable cognitive phenotype.
- b. Apply the taxonomy to four childhood attention disorders, making functional comparisons within each disorder.
- c. Demonstrate compatibility of the taxonomy with the neurobiology of attention.
- d. Compare behavioral profiles of inattention, impulsivity, and hyperactivity across disorders and predict treatment responsiveness.
- e. Discuss some theoretical issues pertaining to attention and predict attention profiles in disorders not yet studied.

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	STIMULUS ORIENTING		RESPONSE CONTROL	
	PUPPET •Bottom-up •Stimulus-driven	ROBOT •Top-down •Task-driven	CRITIC •Looped •Model-driven	ACTOR •Top-down •Action-driven
Activate	<i>Engage</i>	<i>Focus</i>	<i>Divide</i>	<i>Sustain</i>
Inhibit	<i>Inhibition of Return</i>	<i>Negative Priming</i>	<i>Conflict</i>	<i>Cancel</i>
Adjust	<i>Disengage</i>	<i>Set Shift</i>	<i>Monitor</i>	<i>Restrain</i>

Fig. 1. Attention taxonomy.

ATTENTION TAXONOMY

Stimulus Orienting Versus Response Control

Models of attention include both stimulus orienting and response control. Stimulus orienting is the automatic capture of attention by salient sensations (James, 1890; more recently, automatic disengaging and shifting attention, Mirsky et al., 1991; Posner & Peterson, 1990; filtering of salience information, Knudsen, 2007; or creating saliency maps, Treue, 2003). Response control is the voluntary direction of a motor response, corresponding to Posner and Peterson's (1990) anterior attention system and to Knudsen's (2007) response selection. Being an unobservable construct, attention is inferred in both stimulus orienting and response control from manipulations of the relation between sensation and movement. In stimulus orienting, inhibition of return (IOR) is inferred from the contingencies between motor engaging and disengaging. In response control, stopping an ongoing action is inferred from the relation between go and stop actions.

A new taxonomy (Figure 1) was prompted by considerations relevant to the specific aims. First, we aimed to integrate stimulus orienting and response control into a single taxonomy. Some models of attention used in childhood clinical disorders focus on only one process; for example, Barkley's (1997) model of attention considers only response control. Second, we wanted to distinguish among operations within stimulus orienting and response control. Some earlier models of attention that considered both processes (e.g., Mirsky et al., 1991; Posner & Peterson, 1990) have not separated activation, inhibition, and adjustment. Third, we aimed to incorporate recent parsings of attendee roles (e.g., the distinction in striatal learning between a "critic" who evaluates performance and an "actor" who performs) and cognitive phenotypes (e.g., the distinction in response control between restraining a prepotent response and canceling an ongoing response). Fourth, we wanted to integrate the three attention dimensions into a factorially structured taxonomy to identify both functional assets and

deficits. Such an integration will facilitate functional comparisons both within and across clinical disorders and generate not only descriptions, but also principled inferences about the neural and behavioral correlates of attention.

Puppet Versus Robot Versus Critic Versus Actor

Within stimulus orienting, the attendee may be a *puppet* or a *robot*. We devised the *puppet* metaphor to capture the idea of a passive attendee whose attention is driven by bottom-up, stimulus-driven orienting to exogenous (external) stimuli, such as a flash of light. We devised the *robot* metaphor to capture the idea of an active attendee whose orienting is top-down and task-driven, in accord with endogenous (internal) programs, scripts, or symbols, such as an arrow.

Within response control, the attendee may be a *critic* or an *actor*. The *critic* metaphor captures the idea of looped, model-driven responding, involving an active attendee who evaluates performance options and reward contingencies in light of an existing model of desired behavior. The *actor* metaphor captures the idea of a top-down, action-driven, active attendee responding in accordance with an instructional set and attention priorities. We adopted the actor-critic metaphors from habit-driven learning (Dayan & Balleine, 2002; O'Reilly & Frank, 2006) and animal studies separating a dorsal striatum (actor) role in performance from a ventral striatum (actor and critic) role in performance and learning (Atallah et al., 2007).

Activation Versus Inhibition Versus Adjustment

The attendee is involved in *activation*, *inhibition*, or *adjustment*. Activation enables directed attention towards the attended material. Inhibition refers to diverse attention processes, some of which concern automatic avoidance of previously attended locations (e.g., IOR), and some of which involve voluntary acts of inhibitory control (e.g., stopping an ongoing action in response to a signal). Adjustment refers to a number of evaluative-regulative processes by which previous attention modifies subsequent attention (Larson et al., 2007), including error detection, performance monitoring, and adjusting the contingency between present action and future reward (Holroyd & Coles, 2002; Nigg et al., 2005; Sagvolden et al., 1998).

Attention Measures Express Cognitive Phenotypes

The italicized entries in Figure 1 are cognitive phenotypes, which are overt and measurable expressions of the attention constructs. For each cognitive phenotype, we identify a representative task measure, below.

The attendee as puppet

Activation enables automatic stimulus engagement. The *engage* cognitive phenotype may be measured by the time

to orient to a target following an exogenous cue, such as a flash of light (Posner & Peterson, 1990). *Inhibition* enables the attendee to avoid orienting to a previously attended location or stimulus. The *IOR* cognitive phenotype is indexed by a longer time to return to a previously attended cue location (Klein, 2000). *Adjustment* enables a disengage process to withdraw attention from one stimulus so that it may be moved to another. The *disengagement* cognitive phenotype may be measured by disengagement cost, the time to detach from an exogenous cue conditionalized on engage time (Dennis et al., 2005a).

The attendee as robot

Activation enables a voluntary focus on the stimulus. The *focus* cognitive phenotype may be measured by time to attend to a target following an endogenous cue, such as a symbol or an arrow (Posner & Peterson, 1990). *Inhibition* slows responses to recently ignored stimulus relative to new stimuli. The *negative priming* (NP) cognitive phenotype may be measured by a longer time to attend to recently ignored stimuli (Tipper, 1992). *Adjustment* enables set shift to redirect attention. The *shift* cognitive phenotype may be measured by shifting attention in response to an endogenous cue (Schmitter-Edgecombe & Langill, 2006).

The attendee as critic

Activation enables a voluntary allocation of attention. The *divide* cognitive phenotype may be measured by the ability to activate concurrent attention streams (e.g., Manly et al., 1999). *Inhibition* enables the suppression of one of two interfering schemata or behaviors. The *conflict* cognitive phenotype may be measured by the ability to perform a controlled act while inhibiting a prepotent or competing response (e.g., to say “Day” for a moon picture, Gerstadt et al., 1994, or to inhibit an automatic process like word decoding in favor of a controlled process like color naming, Stroop, 1935). *Adjustment* enables monitoring of response conflict, errors, and top-down response control (Larson et al., 2007). The *conflict* cognitive phenotype may be measured by error detection and performance adjustment (Holroyd & Coles, 2002).

The attendee as actor

Activation concerns response control. The *sustain* cognitive phenotype may be measured by the slope of the curve of attentional vigilance over time (Seidel & Joschko, 1990). *Inhibition* enables cancellation or withdrawal of a response being executed. The *cancel* cognitive phenotype may be measured by canceling an act being executed (e.g., the stop-signal task, Logan, 1994). *Adjustment* enables withholding or delaying a prepotent response. The *restrain* cognitive phenotype may be measured by restraining a response (e.g., Axelrod et al., 1978) or delaying an action (e.g., Gordon, 1983).

Interrelation of Attention Processes

The taxonomy parses a set of attention processes and roles that normally work together in a complex choreography (Mesulam, 1990). Bottom-up, top-down, and looped operations operate in the same attention space. Stimulus orienting serves as a circuit breaker for response control (as when a flash of lightning draws attention from a book and we orient to the window). Dissociable inhibitory processes work together; for example, IOR not only aids visual search, but also helps adjust behavioral demands in a dynamic environment (Ivanoff & Taylor, 2006). Arousal influences the level of activation. Controlled attention helps maintain instructional set, even for stimulus orienting tasks.

While the key to the taxonomy is attendee roles in stimulus orienting and response control, time is a thread throughout, and the terms *time binding* (Dennis, 2006) and *intertemporal competence* (Barkley, 1997) characterize temporal processes in attention, such as time estimation, time management, and rule maintenance (Barkley et al., 1997). The taxonomy does not explicitly address working memory, which is the product of currently activated attention, including the activated subset that can be manipulated or inhibited (i.e., the products of the right side of Figure 1). Although it is not an account of executive function, the taxonomy does address executive constructs like response control, conflict, and monitoring.

APPLYING THE TAXONOMY TO CHILDHOOD ATTENTION DISORDERS

Overview

Disordered attention characterizes several childhood conditions, including attention deficit hyperactivity disorder considered as a *primary* form of adaptive impairment (P-ADHD, Barkley, 1997), and other congenital brain malformations such as spina bifida meningomyelocele (SBM; Dennis et al., 2005a, 2005b) and Sotos syndrome (a haploinsufficiency of the Nuclear receptor Set Domain containing protein 1 gene, NSD 1; de Boer et al., 2006; Kurotaki et al., 2002). Impaired attention is also a consequence of acquired brain insults, including traumatic brain injury (TBI; Levin et al., 2007), childhood brain tumors (Dennis et al., 1998), and acute lymphoblastic leukemia (ALL; Schatz et al., 2004).

Using the taxonomy in Figure 1, we now review attention in four disorders that vary in prevalence, etiology, and developmental course: P-ADHD, TBI, SBM, and ALL. Attention deficits define P-ADHD, and occur in a subset of children with the three other disorders, in which the presenting problem is explicit brain injury. In P-ADHD and SBM, attention is disordered early in development; in contrast, in TBI and ALL, disordered attention is acquired after some period of normal development.

To facilitate the review, we consider two paradigmatic tasks for stimulus orienting (covert orienting, Figure 2) and response control (stop signal, Figure 3). Most components

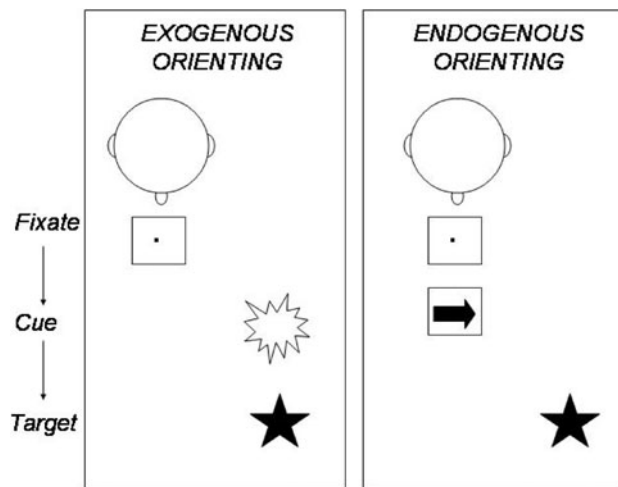


Fig. 2. Covert stimulus orienting paradigms. In exogenous orienting (left), the participant maintains central fixation and then an exogenous cue, such as a luminance change, appears to one side of fixation, followed by a target, to which the participant must respond. In this example, the brightness cue will facilitate target detection because it draws attention to the side on which the target will appear (a misleading cue would have appeared on the side opposite to the upcoming target). In endogenous orienting (right), the participant maintains central fixation, which is then replaced by a central endogenous cue, such as an arrow, followed by a target, to which the participant must respond. In this example, the arrow cue will facilitate target detection because it draws attention to the side on which the target will appear (a misleading arrow would have directed attention to the side opposite to the upcoming target).

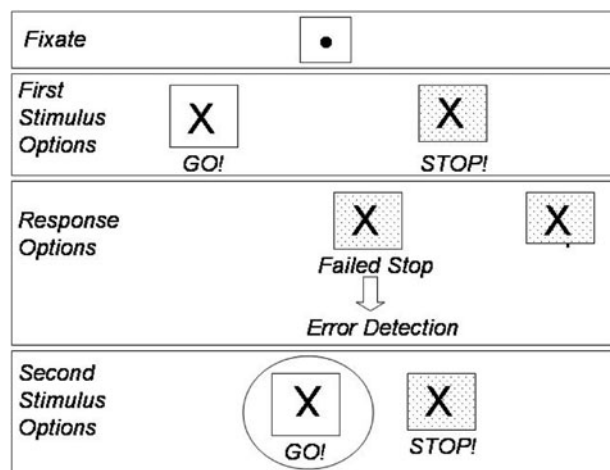


Fig. 3. Stop signal paradigm. The participant fixates a central dot and then a go stimulus appears, either an X indicating a left hand response (shown in figure) or an O indicating a right hand response. One-third of the trials involve a stop signal (a background color change) following the go signal to indicate that the participant should not respond. Because of the adaptively manipulated delay interval between go and stop signals, each participant will fail to stop on half of the stop trials. Failed stop trials activate the error detection system, so that go trials that follow a failed stop trial (circled in figure) will be slower than go trials that do not follow failures to stop.

of Figure 1 can be understood in the context of these two tasks. We also summarize the review in Table 1.

Primary Attention Deficit Hyperactivity Disorder

P-ADHD affects 4–7% of children worldwide (Szatmari, 1992) and is defined by hyperactive, impulsive, inattentive, and/or maladaptive behavior (Barkley, 2004; Goldman et al., 1998; Jensen et al., 1999). P-ADHD persists into adulthood, although heterogeneity in ADHD is considerable throughout development (Barkley et al., 2008), with hyperactivity being more common in children than in adults (Biederman et al., 2000).

Puppet and robot

Meta-analysis of 14 studies on exogenous stimulus orienting (Figure 2) in children with P-ADHD show preserved engage and move operations (Huang-Pollock & Nigg, 2003). Children with P-ADHD have normal IOR (Li et al., 2003). Neither adolescents (Pritchard et al., 2007) nor adults (Nigg et al., 2002) with P-ADHD exhibit difficulties with NP. Children with P-ADHD show no set shifting deficits (Piek et al., 2007; Riccio et al., 2006).

Critic and actor

Children with P-ADHD often have response inhibition deficits (Barkley, 1997; McLean et al., 2004; Nigg, 2003; Pennington & Ozonoff, 1996; Rhodes et al., 2005; Schachar et al., 1995; Westerberg et al., 2004), especially on the stop signal task (Figure 3; Willcutt et al., 2005). Children with P-ADHD have deficits both in the ability to cancel a prepared response with a signal to stop (stop signal presented with a variable delay after go signal) and to restrain a strong response tendency pending a signal to stop (stop and go signals presented concurrently) (Schachar et al., 2007). These children cannot delay responding to achieve a motivationally salient outcome (Kuntsi et al., 2001; Neef et al., 2001; Schweitzer & Sulzer-Azaroff, 1995; Sonuga-Barke, 1994; Sonuga-Barke et al., 1996; Tripp & Alsop, 2001).

Divided attention is impaired in children with P-ADHD, whether treated or drug-naïve (Pasini et al., 2007; Tucha et al., 2006). Children and adults with P-ADHD are impaired on Stroop interference (Lansbergen et al., 2007), suggesting conflict impairment. Children with P-ADHD fail to slow after errors, indicating difficulties in monitoring errors, post-error adjustment, or conflict management (Schachar et al., 2004b).

Children with P-ADHD struggle to sustain attention over time (Seidel & Joschko, 1990); they show increased reaction time (RT) over trial blocks and commit more omission errors (Anderson et al., 2006; Brewer et al., 2001; but see Huang-Pollock & Nigg, 2003). Increased performance variability includes transient temporal fluctuations in attention (Teicher et al., 2004), more variable RT to primary task stimuli (Alderson et al., 2007), lower response predictabil-

Table 1. Robots, puppets, critics, and actors: The fractionation of attention in developmental disorders

	Puppet	Robot	Critic	Actor
P-ADHD				
<i>Activate</i>	Intact	Intact	Impaired	Impaired
<i>Inhibit</i>	Intact	Intact	Impaired	Impaired
<i>Adjust</i>	Intact	Intact	Impaired	Impaired
SBM				
<i>Activate</i>	Impaired	Impaired	?	Intact
<i>Inhibit</i>	Impaired	Intact	Intact	Intact
<i>Adjust</i>	Impaired	Intact	Intact	?
TBI				
<i>Activate</i>	Intact	?	Impaired	Impaired
<i>Inhibit</i>	?	Intact	Impaired	Impaired
<i>Adjust</i>	Intact	Intact	Impaired	Impaired
ALL				
<i>Activate</i>	Impaired	Impaired	?	Intact
<i>Inhibit</i>	?	?	?	Intact
<i>Adjust</i>	Impaired	Impaired	?	Intact

Note. Bold entries are from strong, clear data; regular text entries represent a less clear, more limited, or more qualified database, or data from related tasks or age group. Intact or impaired refer to group, not individual, data.

ity (Aase et al., 2006; Aase & Sagvolden, 2005), higher intraindividual variability (Douglas, 1999; Klein et al., 2006), and increased RT, especially in the slow tail of the distribution (Hervey et al., 2004, 2006; Leth-Steensen et al., 2000).

Spina Bifida Meningocele

Spina bifida is a common disabling birth defect, occurring in about 19.3 per 100,000 live births in North America (Martin et al., 2006). The most severe form, spina bifida meningocele (SBM), occurs in 90% of cases (Detrait et al., 2005). Compared to siblings or to normative samples, children with SBM are more distractible and less attentive (Ammerman et al., 1998; Burmeister et al., 2005; Colvin et al., 2003; Rose & Holmbeck, 2007; Vachha & Adams, 2005) and around one-quarter to one-third is inattentive (Burmeister et al., 2005; Davidovitch et al., 1999; Fletcher et al., 2005). It is not clear whether attention deficits extend into adulthood.

Puppet and robot

Children with SBM show accurate but slow covert orienting to both exogenous and endogenous cues (a deficit in the engage and focus processes; Figure 2), but increased disengagement costs only to exogenous cues (a deficit in the disengage/move process, Dennis et al., 2005a). Those with beaking of the midbrain tectum show attenuated IOR (Dennis et al., 2005b). Their normal disengagement costs for endogenously cued information suggests intact NP.

Critic and actor

Children with SBM have not been evaluated on tests of divided attention. They exhibit difficulties with speed of response rather than conflict; for example, they show defi-

cient naming speed but not poorer Stroop interference (Fletcher et al., 1996a,b). Some monitor skills appear intact: They adapt to prismatic distortion (Colvin et al., 2003), respond to error information with corrective saccades (Salman et al., 2006b), and recalibrate movement after forced ballistic movement errors (Dennis et al., 2006).

Children with SBM can sustain attention over time, and their RT does not increase over time (Brewer et al., 2001; Swartwout et al., in press). Studies of commission errors are not consistent even on similar tasks, with some finding decreased (Colvin et al., 2003) and others finding increased error rates (Swartwout et al., in press), so the nature of restraint is unclear, especially in the absence of prototypic response control tasks (see Figure 3). Delay has not been studied.

Traumatic Brain Injury

Traumatic brain injury (TBI) is frequent in children and adolescents (180 in 100,000 youths annually, Kraus & McArthur, 1996; Kraus et al., 1990), and long-term neuropsychological deficits include inattention (Yeates, 2000). Premorbid P-ADHD contributes to post-TBI attention symptoms (Gerring et al., 1998, 2000; Levin et al., 2007; Max et al., 1998), although some 15–20% of TBI survivors with no ADHD history meet diagnostic criteria for secondary ADHD (S-ADHD; Gerring et al., 1998, 2000; Herskovits et al., 1999; Max et al., 2005b, 2005c; Slomine et al., 2005; Yeates et al., 2005), and even more develop subthreshold attention symptoms.

S-ADHD is associated not only with severe TBI (Max et al., 2004) but also with mild or moderate TBI (Gerring et al., 1998, 2000; Herskovits et al., 1999; Max et al., 1998; Max et al., 2005a, 2005b; Schachar et al., 2004a; Slomine et al., 2005), as well as with high levels of pre-

injury behavioral difficulties (Schachar et al., 2004a), maladaptive function, or psychosocial adversity (Gerring et al., 1998; Max et al., 2004, 2005b, 2005c). S-ADHD manifests as early as 3 months post-TBI (Gerring et al., 1998, 2000) and persists over years (Gerring et al., 1998; Levin et al., 2007; Max et al., 2005c). Inattentive symptoms peak at six months post-injury and later decline, whereas hyperactive/impulsive symptoms fluctuate for two years (Levin et al., 2007) and then diminish (Levin et al., 2007; Max et al., 2005b, 2005c). Although poor inhibitory control improves over time (Leblanc et al., 2005), children with TBI with or without S-ADHD exhibit impaired long-term attention (e.g., Dennis et al., 1995; Konrad et al., 2000a, 2000b; Schachar et al., 2004a).

Puppet and robot

Young adults with severe TBI have intact engage and disengage/move processes (Bate et al., 2001). Adults with TBI shift set as well as controls (Schmitter-Edgecombe & Langill, 2006) and show no NP deficits (Ries & Marks, 2005; Simpson & Schmitter-Edgecombe, 2000), suggesting intact endogenous stimulus orienting.

Critic and actor

Children with mild or severe TBI slow less than controls after making an error (Ornstein et al., in press), suggesting monitor impairment. Children with severe TBI have divided attention deficits, especially as task demands increase (Cattoppa et al., 2007). Conflict difficulty characterizes TBI: Stroop errors occur more frequently in both the short term and long term in children and adolescents with mild TBI complicated by abnormalities detected by computed tomography (CT) scans (Levin et al., 2008) or with moderate-severe TBI (Chadwick et al., 1981; Nolan & Mathieu, 2000; Ward et al., 2006). Children with TBI have difficulty sustaining attention (Dennis et al., 1995), and those with S-ADHD tend to have longer RT (Slomine et al., 2005). Children with TBI, or children with S-ADHD and TBI, exhibit longer stop signal RT than controls (Konrad et al., 2000a, 2000b; Leblanc et al., 2005), especially those with more severe injury and a *de novo* diagnosis of S-ADHD (Schachar et al., 2004a), suggesting cancel difficulty. Children with TBI make more commission errors (Konrad et al., 2000a; Levin et al., 2004), and exhibit an inability to delay their motor responses, with or without reward (Dennis et al., 1995; Konrad et al., 2000a), suggesting difficulty with cancel/restrain processes.

Acute Lymphoblastic Leukemia

Of the 3,000–6,000 people diagnosed with acute lymphoblastic leukemia (ALL) each year in the United States, two-thirds are children (Cortes & Kantarjian, 1995; Parker et al., 1997), and 80% will survive (Parker et al., 1997). ALL is associated with cognitive deficits, including poor attention, after treatment with cranial radiation therapy (CRT) and/or

chemotherapy, (Christie et al., 1995; Cousens et al., 1988; Fletcher & Copeland, 1988; Goff et al., 1980; Hertzberg et al., 1997; Mennes et al., 2005), especially with early exposure and longer time since treatment (Cousens et al., 1988; Jankovic et al., 1994). Attention problems are evident at least 7–14 years following ALL treatment (Lockwood et al., 1999; Schatz et al., 2004), especially in females and in children treated with CRT at a younger age (Lockwood et al., 1999; Schatz et al., 2004).

Puppet and robot

Children with ALL treated with CRT, especially those radiated in early childhood, have difficulties with stimulus orienting (Lockwood et al., 1999). ALL participants treated with CRT demonstrate an exaggerated cue validity effect, suggesting disengage difficulties (Schatz et al., 2004). Compared to controls, children with ALL have slower but equally accurate focused attention (Mennes et al., 2005). Set shifting is compromised in children with ALL, even those with chemotherapy but no CRT (Buizer et al., 2005), although intensive chemotherapy (Buizer et al., 2005) and CRT (Lockwood et al., 1999) both exacerbate deficits.

Childhood survivors of ALL are slow but accurate when focusing attention (Lahteenmaki et al., 2001), especially if treated with methotrexate (Mennes et al., 2005). IOR and negative priming are yet to be studied.

Critic and actor

Divided attention, monitoring, and conflict have not been studied in ALL. Some studies report intact sustained attention in children with ALL (Mennes et al., 2005; Rodgers et al., 2003; but see Spiegler et al., 2006). However, Lockwood et al. (1999) found that children with ALL had poor sustained attention, especially if female, diagnosed at a younger age, treated with higher doses of methotrexate (Buizer et al., 2005), or treated with CRT at a younger age.

Children with ALL treated with methotrexate chemotherapy are slower and more variable than (but as accurate as) controls on response inhibition tasks (Mennes et al., 2005). CRT does not exacerbate inhibition problems (Lockwood et al., 1999). Children with ALL treated with chemotherapy differ from age norms in delayed response tasks (Spiegler et al., 2006). The sustain process appears intact in many, if not all children with ALL, and response inhibition (whether cancel or restrain/delay) processes are sometimes unimpaired.

NEUROBIOLOGY OF ATTENTION

The taxonomy is function-based, but we next demonstrate that: a) the elements of the taxonomy are compatible with the neurobiology of stimulus orienting and response control; and b) within each of the childhood attention disorders, functional deficits are present when the putative neural substrates are impaired and absent when they are intact.

Stimulus Orienting and the Posterior Cortex, Midbrain, and Superior Colliculus

Deficits in disengaging (not shifting attentional set) are associated with superior colliculus and posterior cortex lesions in adults (Posner et al., 1984). IOR deficits in adults are associated with superior colliculus lesions (Rafal et al., 1989, 1991; Sapir et al., 1999) and midbrain lesions such as those in progressive supranuclear palsy (Rafal & Grimm, 1981; Rafal et al., 1988).

SBM involves malformations of the cerebellum, hindbrain, and midbrain (Fletcher et al., 2005; Salman et al., 2006a). The midbrain abnormality, *tectal beaking* (Barkovich et al., 2005; Fletcher et al., 2005), is a mechanical consequence of brain development in a small posterior fossa (McLone & Knepper, 1989). Brain growth in SBM affects posterior more than anterior cortical structures, producing selective reduction in posterior cortical volume (Dennis et al., 1981; Fletcher et al., 1996a,b; Juranek et al., 2008) and impairments in white matter integrity (Hasan et al., 2008a), with relative preservation, even hypertrophy, of anterior cortex (Juranek et al., 2008) and some cortical and subcortical structures (Hasan et al., 2008b; Miller et al., 2008).

In SBM, deficits in disengaging and shifting are associated with tectal beaking and posterior brain volume loss (Dennis et al., 2005a), and attenuated IOR is associated with tectal beaking (Dennis et al., 2005b). Individual differences in stimulus orienting deficits are correlated with individual differences in structural brain damage observed in children with SBM in the posterior cortex, midbrain, and superior colliculus.

The midbrain is involved in dopamine dysfunction in children with P-ADHD. Ernst et al. (1999) found that accumulation of [F-18]DOPA, a measure of dopa decarboxylase activity in synaptic terminals of dopaminergic-rich regions, was nearly 50% higher in the right midbrain in children with ADHD, measures being correlated with symptom severity. However, this excess of midbrain dopamine does not disrupt stimulus orienting in P-ADHD.

We predict that structural or neurochemical damage to the midbrain should produce stimulus-orienting deficits at any age. For example, childhood acquired midbrain tumors, such as pineal germinomas, should be associated with impaired stimulus orienting.

Response Control and Frontal-Striatal Circuits

Response control develops throughout childhood (Davidson et al., 2006; Rubia et al., 2006) and involves two principal dopamine-modulated pathways: a prefrontal-dorsal striatum circuit and an orbitofrontal-ventral striatum circuit. Activation of the superior, medial, and inferior frontal gyri as well as midline networks are associated with withholding and monitoring motor responses (Chevrier et al., 2007; Rubia et al., 2001). The withdrawal or canceling of responses has been shown to activate the right inferior fron-

tal gyrus, the right anterior cingulate cortex (ACC), supplementary motor cortex, and caudate (Chevrier et al., 2007; Rubia et al., 2001). These data are congruent with the idea of distinct brain systems for intentional actions and the withholding of intended actions (Brass & Haggard, 2007). Frontal regions are also implicated in conflict management: Better interference control is associated with activity in the right middle frontal gyrus and left inferior frontal gyrus (Bunge et al., 2001).

There is ongoing debate about the neural basis of error detection and performance monitoring. The extensive limbic connections of the ACC contribute to the modulation of emotional expression (Critchley, 2005), and the dorsal ACC is directly involved in higher-order cognitive functions such as attention and response selection (e.g., Bush et al., 2000). Although the striatum has an important role as critic, the ACC also supports evaluative aspects of performance monitoring (Dias & Aggleton, 2000) that require integration of cognitive and limbic input. It is activated preceding errors (Li et al., 2007), responds to both internal and external error signals (Holroyd et al., 2004), and is more activated by error responses than by correct responses and by error feedback than by correct feedback (Holroyd et al., 2004). The dorsal ACC is particularly activated by error detection invoked by failed inhibition trials of the stop signal task (Chevrier et al., 2007), and by changes in task "sets" that signal adaptive changes in response style (Woodward et al., 2008). The role of the ACC in conflict monitoring may be domain-general because dorsal ACC activity is related to a cognitive conflict between major and minor musical modes (Mizuno & Sugishita, 2007).

Individuals with P-ADHD show selective cortical thinning of networks serving attention and executive control (Makris et al., 2007). Structural imaging of children with P-ADHD has identified volume reduction in the whole cerebrum (Castellanos et al., 2002), cortical gray matter, caudate nucleus, globus pallidus, prefrontal cortex, ACC (Castellanos et al., 1996; Filipek et al., 1997; Seidman et al., 2006), and especially, the right inferior frontal cortex (Durstun et al., 2004; Sowell et al., 2003), with relatively lesser involvement of the posterior cortex, midbrain, and cerebellum (Berquin et al., 1998). Various brain regions have been implicated in P-ADHD symptoms (Seidman et al., 2005), including dorsolateral prefrontal cortex and orbitofrontal cortex (Spencer et al., 2007), and dysfunction has been identified in dorsolateral and orbitofrontal cortices, ACC, inferior frontal gyri, and striatal-pallidal-thalamic circuits (e.g., Bush et al., 2005; Spencer et al., 2007).

Inhibitory control deficits associated with P-ADHD are correlated with functional alterations in a frontostriatal network. Poor interference control in children with P-ADHD is associated with decreased activation of the left inferior frontal gyrus (Vaidya et al., 2005), and hypoactivation of the dorsal ACC occurs in adults with P-ADHD (Bush et al., 1999). During inhibition trials, children with P-ADHD fail to activate the right inferior frontal or precentral gyri (Vaidya et al., 2005), exhibit abnormal activity in the dorsal ACC

(no activation, Durston et al., 2003, hypoactivation, Tamm et al., 2004), and exhibit striatal dysfunction, including decreased left caudate activity (Durston et al., 2003; Vaidya et al., 1998). Poor cancellation by P-ADHD patients is associated with hypoactive mesial prefrontal cortex (i.e., near the dorsal ACC) and ventrolateral prefrontal cortex, and decreased left caudate activity (Rubia et al., 1999).

TBI is frequently associated with dysfunction of the prefrontal and frontal cortex (Levin et al., 1996; Wilde et al., 2005), and corpus callosum and frontal lobe white matter (Levin et al., 2000), with some cerebellar involvement (Spanos et al., 2007). S-ADHD is associated with damage to neural regions known to be abnormal in P-ADHD, including the orbitofrontal gyrus (Max et al., 2005b), thalamus (Gerring et al., 2000), and basal ganglia (Gerring et al., 2000; Herskovits et al., 1999; Max et al., 2005a; and see Max et al., 2005c). Children with TBI/S-ADHD also exhibit response inhibition deficits, although there is no clear association with lesion site (Leblanc et al., 2005; Levin et al., 2004). ACC activity in adults with TBI is abnormal during conflict tasks (Soeda et al., 2005), possibly because of diffuse axonal damage disrupting frontal-cortical and subcortical networks, leading to reduced post-error slowing (Larson et al., 2007). In children with TBI, inhibitory impairments are correlated with prefrontal injury. Restraining a prepotent response on go/no-go tasks is related to volume of left prefrontal lesions (Levin et al., 1993, 2004), as well as to injury severity (Konrad et al., 2000a; Levin et al., 2004, but see Leblanc et al., 2005).

Children with P-ADHD and children with TBI have deficits in response control, with individual differences being correlated with individual differences in prefrontal-dorsal striatum and orbitofrontal-ventral striatum circuits. The caudate, putamen, and globus pallidus are part of a striatal-pallidal-thalamic circuit. In feedforward and feedback relations with the cortex, this circuit modulates inhibitory control and sensitivity to reward (Alexander et al., 1986), which is consistent with the idea that dopaminergic and/or noradrenergic medications affect ADHD by increasing inhibitory effects of frontal cortical activity on subcortical brain regions (Zametkin & Rapoport, 1987). The thalamus is part of two subcortical networks that are implicated in P-ADHD symptomatology as well as response inhibition (Gerring et al., 2000), and damage to the thalamus produces distractibility and disinhibition (Gentilini et al., 1987). To be sure, there is considerable heterogeneity in the brain systems associated with inhibition. Furthermore, the link of S-ADHD or P-ADHD to the thalamus is less clear than that of either of these disorders to the caudate, putamen, or inferior frontal gyrus.

CROSS-DISORDER COMPARISONS OF INATTENTION, IMPULSIVITY, AND HYPERACTIVITY

Inattentive and/or hyperactive-impulsive behaviors are features of P-ADHD, and exhibited by some children with TBI, SBM, and ALL. The association of inattention, impul-

sivity, and hyperactivity is strongest in P-ADHD. Between 65% and 90% of children with P-ADHD are hyperactive (Morgan et al., 1996; Paternite et al., 1995; Spencer et al., 2007; Wolraich et al., 1996), although the recession of hyperactivity with age may imply an age-dependent association. In TBI and S-ADHD, inattention and impulsivity, but not hyperactivity, are associated, in that most children with S-ADHD do not show hyperactivity, although they may exhibit inattention and impulsivity (Max et al., 2005a, 2005b, 2005c). Children with ALL exhibit some hyperactive symptoms, with approximately one-third of children with ALL achieving a mildly atypical score on the Conners' Parent Rating Scale-Hyperactivity Index (T-score > 60; Conklin, personal communication, November 26, 2007). Unlike those with P-ADHD or S-ADHD, children with SBM rarely show hyperactivity or impulsivity (Fletcher et al., 2005).

The cross-disorder comparison of inattentiveness, hyperactivity, and impulsivity prompts three testable predictions about stimulant medication treatment. Methylphenidate blocks the dopamine transporter (Dresel et al., 2000), which has a greater effect on the striatum than on the prefrontal cortex (Durston et al., 2005), given that the dopamine transporter gene is more highly expressed in the striatum than in the cortex, where other means of dealing with intrasynaptic dopamine predominate. The first prediction is that methylphenidate should affect response control more than stimulus orienting. This prediction is supported by the data reviewed in this article and also by evidence that methylphenidate specifically affects sustained attention and top-down control, rather than arousal (Johnson et al., 2008).

P-ADHD is a disorder of neurotransmission (synthesis, release, reuptake, effect) mediated by genes that code for dysfunctional or suboptimally functioning dopamine, whereas S-ADHD arises from damaged neural networks, which may or may not include striatal dysfunction. Given the different underlying mechanisms in the two conditions, the second prediction is that methylphenidate will be more effective in treating response control in P-ADHD than in S-ADHD. Methylphenidate also improves behavioral (not cognitive) hyperactivity/impulsiveness in S-ADHD, although improvement occurs acutely and is less robust than that in children with P-ADHD (Jin & Schachar, 2004).

Combined inattention and hyperactivity may reflect striatal and prefrontal-striatal dysfunction, whereas inattention may be primarily a problem of the prefrontal cortex and the prefrontal-parietal circuit (Diamond, 2005). This distinction, made within P-ADHD, prompts a third prediction across disorders, that treatment responsiveness should be positively correlated with hyperactivity and impulsiveness (specifically, should be highest in P-ADHD, moderate in S-ADHD, lower in ALL, and lowest in SBM). Stimulants have effectively treated P-ADHD for over more than three decades of randomized clinical trials, with 65% to 75% of individuals with P-ADHD being clinical responders, compared to 4% to 30% of individuals treated with placebo (Greenhill, 2002). Granted that a smaller proportion of children with S-ADHD (7%; Levin et al., 2007)

than P-ADHD (56%; Visser et al., 2007) receive stimulant treatment, methylphenidate does improve attention in children after TBI (Mahalick et al., 1998). Methylphenidate ameliorates attention problems in ALL (Conklin et al., 2007), and both moderate and low doses of methylphenidate improve parent and teacher attention ratings of ADHD symptoms, as well as teacher ratings of social-academic competence (Mulhern et al., 2004). Inattention in children with SBM does not respond robustly to methylphenidate treatment. In a large double blind, placebo-controlled trial of methylphenidate medication in a clinic population of children with SBM, Davidovitch et al. (1999) found no statistically significant medication effects (but see positive medication effects in a smaller, heterogeneous sample in Mayes et al., 1994).

INFERENCES AND PREDICTIONS

The application of the taxonomy to childhood disorders of attention has produced descriptive comparisons within and between disorders. New information has been added for each condition: for P-ADHD, the diversity of deficits in response control and the intactness of stimulus orienting; for SBM and ALL, the intactness of response control; and for S-ADHD, the similarity to P-ADHD with respect to deficits in response control and adaptive regulation. Individual children with P-ADHD are known to have uncorrelated deficits in multiple processes (e.g., delay intolerance is independent of inhibitory dyscontrol, Solanto et al., 2001; Sonuga-Barke et al., 1994), and we show this to be true for other childhood attention disorders. In addition, our data prompt inferences about theoretical questions and predictions about attention in disorders not yet studied.

Double Dissociation Between Automatic and Controlled Attention

Within- and between-disorder comparisons in childhood conditions have provided functional and neurobiological evidence of a double dissociation between automatic and controlled forms of attention. This is congruent with a long history of adult lesion studies, and also with new evidence from markers of event-related potentials. For example, listening to Mozart's D major sonata K.448 has opposite effects on event-related potentials markers of involuntary and voluntary attention (Zhu et al., 2008).

Inhibition Is a Diverse Attention Construct

Inhibition constitutes a diverse and often uncorrelated group of functions. In children with P-ADHD, the puppet is intact (normal IOR), but the actor is impaired (poor restrain). Different conditions have the same inhibitory impairment (P-ADHD and TBI both show impaired actor role and have restrain deficits). Adult disorders show differently dissociated forms of inhibition; for example, patients with

Parkinson's disease demonstrate intact IOR but impaired endogenously evoked inhibition (NP) (Grande et al., 2006).

Dissociations also exist within and between top-down and bottom-up processes. Children with P-ADHD are impaired on top-down response inhibition of the actor but not on endogenous top-down stimulus orienting of the robot. In children with SBM, bottom-up exogenous stimulus orienting deficits in the puppet are uncorrelated with intact top-down endogenous stimulus orienting in the robot.

Brain Injury Trumps Age at Onset

Anatomy trumps age at onset with respect to the puppet in stimulus orienting, in that SBM and ALL share deficits in this domain despite differences in age at onset, although stimulus-orienting deficits may have different anatomical origins in these two conditions. Anatomy certainly trumps age at onset for response control. Despite differences in age at onset, response control is largely unimpaired in SBM and ALL, and impaired in P-ADHD and TBI/S-ADHD, highlighting the role of anterior brain regions and the subcortical dopaminergic system in the production of response control deficits. Age at onset seems relevant to persistence of deficits in the two congenital conditions and the relative volatility over time of deficits in acquired conditions.

Although both P-ADHD and SBM are congenital conditions, they have distinct attentional and neurobiological profiles. Children with SBM have difficulties with error detection, although not with post-error adjustment. It is possible that they share error detection difficulties with children with P-ADHD because of shared midbrain dysfunction, perhaps related to mesolimbic dopamine (Di Chiara, 1998), or mesencephalic function more generally, affecting dorsal ACC and right frontal function. We predict some error detection deficits in children with other forms of structural or neurochemical midbrain dysfunction (e.g., children with pine tumors).

Temporal Stability of Attention Deficits

Both P-ADHD and SBM have attention deficits that persist over time and, at least for P-ADHD, into adult life. Their common pattern of stability of attention problems over time is consistent with their shared status as genetically based life-long developmental conditions with no period of normal development.

Compared to P-ADHD, children with TBI and S-ADHD have more volatile attention symptoms over time. Children with P-ADHD are evaluated in the school-aged years, and are already into the chronic stage of their attention disorder. It is unclear whether the picture of enhanced symptom volatility in children with TBI and S-ADHD arises because they have been studied in relatively short terms in relation to injury onset. Perhaps S-ADHD recovers over time, while formative brain anomalies ensure that P-ADHD will persist. However, one issue is that the time frame for resolution of cognitive deficits matches long-term TBI degenerative

processes (e.g., loss of axonal connections and secondary focal brain atrophy) better than the short-term recovery mechanisms known to be operative (e.g., reduction in inflammatory processes and brain swelling).

Based on our data, we predict that attention deficits, whatever their form, will be more stable over time in developmental conditions (e.g., Sotos syndrome) than in acquired conditions (e.g., childhood strokes).

Cognitive-Energetic Models, White Matter Damage, and Activating the Actor

In their cognitive-energetic model of attention, Russell et al. (2006) propose that P-ADHD involves inefficient astrocyte function from deficient ATP production in neurons, over milliseconds, and deficient myelination of axons during development, over months and years. Poor formation and supply of lactate produces inefficient and inconsistent performance in P-ADHD and other white matter disorders (Russell et al., 2006).

Although ALL treatment produces cortical atrophy (Baron et al., 1995), CRT and/or chemotherapy (Moore, 2005) particularly disrupt white matter, disturbances of which range from folate deficiency and subacute myeloencephalopathy to leukoencephalopathy with myelin degeneration and white matter necrosis (Maria et al., 1993). Antimetabolites such as methotrexate involved in ALL treatment particularly affect white matter (Cole & Kamen, 2006). SBM involves two types of white matter disruption, the first involving abnormal white matter development and the second, persistent white matter degeneration with increased age. Diffusion tensor tractography in children with SBM reveals abnormal development in the association pathways (e.g., poor visualization of tracts, impairment in myelination) as well as acquired abnormalities in intrinsic axonal characteristics (Hasan et al., 2008a).

The cognitive-energetic model of Russell and colleagues (2006) predicts increased response variability with compromised white matter, congenital or acquired. Children with SBM or ALL have little increased variability on sustained attention tasks despite impaired white matter, so our data provide no support for this prediction.

Temporal Processing and the Cerebellum

Temporal processing has been proposed as a key deficit in P-ADHD (e.g., Barkley et al., 1997), with a central importance being given to cerebellar abnormalities (Berquin et al., 1998). The cerebellar abnormalities in P-ADHD are minor compared to the massive cerebellar compromise in SBM, which is associated with abnormalities (in 98%) and volume reductions in the lateral cerebellum (Fletcher et al., 2005) and with upward and downward displacement of the cerebellar vermis (Salman et al., 2006a). Children with S-ADHD exhibit the typical P-ADHD attention profile, and are less likely than children with SBM to have cerebellar damage.

Perception of brief time intervals (around 400 ms) is consistently impaired in childhood cerebellar disease (SBM, Dennis et al., 2004), adolescents with cerebellar degenerative disease (ataxia-telangiectasia, Mostofsky et al., 2000), and adult survivors of childhood cerebellar tumors (Hetherington et al., 2000). Temporal estimation tasks, involving cognitive estimations of longer durations ranging from 4 minutes to a half hour, is intact in adult survivors of childhood cerebellar tumors (Hetherington et al., 2000). Children with P-ADHD cannot estimate long durations, around 4 sec, although they accurately perceive brief durations (Radonovich & Mostofsky, 2004), suggesting that the temporal impairment in P-ADHD involves failure to monitor temporal information in working memory rather than a cerebellar central timing deficit (Mahone et al., 2007).

Our data temper the view that the cerebellar temporal processing is of primary importance in P-ADHD. Not only is the degree of cerebellar damage unrelated to attention profiles, but children with P-ADHD have temporal estimation rather than timing deficits.

Can Puppets as Well as Actors be Candidate Endophenotypes?

Endophenotypes are hypothetical constructs intervening between genes and symptoms (Almasy & Blangero, 2001). Endophenotypes were originally proposed in the context of psychiatric disorders (Gottesman & Gould, 2003), but have more recently been used in neurodevelopmental disorders such as ADHD (Aron & Poldrack, 2005). Endophenotypes have more explanatory power than the cognitive phenotypes italicized in Figure 1, so we consider whether two of our cognitive phenotypes, response inhibition and IOR, might be considered as candidate endophenotypes. Crosbie et al. (2008) examined the theoretical rationale and *a priori* criteria for validating an endophenotype in P-ADHD and, with Biederman et al. (1995), propose that a candidate endophenotype should be common in affected individuals (sensitive), relatively unique to the disorder (specific), and relatively uncommon in the general population; they suggest, further, that a powerful way to establish endophenotype status would be to identify markers in cases with genetic, but not acquired, ADHD.

Response inhibition is a candidate endophenotype for P-ADHD (Aron & Poldrack, 2005; Crosbie et al., 2008). While response inhibition deficits are common in P-ADHD, but not in SBM or ALL, and are relatively uncommon in the general population, they also occur in S-ADHD, an acquired condition. A convincing non-genetic etiology for acquired conditions is difficult to demonstrate (Crosbie et al., 2008), and children with S-ADHD, considered as a group, have elevated rates of P-ADHD. Informative comparisons for invalidating response inhibition as a putative endophenotype would be a study of response inhibition in mild TBI, and a comparison of response inhibition deficits in children with TBI and a *de novo* S-ADHD diagnosis and those with TBI and preexisting P-ADHD.

Endophenotypes should be state-independent (i.e., should not vary with disease progression or treatment, Crosbie et al., 2008). Both acquired conditions, TBI and ALL, show cognitive phenotypes that fluctuate with disease characteristics, time since diagnosis, or treatment. Attention is worsened by severe injury in TBI and by adjuvant CRT treatment in ALL, in contrast to the relatively stable patterns of congenital conditions over the lifespan (in P-ADHD and, possibly, in SBM). Cognitive phenotypes in acquired conditions are perhaps less likely than those in congenital conditions to be endophenotypes marking genetic risk.

IOR may be a candidate endophenotype. Children of mothers with a genetic mutation in the folate metabolic pathway share the genetic mutation, have the upper spinal cord lesions (Volcik et al., 2000) associated with tectal beaking (Fletcher et al., 2005), and have IOR deficits primarily when they have tectal beaking (Dennis et al., 2005b). Children with ALL have a dual compromise of folate metabolism. Part of their treatment-related delayed neurotoxicity is a pharmacologic disruption of CNS folate physiology (Cole & Kamen, 2006), and they have a genotype involving a folate metabolic mutation linked to inattentive symptoms (Krull et al., 2008). While it is not clear that the identical mutations are involved (the C677T methylenetetrahydrofolate is the risk factor for upper spinal lesion level deficits in SBM, Volcik et al., 2000, the A1298C methylenetetrahydrofolate genotype is the predominant link to inattention in ALL, Krull et al., 2008), there is evidence for a link between folate mutations and inattention. More broadly, candidate endophenotypes for attention disorders might exist, not only in the actor and critic, but also in the puppet and robot.

CONCLUSIONS

Taxonomies are useful to the extent that they organize what is known and direct the search for the unknown. Our taxonomy describes attention within and across childhood attention disorders, is congruent with known neurobiology of attention, and predicts treatment responsiveness. It facilitates critical evaluation of some theoretical positions on attention and predicts specific attention deficits in clinical conditions not yet studied.

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