Neuropsychologic Theory and Findings in Attention-Deficit/Hyperactivity Disorder: The State of the Field and Salient Challenges for the Coming Decade

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The past decade has witnessed the establishment of several now well-replicated findings in the neuropsychology of attention-deficit/hyperactivity disorder (ADHD), which have been confirmed by meta-analyses. Progress has been notable from the importing of cognitive science and neuroscience paradigms. Yet these findings point to many neural networks being involved in the syndrome and to modest effect sizes suggesting that any one neuropsychologic deficit will not be able to explain the disorder. In this article, leading theories and key findings are briefly reviewed in four key domains: attention, executive functions, state regulation and motivation, and temporal information processing. Key issues facing the field of neuropsychologic research and theory in ADHD include 1) the need for more integrative developmental accounts that address both multiple neural systems and the socialization processes that assure their development; 2) consideration of multiple models/measures in the same study so as to examine relative contributions, within-group heterogeneity, and differential deficit; and 3) better integration of cognitive process models with affective and temperament theories so that early precursors to ADHD can be better understood. Overall, the field has witnessed notable progress as it converges on an understanding of ADHD in relation to disruption of a multicomponent self-regulatory system. The next era must articulate multipathway, multilevel developmental accounts of ADHD that incorporate neuropsychologic effects.

Key Words: Attention-deficit/hyperactivity disorder, executive functions, state regulation, reward response, temperament, neuropsychology, development

The past 2 decades have witnessed resurgent interest in neuropsychologic models of attention-deficit/hyperactivity disorder (ADHD), echoing conceptions and interests from earlier in the 20th century (Strauss and Lehtinen 1947). Contemporary approaches have been aided by increased sophistication in the understanding of brain behavior relations and of pharmacologic effects, as well as by newer behavioral and cognitive probes. Indeed, the infiltration and importation of measurement tools and constructs from the cognitive sciences, encouraged by Sergeant and Scholten (1985) and by Douglas (1972), continue (e.g., Castellanos and Tannock 2002; Nigg 2001; Schachar et al 1993). In the past decade, new models of attention, response suppression/inhibition, and motivation again have reshaped conceptions of ADHD. Increasingly, this syndrome is not seen as a disorder of attention at all but as a disorder in key aspects of self-regulation. "Self-regulation" as used here refers to both the effortful and, in some models, relatively automatized mechanisms that enable behavior to be adapted appropriately to a changing context. Evolving understanding in light of neural and cognitive discoveries continues, as illustrated more recently by growing interest in paradigms designed to probe cerebellar functions. Thus, a fresh look at the state of this field is as timely as ever.

The importance of clarifying such neuropsychologic deficits and, if justified, eventually including them in diagnostic criteria is considerable. As they stand now, the diagnostic criteria for ADHD are merely behavioral descriptors that inevitably overlap in several ways with a range of other psychopathologies. This causes problems with regard to clinical assessment and contributes to societal concerns about over-medicalization of behavior in ADHD (Searight and McLaren 1998) and disputes as to whether ADHD is a "valid" disorder. Indeed, from the viewpoint of disease theory, a valid disorder should feature a mechanism dysfunction in the child (Wakefield 1992); neuropsychologic weaknesses are increasingly seen as potentially representing that mechanism (Barkley 1997), regardless of what combination of etiologic factors led to the neuropsychologic difficulty.

Historically, numerous such functions have been considered, yet they converge on key neural networks. Neuroimaging studies so far have indicated involvement of processes involving subcortical–thalamocortical neural loops, along with cerebellar–frontal networks (Giedd et al 2001; Swanson and Castellanos 2002). These loops are influenced by catecholamine activity (dopamine and norepinephrine), although other neurotransmitters, notably γ-aminobutyric acid, are likely to prove equally important (Casey et al 2002; Sagvolden et al, in press). Other neural circuits might prove important as well. In this article, I will 1) highlight a handful of cross-cutting and to-date relatively intractable issues that concern the entire enterprise of neuropsychologic and cognitive research in ADHD, as well as other psychiatric conditions; 2) consider the status of the leading candidates for neuropsychologic dysfunction in ADHD; and 3) offer integrative comments.

Cross-Cutting Issues

Several issues cut across all neuropsychologic studies in ADHD; many of these issues are common to cognitive and physiologic measures in all of psychopathology. First, measures that detect robust between group-effects nonetheless have poor individual specificity and sensitivity in relation to the DSM-IV behavioral criteria for ADHD to date (Doyle et al 2000; Grodzinsky and Barkley 1999). To quantify the magnitude of group effects in this review, I refer to the effect size

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statistic *d*.[1] The group effects for ADHD versus healthy control subjects are generally modest in size, ranging from $d = .50$ to 1.0, suggesting a 30%–50% overlap of the ADHD and normal score distributions on most tasks (see Nigg et al 2005, for further discussion). The same pattern is apparent on neuroimaging (Swanson and Castellanos 2002), indicating that this is not merely a level-of-analysis issue. As noted, the same pattern holds with other forms of psychopathology, thus this issue is hardly unique to ADHD. Nonetheless, it is one that requires further work to enable specification of the clinical utility of neurocognitive probes and might in time contribute to refined operational definitions of the phenotype.

Second, as will be noted at several points in this review, heterogeneity remains under-investigated. One issue is the DSM-IV subtypes. Nearly all of the neuropsychologic literature on ADHD pertains to the group now designated as ADHD-combined type (ADHD-C). The primarily inattentive subtype of ADHD (ADHD-PI) remains relatively under-investigated with regard to potentially relevant cognitive functions (Huang-Pollock et al, in press; Milich et al 2001; Nigg et al 2002a). Thus, neuropsychologic process theories of ADHD-PI remain necessarily rather speculative compared with accounts of ADHD-C. The ADHD hyperactive subtype (ADHD-H) has yet to be seriously addressed as a separate subtype at all, with most neuropsychologic and cognitive studies either ignoring it (in part owing to its suspected rarity in school-age populations; Hart et al 1995) or lumping this group in with ADHD-C. Yet recent data from twin studies suggest that in the elementary school years, the etiologies for ADHD-C and ADHD-H are unlikely to converge (Willcutt et al 2000). Other sources of phenotypic heterogeneity include the comorbid behavioral and psychiatric profile (e.g., conduct disorder, anxiety disorder, major depressive disorder, or learning disorder; Jensen et al 1997), family history of ADHD (Seidman et al 1995), and gender-based presentation (Hinshaw et al 2002; Nigg et al 2002a). Furthermore, ADHD-PI includes both children who are active but shy of cutoffs for ADHD-C and children who are sluggish and hypoactive; these might represent etiologically distinct phenomena (Carlson and Mann 2002; McBurnett et al 2001). We now know that many ADHD-C deficits are not fully accounted for by comorbid learning and conduct disorders (Nigg 1999; Nigg et al 1998), but more work to clarify how neuropsychologic function varies with comorbid phenotype is needed. When I refer to heterogeneity later, I will be designating all of these issues unless otherwise noted.

Third, it remains to be demonstrated that neuropsychologic deficits are in fact causal in the development of ADHD. Demonstrating causality in human studies is, of course, quite difficult. Yet some testable hypotheses follow. One is the idea of the endophenotype, introduced to psychopathology research 3 decades ago by Gottesman and Shields (1972) and now much discussed (Almasy and Blangero 2001; Gottesman and Gould 2003). The idea is that a predisposing vulnerability or liability marker should 1) be correlated with ADHD symptoms or disorder in the probands; 2) help to identify more etiologically pure phenotypes for genetic and other etiologic research; 3) be familial; and 4) appear in unaffected relatives. A causal cognitive (or other) marker should appear in some unaffected relatives because it presumably combines with other factors in only some family members to cause the full disorder. Failing this test, the cognitive marker might only be another symptom of the disorder but not causal or at least not related to the genetic causal processes. This basic prediction often has failed in the few studies of ADHD to check it, although some effects were supported, with qualifications, in more recent work (Nigg et al 2004a; Slaats-Willemse et al 2003). A further sort of evidence on this front might be a demonstration of differential deficit for a particular neuropsychologic domain (i.e., that the deficit in ADHD is specific to one domain over and above others); however, attacking those methodologic issues (see Miller et al 1995) scarcely has been attempted in the ADHD field.

These last two issues underscore the likelihood that if particular neuropsychologic deficits contribute causally to ADHD, they probably do so only for some children, exemplifying the multiple causal and developmental pathways to this syndrome. Only recently have theories of ADHD formally included multiple pathways (Nigg et al 2004b; Sagvolden et al, in press; Sonuga-Barke 2002). As a result, too few studies have examined the various neurocognitive mechanisms together in the same sample, to evaluate whether children tend to have diffuse problems in all areas or whether there are discrete neurocognitive groups (see Nigg et al 2005). Along the same lines, theories of ADHD generally still lack more than a superficial account of how, developmentally, neuropsychologic difficulties lead to ADHD. For instance, theories typically have failed to fully integrate how family and peer relationship processes participate along with cognitive deficits in the development and maintenance of the syndrome (Johnston and Mash 2001; Olsen 2002; Sagvolden et al, in press). Evidence that such socialization effects are causally powerful apart from genetic influence in ADHD symptoms per se has been sparse, yet at the same time there is fairly extensive evidence that genetic effects might be mediated via socialization, owing to genotype–environment correlations. For example, Barkley and Cunningham (1979) showed that maternal negative/controlling parenting behaviors improved when children were medicated, suggesting that child difficult temperament might drive unfortunate socialization processes. How unfolding difficulties in child self-regulation—which must interact with socialization to develop at all—actually occur in the early development of ADHD and associated cognitive difficulties remains unclear. Such prospective developmental accounts will be crucial to integrating models involving temperament, neuropsychology, genetics, and developmental interpersonal processes (Nigg et al 2004b).

Update of Salient Neurocognitive Domains in ADHD

Selection of Domains and Studies for Review

This review is of necessity selective. Noteworthy publications on ADHD that I bypass here relate to the role of memory, motor control, fight–flight response, serotonergic function, sympathetic–parasympathetic peripheral nervous system responses as indices of central nervous system (CNS) function (see Beauchaine et al 2001), language processing, electroencephalogram/evoked response potential findings (see Barry et al 2003a, 2003b for

[1] *d* describes the magnitude of an observation in behavioral research. It is expressed in terms of the distance between two group means in standard deviation units. A $d = .50$ (half a standard deviation) is considered a medium-sized effect, which Cohen (1988) notes is approximately the same as the difference in height between 14- and 18-year-old girls (approximately 1 inch) or in intelligence quotient (IQ) between clerical/semi-skilled workers and professionals/managers (approximately 8 points). $d = .8$ is considered a large effect, which Cohen (1988) notes is equivalent to the difference in IQ between Ph.Ds and college freshmen or between college graduates and those with only a 50/50 chance of passing high school, or the average height difference between 13- and 18-year-old girls.

Table 1. Key Tasks Used in Neuropsychological and Cognitive Studies of ADHD

Task	Description
Continuous Performance Test	The Continuous Performance Test (CPT) has several varieties. Their common element concerns the ability to respond to a rare target over a period of extended time (15 min or longer). For example, the computer might show a different letter every 2 sec; however, when an "X" appears that was preceded by an "A" the child is to press the response button. The target will appear only on 25% or less of trials. Successful detection of a rare target amid many nontargets is an index of vigilance. Signal detection theory can be used to compute a parameter called d-prime (d'), which combines hits and misses to calculate sensitivity to the signal. One can also look at the relative weighing of commissions and omissions and calculate a parameter called beta, which signifies the response bias (e.g., tends to overrespond or underrespond).
Go/No-Go	The inverse of a CPT, one must withhold response on rare "no-go" trials. In a typical version of the task, randomly alternating stimuli are presented (e.g., an "A" and a "B" or two different visual designs). The child is instructed to make a response when they see the "A" but not when they see the "B". The "A" is presented more often to create a response set or prepotency toward responding. Errors in response to the "B" are taken as an index of failed inhibitory control. In the "event rate" version of this task, the rate at which stimuli are presented is varied (e.g., every 1 sec, every 4 sec, every 8 sec). The faster even rates are more "activating" for the child up to an optimum, then become too fast and lead to performance decline. Thus, in general, a child with ADHD is expected to approach normal performance more closely at the faster event rates. In "motivated" designs, more stimuli are used (e.g., several numbers), some of which are paired with a reward (if you press the key when you see the "A," you win 25 cents) and some with a response cost or punishment (if you press the key when you see a "B," you lose 25 cents). Various configurations of rewards and punishments are possible.
Stop Task	Presents equally probable stimuli (e.g., an "X" and an "O") with the instruction to press a corresponding key as quickly as possible, depending on which letter appears, creating a prepotent tendency to respond on most trials. On a minor ity of trials (25% typically), a signal (e.g., a tone) indicates that the child is not to respond. Timing of the tone is varied to estimate the speed of the "inhibition process" (essentially, how much warning does the child need to interrupt the response, which is mathematically independent of the speed of the response output process). Physiologic data indicate that a central (cognitive) process and a peripheral motor process are involved; responses can be interrupted even after peripheral nerves (on arm and hand muscles) have begun to fire (see Logan 1994). To measure inhibitory ability, older versions of the task calculated stop signal reaction time (SSRT) slope (degree of success drop-off at preset warning intervals). Newer versions use a dynamic tracking algorithm to directly estimate SSRT or warning time needed. The "go" trials of the task provide a strong measure of rapid decision-response time, and the variability of those response times is an index of response variability.
Antisaccade	Oculomotor task in which eye movements are monitored. On each trial, a signal appears in the visual periphery, creating a reflex response to move the eyes toward that signal. The reflex is difficult to resist. On some blocks of trials, children are told not to move their eyes toward the signal. Instead, they might be instructed to delay their response or to move their eyes away from the signal. Errors toward the signal, as well as presignal anticipations, are taken as indices of inhibitory ability or ability to suppress motor response.
Stroop task	This classic task has two or three conditions, depending on the design. The usual control condition is to name aloud as fast as possible the ink color of rows of x's (e.g., xxxx printed in red, green, and blue ink). Speed on this task is compared with speed on the interference task. In the latter, the child must name as fast as possible the ink color of a sequence of words, each of which is a color word different from the color of the ink (e.g., the word "red" printed in blue ink, the word "blue" printed in green ink). Because reading the word is a faster, more automatic process than naming the color, normal children and adults are slower to name the colors in the interference condition; the extent of this slowing versus the control condition is taken as an index of the effectiveness of an interference suppression mechanism. A range of related stimulus incompatibility tasks tap interference control without requiring reading.
Directed Forgetting	This task is widely used in cognitive psychology but as yet little investigated with ADHD. The child views a sequence of easily-named pictures and is then told to forget that list. They then view a second list, which they are told to remember. Recall of words to be remembered and words to be forgotten is then examined, across varying mixes of these conditions. In general, failure to recall a normal number of "remember" words or excess recall of "forget" words is taken as inhibitory failure.
Flanker Task	A type of selective attention task that can be designed to require perceptual or cognitive suppression of competing information. Similar to Stroop, except information is spatially distinct. For example, the child would view a target area in the center of a computer screen, with an instruction to press the corresponding key depending on whether the "X" or "N" appears in the center. Immediately adjacent to the center letter are two "flanking" distractor letters that are to be ignored. The flankers can be incompatible (X or N) or neutral (e.g., F or D). It takes longer to respond to XNX than to FNF because in the first instance the flanker is a possible response that must be suppressed.
Negative Priming	An important variant of selective attention measures, this task has several versions, all of which exploit the principle that when two stimuli appear that differ on the task-relevant property (e.g., presented with a red car and a blue house, name the red object), the unchosen object property tends to act as though it has been suppressed, in that on the next trial it would take longer to name the red house, because the house had been suppressed on the prior trial, versus if the prior trial had not included a house. Failure to show this normal delay to name the previously "cued-wrong" object is taken by some as evidence of failure in an attentional inhibitory process.

reviews), and response distribution properties in children with ADHD (Leth-Steenson et al 2000). I instead update the status of four key domains: attention, executive functions, state regulation and motivation, and processing of temporal information. Even then, however, the literature is sufficiently large that this review is highly selective (an initial *Psych Info* literature search gener-

Table 1. Continued.

Task	Description
Spatial Orienting	Often called the "Posner orienting task" or the "covert orienting task," the child fixes their eyes on the center of the computer screen, with an instruction to press the key as quickly as possible when they see the target appear in either the left or the right periphery. The target is preceded by a warning cue that is either correct or incorrect in its spatial (left or right) visual field location. The degree to which reaction time to the target is slowed down by the incorrect warning cue is sometimes interpreted as an index of inhibitory control, although that reaction time slowing could also be due to failure of several possible attentional mechanisms other than inhibition.
Trailmaking	From the Halstead Reitan Battery and other batteries; in Trails "A," the child traces a line to a series of letters scattered randomly on the page (A-B-C-D, etc.). In Trails "B," the child must alternate letter-number-letter, thus tracing A-1-B-2-C-3, etc. The difference between B and A time is viewed as an index of set shifting ability, a type of executive functioning.
Tower	Several tower tasks exist, including the Tower of London, the Tower of Hanoi, the Stockings of Cambridge, and others. The idea in all of them is that discs or balls must be moved around on pegs (either manually or on a computer screen) according to certain rules, to arrive at a predetermined arrangement. The task required visualizing the moves in advance and can be designed to place heavy loads on visual working memory and sequencing.
Spatial Span	A spatial span task will typically ask a child to remember the sequence of a series of shapes or locations. For example, the Finger-Windows test asks the child to touch a pencil to a series of locations in the correct order on a paper after seeing the locations touched by the examiner. Many variations exist either on the computer or with paper and pencil.
Wisconsin Card Sort	The Wisconsin Card Sort is a classic "executive function" measure. The child must match a series of cards to a target card; cards include varying numbers of shapes, varying shapes, and varying colors. Thus, the child must decide whether to sort by color, by number, or by shape. After 10 consecutive correct matches, the sorting rule changes, but the child is not told of the change. Thus, they must notice that the old rule is no longer working, determine the new rule, and again this continues for 10 correct matches, up to a total of six categories (in the full-length version of the test). The test requires working memory, abstraction, and set shifting abilities and activates prefrontal cortex.
Fluency	Several versions of "fluency" tasks exist. They all share the property of asking the child to think of as many words as possible in a limited time period, meeting a certain rule. For example, they may be asked to name as many animals, or as many words starting with the letter "C" as possible in a 30-sec period.
Mazes	The Porteus Mazes and related tasks ask a child to draw a line rapidly and accurately, showing the way out of a visual maze on a sheet of paper. The task requires mental planning as well as good motor control.

ADHD, attention-deficit/hyperactivity disorder.

ated 626 citations from 1990 to 2004 potentially relevant to this review). I therefore highlight relatively well-established or important findings (emphasizing meta-analytic results where possible) and overarching issues and questions that remain. Table 1 provides a description of the measurement tasks most frequently used in this literature, including those mentioned either in Table 2 or throughout this text. Key findings of eight available meta-analyses of neuropsychologic function in ADHD are summarized in Table 2 and referred to en route. One recent meta-analysis (Hervey et al 2004) is excluded because it focused on adults; however, those results were similar to the ranges shown in Table 2. Figures 1A and 1B provide a schematic portrayal of portions of the key neuroanatomy and neurochemical pathways discussed under several cognitive domains.

Attention

For present purposes, the broad universe of attention theories, models, and measures can be simplified by considering that, in the visual domain, attention must be directed to a location in space (spatial orienting). Then, object selection must occur. Both orienting and selection involve 1) stimulus-driven, bottom-up, relatively automatic processes; and 2) goal-driven, relatively effortful, and controlled processes. These two types of processes work in tandem to direct attention, but experimentally they can be dissociated by a variety of paradigms. Distributed neural models of spatial orienting (Posner and Petersen 1990) sparked renewed interest in orienting in ADHD in the period from 1991 to the present. Work in this area was kicked off by a finding of an interesting pattern of lateralized orienting deficits by Swanson et al (1991). In the interim, more than a dozen studies reported diverse and interesting results; however, a recent meta-analysis (Huang-Pollock and Nigg 2003; Table 2) revealed that no consistent effects emerged across these studies. Contrary to speculation that ADHD might fit a right-lateralized or neglect profile, there were no consistent lateral engage or disengage effects. Thus, spatial orienting is not a promising core deficit, at least in ADHD-C.

Despite early interest in selection, selection was thought to be intact in ADHD by the end of the 1980s (Sergeant and van der Meere 1990). Yet questions continue about revisiting attention selection in ADHD (Douglas 1999), which might be warranted in particular as cognitive science introduces newer, integrative models of selection processes (Lavie and Tsal 1994). Initial studies suggest, however, that ADHD-C is associated with normal selection, even on newer load-dependent paradigms (Huang-Pollock et al, in press). These paradigms vary the perceptual "load" (e.g., number or difficulty of items to be processed in a rapid-decision task) to differentiate early, relatively automatic or perceptual selection mechanisms that mature during early childhood and are mediated by parietal–subcortical neural circuits from late-stage, relatively effortful or cognitive selection mechanisms that mature later in adolescence and are mediated by frontal–subcortical neural circuits (Haung-Pollock et al 2002). Yet, it might be that children with sluggish cognitive tempo have abnormal perceptual (early, posterior) selection mechanisms (Huang-Pollock et al, in press). In all, primary attention deficits are not likely as an explanation of ADHD-C but remain underinvestigated in ADHD-PI, particularly in children who are hypoactive.

Executive Functions

Background. Executive functioning in the neuropsychologic literature refers to maintenance of behavior on a goal set over

time and complex organizing of behavior (Lyon and Krasnegor 1996; Pennington, 1997). The construct is still criticized for conceptual underspecification, although tractable models are beginning to emerge (Lyon and Krasnegor 1996; Zelazo et al 2003). These more tractable models have been able to operationalize components of cognitive control, such as detecting a mismatch from expectations, interrupting a response, shifting a response, conflict detection, sustaining working memory via control of mental interference, inhibition of competing responses, and regulation of response via alertness or allocation of effort (Botvinick et al 2001; Posner and DiGirolamo 1998). Similarly, it is now clear that clinical executive function tasks also capture multiple-component operations (Miyake et al 2000) variously labeled as set shifting, interference control, inhibition, planning, and working memory and others (Pennington and Ozonoff 1996), though these are not always operationalized as narrowly as they are in cognitive designs. These different mechanisms might be related to distinct parallel thalamo–cortical–basal ganglia neural loops, which are modulated by dopamine (Figure 1A). The result is that, in addition to an extensive body of literature on classic clinical executive function tasks (many of which are described in Table 1), chronometric tasks borrowed from cognitive psychology have now had a substantial trial with ADHD samples (again, see Table 1 for some examples, such as the stop task).

ADHD Effects. Pennington and Ozonoff (1996) (Table 1) identified several deficits associated with ADHD in executive function tasks that seemed to be related to response inhibition/suppression and planning (see Table 2). They concluded that these effects were relatively specific to ADHD, in that no studies of conduct disorder (CD) showed these effects when ADHD was controlled. Subsequent research has not overturned those conclusions, although as is often the cases when literatures mature, effect sizes (such as *d*) are not as large as they once seemed to be (Table 2; Willcutt et al 2005); however the emphasis has shifted since the mid-1990s. First, work has continued apace to measure efficiency of response suppression with the chronometric Logan Stop Task (Logan and Cowan 1984; Table 1). Recent work indicates that the ability to interrupt an about-to-be-executed response requires activation of the right inferior frontal cortex (Aron et al 2003), as well as regions in basal ganglia, including the caudate (Casey et al 2002). Oosterlaan et al (1998) reported a meta-analysis of the first eight studies of this task. They noted an ADHD deficit in stop signal reaction time (SSRT) (average $d = .64$, Table 1) and smaller deficits in response variability and go reaction time. They noted that similar though smaller SSRT effects were observed in CD; however, up to that time, few studies of ADHD had controlled for CD symptoms (or vice versa) to truly evaluate whether ADHD effects survived such control.

Nigg et al (1998), however, found that a composite executive measure was associated with ADHD symptoms independently of CD symptoms, but the reverse was not true (but note that Seguin et al [1999] did find executive deficits in aggressive boys after controlling ADHD). Nigg (1999) reported a similar finding using the stop signal task: ADHD deficits held with CD symptoms controlled. These specificity findings were confirmed by Willcutt et al (2001) for executive tasks generally and by Schachar et al (2000) for the stop task. Thus, in a review of nearly 20 stop task studies along with other measures of response inhibition, such as the antisaccade task and the go/no-go task, Nigg (2001) concluded that this ability was associated with ADHD (see Willcutt et al 2005, for a further update on this task and its effect sizes across some 25 studies).

Yet, the field is far from settling on response suppression as the main executive function problem in ADHD. In particular, interest has accelerated regarding the role of visual working memory in ADHD, on the basis of early findings of large problems on tasks such as the Tower of Hanoi (Barkley 1997; Pennington and Ozonoff 1996) and now supported by additional studies that attempt to parse components of the working memory system, which includes dorsolateral prefrontal cortex and other cortical structures (Table 1; Martinussen et al, in press). The number of studies used to estimate these effects remains rather modest, and further work to clarify the specificity of this finding is under way.

Summary. Several well-established group deficits in executive functioning now exist and provide considerable explanatory appeal. Difficulties in response suppression and visual working memory are most noteworthy. In addition, newer, potentially developmentally relevant conceptions of executive functioning (Diamond et al 1997; Zelazo et al 2003) have yet to fully infiltrate the ADHD literature. Nonetheless, many findings are sufficiently well described that further studies of children with ADHD-C versus control children on many executive measures might no longer be needed. Instead, studies to examine such issues as comorbidity and specificity, gender differences, subtype differences, reliability of deficits over time, and crucially, relations among mechanisms (e.g., response inhibition and working memory, or these operations and state regulation [see the next section]) should become the norm. In short, executive functioning might be the most well-developed arena of research in ADHD, yet it now faces quite difficult questions and a need for shift in focus before it can be decisively advanced.

State Regulation: Arousal, Activation, and Motivation

Background. The classic temperament conception of arousal (variously defined historically by Pavlov and colleagues; for a review see Strelau 1994) eventually was divided into two functions but in different ways by temperament and cognitive researchers. Temperament models went on to distinguish between 1) arousal (phasic signal detection efficiency based in corticoreticular loops); and 2) activation, as reward responsivity or affective motivational response based in limbic reactivity (Gray 1982). Cognitive models instead distinguished arousal and activation according to somewhat different definitions that de-emphasize motivation and affective response, as detailed in the next two paragraphs. As a result, one thread of ADHD theory now draws on cognitive traditions and emphasizes demand–response preparation generally; a second thread emphasizes reward and reinforcement responsivity. Here, the focus first will be on the influence of the cognitive tradition on ADHD research.

Arousal and ADHD. Arousal in most classic cognitive models refers to a function that involves right-lateralized noradrenergic neurons ascending from the locus coeruleus to the cortex (Figure 1B). Functionally it entails alerting, phasic responding, and enhancing signal/noise ratio in attention (Pribram and McGuinness 1975; Tucker and Williamson 1984; again see Strelau 1994). It thus is closely related to the concept of alerting (Posner and Peterson 1990) and is most relevant to the early stages of information processing (Sergeant et al 1999; Tucker and Williamson 1984). Conceptions of this type of under-arousal in cortical or other CNS systems have long appealed to ADHD theorists (Satterfield et al 1974; Zentall and Zentall 1983) for their ability to

Table 2. Selected Meta-analytic Findings in Neuropsychology of ADHD Versus Non-ADHD Children

Measure	Effect Size (*d*)
Spatial Working Memory (Spatial Span)	.75[a] to .85[b] to 1.14[b]
Response Suppression (Stop Task SSRT/SSRT Slope)	.61[a] to .64[c] to .94[d]
Signal Detection (CPT d-prime) Arousal	.72[e]
Stroop Naming Speed	.69[f]
Full Scale IQ	.61[g]
Set Shifting (Trails B Time)	.55[a] to .59[g] to 0.75[d]
Planning (Tower of London/Hanoi)	.51[a] to .69[a]
Mazes	.58[a]
Verbal Working Memory	.51[a] to .41[b]
Decision Speed on Go-Task	.49[c]
WCST Perseverations	.35[g]/.36[a] to .53[h]
Fluency	.27[d]
Stroop Interference	.25[f]
Covert Visual Spatial Orienting	.20[i]

See Table 1 for task descriptions. The effect size "*d*" indicates the standard deviation unit difference in group means; it is computed as m1 -m2/ mean SD. ADHD, attention-deficit/hyperactivity disorder; SSRT, stop signal reaction time; CPT, continuous performance task; IQ, intelligence quotient; WCST, Wisconsin Card Sort Test.

[a]Willcutt et al 2005 (2005).

[b]Martinussen, in press (verbal working memory [WM] storage $d = .4$, verbal WM executive $d = .54$, visual WM storage $d = .85$, visual WM central executive $d = 1.14$).

[c]Oosterlaan et al 1998.

[d]Pennington and Ozonoff 1996.

[e]Losier et al 1996.

[f]van Mourik et al, in press.

[g]Frazier et al 2004.

[h]Romine et al 2004.

[i]Huang-Pollock and Nigg 2003.

explain a range of behavioral and physiologic observations. Recent theories have been similar but more closely related to psychopharmacology, again emphasizing disruption in the ascending noradrenergic neurons that support signal/noise detection (McCracken 1991; Figure 1B). Sergeant et al (1999), drawing on Pribram and McGuinness (1975), suggested that responses early in the task are particularly informative. Thus, appearance of reaction time or accuracy deficits early on tasks (e.g., commonly observed slow and variable reaction time on initial task trials; Oosterlaan et al 1998) is one source of support for an arousal model. Early and recent electroencephalogram and evoked response potential findings tend to support this model as well (Barry et al 2003a, 2003b), in that they reveal excess slow-wave activity in children with ADHD.

Closely related to this conception of arousal is the ability to alert rapidly to novel stimuli. That ability is also theorized to depend on a right-lateralized vigilance network with noradrenergic involvement (Posner and Petersen 1990). Data on alerting therefore also might support an under-arousal model or enable it to be reformulated. Some data suggest such alerting is deficient in children with ADHD and their biologic but not adoptive relatives (Nigg et al 1997), but two other studies found smaller effects on this probe (reviewed by Huang-Pollock and Nigg 2003).

Perhaps the best performance support for an arousal deficit in ADHD comes from consistent findings of deficit on the continuous performance test *d*-prime parameter, a consensus index of arousal as defined in the theories cited here (Losier et al 1996; see Tables 1 and 2). Arousal thus is a quite viable candidate for an ADHD neurocognitive deficit. Questions remain, however, owing to lack of time on task data in many published cognitive studies (Sergeant et al 1999).

Activation and ADHD. In the cognitive tradition, activation was historically viewed as a left-lateralized process involving dopaminergic neurons in response to motor preparation for response output (Pribram and McGuinness 1975). Whereas arousal is related to early-stage information processing, activation is related to response preparation and readiness to respond. It is a tonic rather than a phasic process. To some extent, activation is related to the concept of sustained attention (Posner and Peterson 1990), except that the emphasis is on sustained readiness of motor preparation (Pribram and McGuinness 1975; Tucker and Williamson 1984).

Confusion therefore often ensues regarding the relationship between activation, vigilance, and sustained attention. Historically, these concepts have had a parallel handling and have filled similar roles in information processing theories, even though they are not isomorphic. Thus what Posner and Petersen (1990) call "vigilance" is also often referred to as "sustained attention" (Mirsky and Duncan 2001). It refers to the ability to maintain a

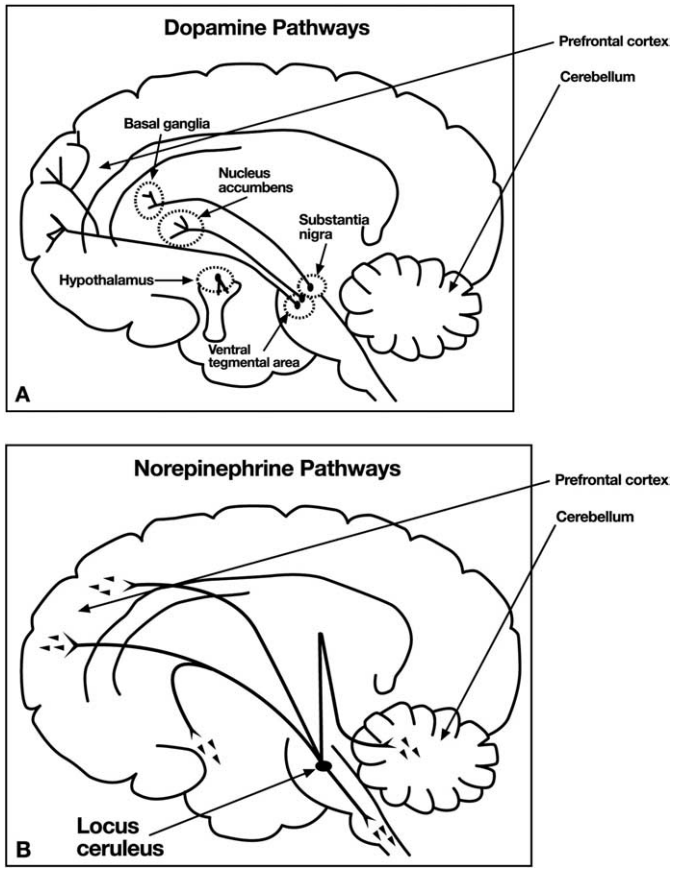

Figure 1. (A) Schematic view of subcortical and cortical regions, with dopamine pathways highlighted. The mesocortical pathway running from substantial nigra on to prefrontal cortex is important in executive functioning as well as in motivation and emotion. The mesolimbic pathway, projecting to nucleus accumbens, is often identified with reward motivation and reinforcement learning. The nigrostiatal pathway projecting to basal ganglia is often identified with motor control. **(B)** Schematic view of noradrenergic projections to prefrontal cortex and cerebellum. Adapted and reproduced with permission from http://www.mastersofpediatrics.com/cme/cme2003/lecture5_2.asp.

tonic state of alertness and wakefulness during prolonged and sustained mental activity (Weinberg and Harper 1993). Performance that deteriorates over the course of minutes or hours (i.e., slower reaction time, greater variability of reaction time, and increased errors) is the "vigilance decrement" (Parasuraman et al 1998).

Whereas both Pribram and McGuinness (1975) and Tucker and Williamson (1984) emphasized the motor relevance of activation and conceptualized it as a left-lateralized, dopaminergically mediated process, Posner and Peterson (1990) argued that phasic arousal and vigilance (their version of activation) both depend on the same network of neural structures as arousal, namely the noradrenergic system of the locus coeruleus (again, Figure 1B), the cholinergic system of the basal forebrain, the intralaminar thalamic nuclei, and the right prefrontal cortex (Derryberry and Rothbart 1997; Parasuraman et al 1998). The reason for this difference in emphasis again is that Posner and Peterson emphasized the attentional rather than response preparation function of vigilance. Construct validation of the distinction between these conceptions remains ongoing.

In the case of research on ADHD, researchers in The Netherlands have emphasized the Pribram and McGuinness (1975) version of activation in influential theories about ADHD (Sergeant et al 1999; van der Meere 2002).[2] Following that approach, Sergeant et al (1999) argued that activation is associated with response output, longer time on task performance, and what are known as "event rate" effects. Event rate effects means that when event rates speed up or slow down appropriately, for example the rate at which trials occur on a continuous performance test (CPT) or a go/no-go task (Table 1), performance of children with ADHD can worsen or significantly improve (van der Meere and Stermerdink 1999). Performance in children with ADHD might even equal that of healthy children under other event rates—that is, the optimal event rate for some tasks might simply be different for children with ADHD than for children without ADHD. These data might support an under-activation explanation of many apparent executive or other findings (e.g., inhibition on go/no-go tasks) as well as of sustained attention effects (Sergeant et al 1999; van der Meere 2002). Output response speed data are also supportive, in that children with ADHD have slow sustained motor responding (Carte et al 1996). Yet other predictions from an activation theory, notably deficits on the CPT β parameter, a key probe (Table 1), have not been supported in ADHD (Losier et al 1996; Table 2), raising problems for an activation account. In all, activation remains a viable candidate deficit in ADHD but is supported better by event rate data than by response bias data.

Reward Response and ADHD. The reward response deficit idea has remained viable, if historically under-emphasized, in the ADHD literature. It is regaining currency in modified form today thanks to new insights from animal models and deeper understanding of dopamine circuitry and pharmacologic action in ADHD (Solanto et al 2001). As noted, Gray (1982) outlined a behavioral "activation" or "approach" system that activates motor response to signals for reward ("conditioned appetitive stimuli") and active avoidance behavior in response to cues for nonreward or punishment. He suggested neural mediation by the ascending dopaminergic fibers in the reward or appetitive system of the brain (Figure 1A). These fibers proceed from the substantia nigra and ventral tegmentum to the basal ganglia (especially the caudate nucleus), limbic system, lateral hypothalamus, and prefrontal cortex.

Following up on this model, Gorenstein and Newman (1980) proposed that an over-responsive approach (reward cue response) system leads to difficulty inhibiting response in the presence of strong reward cues for children with ADHD. Sole difficulty in an overactive reward system would lead to over-responding only in the presence of reward cues. Difficulty in consistently isolating over-responding by children with ADHD to such a relevant incentive context has been a key obstacle (Iaboni et al 1995; Oosterlaan and Sergeant 1998). Other data suggest that children with ADHD are over-responsive to recent or immediate reward but under-responsive to more time-distal contingencies (Tripp and Alsop 1999).

Consistent with the latter conclusion, under-response to contingencies has been reinterpreted recently in light of animal data by researchers in Norway. These more recent conceptions emphasize reinforcement response, in particular the tendency to under-respond unless reinforcers are temporally relatively immediate. The operative concept in these more recent models involves a dysfunction in the tonic or phasic properties of the modulatory dopaminergic response to reinforcers (or reward cues) in the mesolimbic/mesocortical dopamine systems (Johansen et al 2002; see Figure 1A). This dysfunction leads to an abnormally steep delay–reward gradient, such that rewards become abnormally low in reinforcing power as they become distant in time (Sagvolden et al 2005). The Oslo group also has suggested that a secondary dysfunction in the nigrostriatal dopamine system (projecting from the substantia nigra to the basal ganglia; Figure 1A) might account for motor control problems (including slow and variable motor responses; Johansen et al 2002). The delay–reward gradient argument rested heavily on basic work in animal cognition with an inbred rodent model (the spontaneously hypertensive rat). Although difficulties have been noted in this particular animal model (Ferguson 2001), the conceptual model has substantial appeal for ADHD. Initial data on learning and extinction in children with ADHD seem to be supportive (Sagvolden et al, in press).

A Word on Reactive Behavioral Inhibition and ADHD. In addition to specifying a reward–incentive approach system, Gray (1982) described a behavioral inhibition system. His conception is similar, for present purposes, to the description by Kagan et al (1987) of behavioral inhibition in young children. These theorists identify interruption of behavior due to events that are unfamiliar or unexpected (novel, unexpected, or signal potential loss of reward or punishment). This construct was long conflated with strategic interruption of behavior (often itself referred to as behavioral inhibition). The conceptual benefits of distinguishing strategic interruption of a prepotent (i.e., prepared, cued, or most likely) response, as an element of executive control, from reactive or anxious interruption of response to scrutinize a stimulus, are now clear, as detailed elsewhere (Nigg 2001). Once this distinction is made, evidence for reactive inhibitory problems in ADHD is sparse in behavioral data. The best evaluation of this system likely will come from physiologic studies. They are just beginning to emerge and should clarify whether reactive inhibition is impaired in a subgroup. I (Nigg 2001) have argued that it is likely to be impaired in cases with comorbid conduct disorder or aggression but not otherwise.

Summary. As a group, these state and motivation theories are quite powerful in accounting for a range of ADHD symptomatology and phenomenology. Yet a number of issues remain.

[2] Sergeant et al (1999), like Pribram and McGuinness (1975), also included an effort pool in their model; effort might be closely related to motivation (see van der Meere 2002; Sergeant 2005) and so is not discussed separately here. See Sergeant's article in this issue of *Biological Psychiatry* for more discussion.

First, the arousal and activation models rely on relatively old neural theories. For example, recent work has attempted, although still without consensus, to specify multiple arousal generators in the brain (Robinson 2001). Updated models based on more recent neural models of affective and regulatory response eventually will be needed. Second, many of the operational definitions of measures of arousal versus activation or effort continue to lack clear consensus, although progress on that front is evident. Yet the same data still can be interpreted in different ways by theorists of competing persuasions. Consensus on operational definitions of these constructs, preferably anchored in current neuroimaging data and recent neural models, will be an important advance. Third, the delay–reward gradient/reinforcement model, reliant on animal research, now requires additional validation and replication in children, including independent replications of extinction learning difficulties in children with ADHD. It has benefited, however, from recently being linked with theories of excess delay aversion in children with ADHD (Sonuga-Barke 2002). Fourth, consideration of heterogeneity and subtyping is only beginning to be published (Clarke et al 2001). Clarifying to which children with ADHD these models pertain will be a crucial advance. In all, the state regulation and motivation models are essential to understanding the neuropsychology and phenomenology of ADHD but face key hurdles to providing a more definitive explanation. Forthcoming advances in measurement, theory, and replication should solidify their contribution.

Temporal Information Processing and Motor Control

Researchers have long believed that impulsivity was related to problems in time perception and/or timing (see Parker and Bagby 1997 for historical review). Renewed interest in this concept as a contributor to ADHD (Barkley 1997; Castellanos and Tannock 2002; Sonuga-Barke et al 1998; Toplak et al 2003) has been sparked by neuroimaging findings of cerebellar abnormality in ADHD (Giedd et al 2001; Swanson and Castellanos 2002). This interest has converged with an evolving neuroscientific understanding of the cerebellum, which is now believed to involve not only motor control and timing but by extension temporal information in cognition and executive functioning (Diamond 2000). An overarching theme has been the integrated concept of processing of temporal information as a key element in behavioral response, dependent on intact cerebellar function. Key measurement indices here include time estimation and reproduction (on the basis of theories that the internal clock requires cerebellar–frontal involvement), as well as motor timing tasks. ADHD deficits are beginning to be replicated in tests of time estimation, time duration, and motor timing (Barkley et al 2001; Smith et al 2002; Toplak et al 2003), using a variety of task designs. Although this body of literature is still young (e.g., poor control of comorbidity, small samples, unstandardized methods), problems in processing temporal information might well emerge as a feature in ADHD.

If so, the key issue here will be the conceptual integration of temporal information processing with the other neurocognitive domains believed to be involved in ADHD. Are timing functions integrally related to executive functioning (see Fuster 1997; Barkley 1997)? Alternatively, do they reflect secondary problems due to poor arousal or state regulation (as might be indicated, for example, by more variable behavior on time estimation motor tasks)? Or are they best thought of as a separate process that informs other cognitive functions, or perhaps as a simpler way to reframe a variety of problems in alerting, learning, and sequencing? Theoretic and empirical specification of these relations in an integrated model will be crucial for next-generation theories of ADHD (see Castellanos and Tannock 2002).

Summary

Numerous neuropsychologic correlates of ADHD are now relatively well established. They highlight that several neural and psychologic processes might be involved in ADHD and that any one process theory (i.e., a theory attempting to explain ADHD as due to breakdown in a particular psychologic or neurocognitive mechanism or process) has difficulty explaining all findings. What now requires resolution is their relative contribution, capacity to survive tests of differential deficit, and conceptual relation to one another. Nonetheless, as a result of these gains, in the past decade or so neuropsychologic theories of ADHD have grown more sophisticated, well specified, and integrated with advances in cognitive science and animal pharmacology research. The main stream of neuropsychology research in ADHD is pursuing key domains important to self-regulation and mediated by frontal–subcortical catecholamine networks in the brain, notably specific components of executive functioning (or cognitive control), state regulation, and incentive response. Each of these subdomains has spawned integrative theories, which themselves will eventually need integrating. In the meantime, they have guided the field to more experimentally sophisticated and theory-driven research.

Relationships Among Processes and Models: A Developmental Perspective

As just mentioned, an overarching issue in this entire body of literature concerns the relationships among these processes from a developmental perspective. How do these varying processes relate to one another, and how do they emerge and influence one another in children's development? Conceptually and neurally, the processes of executive control, state regulation and response readiness, and reward response are closely interrelated in the control of real behavior. All point to related neural systems in basal ganglia, limbic system, thalamus, and prefrontal cortex (Barkley 1997; Gray and McNaughton 1996), as well as, in some instances, cerebellar–cortical circuits. Dynamically, in real-world behavior, the relationship is reciprocal between executive functioning and activation or arousal, as well as between these and motivational processes, such as reward response. Measurement technologies by which to capture the rapid neural dynamics at issue here have yet to emerge, so that at one level the problem of primacy in dynamic activation is not yet tractable. Yet the field is needlessly limited by the tendency of studies to examine only one model at a time in any real way. Relatively few studies have attempted to measure these multiple processes in the same sample (Brandeis et al 1998; Oosterlaan and Sergeant 1998; Scheres et al 2001; Solanto et al 2001). Furthermore, statistical modeling techniques, such as latent variable and confirmatory factor models, might be more regularly applied to evaluate the relationships among the measures, constructs, and levels of analysis at issue across the interrelated theories that have been described herein.

In addition to learning how measures of these constructs perform when studied together and how they are related to one another structurally, it also will be of value to understand in what cases, and to what extent, each process is contributing to the behavioral dysregulation observed. That goal mandates examination of individual differences in ADHD (Nigg et al 2005).

Cutting across these integrative concerns is the question of developmental sequences of these systems' influence on behav-

ior. How do early development precursors lead to the breakdown of executive processes? Which system breaks down first? How would that be measured? Although no theory yet offers a unique developmental account of how ADHD emerges, the bases for such accounts are emerging (Calkins and Fox 2002; Posner and Rothbart 2000). These normal developmental accounts tend to emphasize emotional regulation and motivation rather than state regulation or cognitive processes. That emphasis is understandable given that in infants and toddlers, emotion and affect are domains more easily assessed than cognition; however, understanding the precursive links between these early temperament measures and later cognitive and self-regulation processes is needed.

Thus, the emergence of self-regulation in early development, through mutually influencing processes of emotional reactivity and emotion regulation, effortful control, arousal reactivity, and language skills, unfolds in a somewhat predictable sequence (Calkins and Fox 2002; Rothbart and Bates 1998; see Nigg and Huang-Pollock 2003). In early infancy, attention, behavior, and affect are largely reflex driven. Later in infancy, however, they begin to be regulated by emotional reactivity or the strength of negative and positive emotions in relation to immediate incentives (a potential reward or a distressing signal). By the beginning of the toddler period (early in the 2nd year), children begin to use effortful redirection of attention to assist with the regulation of affect states (e.g., turning their attention away from upsetting events to calm down). This effortful redirection of attention continues to develop rapidly in the 2nd through 4th years of life; by approximately 30 months of age, children can begin to inhibit stimulus-driven motor response (e.g., reaching for an attractive item as soon as they notice it; Diamond et al 1997; Posner and Rothbart 2000) and thus begin to be evaluated with laboratory measures of attention and impulse control similar to those used in childhood to study ADHD, such as interference control and stopping tasks (Diamond et al 1997). Diamond et al (1997) documented a steady development in the ability to inhibit competing, stimulus-driven motor responses by ages 3, 4, and 5 years (see also Zelazo et al 2003). Effortful control in the form of strategic attentional and impulse control continues to develop throughout childhood, yielding steady progress in the ability to ignore competing stimulus-driven responses during the 2nd to 4th grades (Huang-Pollock et al 2002) and the ability to inhibit primary responses during ages 5–7 years and older (Carver et al 2001). The ability to suppress prepotent responses continues to develop through adolescence (Bedard et al 2002), presumably aided by ongoing myelination and pruning of frontal cortical neural networks (Benes 2001) and socialization and learning.

Arousal and alerting processes, of course, interact with the effortful and reactive processes just highlighted. Strong reactive responses (e.g., intense anxiety, excitement, or anger) influence arousal levels (Gray 1982), with a cascade of associated physiologic responses. Moreover, executive control contributes to regulation of arousal, and optimal arousal is necessary for effective effortful control (Barkley 1997; Derryberry and Rothbart 1997). Finally, also essential to the early regulatory development are verbal learning and language problems (Bodrova and Leong, 2003; Nigg and Huang-Pollock 2003), which suggests that language functions might warrant more scrutiny in ADHD (Barkley 1997; Tannock and Schachar 1996).

Thus, integration of insights from cognitive neuroscience with those from affective neuroscience will be essential to identifying early precursors of ADHD as distinct from precursors to antisociality or externalizing behavior generally. One means of such integration will be by consideration of how measures of temperament might map onto neuropsychologic measures. As should be clear, such mapping is not as simple as matching up the terminologies in these studies; however, conceptual linkages nonetheless can be made (Nigg et al 2004) and might suggest means of subtyping ADHD. For example, ADHD seems to be linked with personality traits, such as low conscientiousness (related to planfulness and temperamental effortful control), as well as with high hostility (underscoring links between ADHD and oppositional/aggressive behavior, as well as between ADHD and negative affect; Nigg et al 2002b).

Conclusions

Neuropsychologic studies of ADHD have generated several now well-established findings in ADHD-C. These findings have been used by theorists in models that emphasize particular components of a unified self-regulatory system, which emerges from the infant to toddler years, consolidates from the preschool to early childhood years, and continues to mature through adolescence. The self-regulatory capabilities of the child depend on the mutually supportive interplay of 1) strength of the affective response to incentive, which eventually implicates reinforcement response and thus socialization; 2) state regulation, including arousal and arousal reactivity; 3) effortful control or the ability to strategically redirect attention or suppress responses to regulate affect and behavior—a likely precursor to the abilities identified as executive functions in childhood; and 4) emerging language capabilities. Conceptually, theorists increasingly have begun to propose dynamic as well as developmental accounts of how these domains "fit together" in the emergence of ADHD (e.g., Barkley 1997; Sagvolden et al, in press). Empirically, we now know that the performance deficits of children with ADHD on neuropsychologic tasks reach across these several conceptual neural systems. It remains unclear whether this is owing to diffuse injury to the system as a whole or whether focal breakdown in one domain leads to secondary problems in other domains. Still other domains remain actively under investigation, including processing of temporal information.

Striking at this stage of the field's evolution are that 1) several effects are relatively well established and might not need further replications; 2) effect sizes are modest, raising serious questions as to the ability of any one neuropsychologic hypothesis to fully account for ADHD; and 3) the diverse domains implicated in ADHD continue to require more refined conceptual integration. Recent models have taken significant steps toward such integration (Barkley 1997; Douglas 1999; Sagvolden et al, in press; Sergeant et al 1999), yet that conceptual effort is far from complete or satisfying to the field as yet.

The next generation of studies in the neuropsychology of ADHD are encouraged to attack the following key challenges: 1) measuring multiple, competing, hypothesized processes together in the same samples; 2) evaluating heterogeneity within ADHD-C and within ADHD-I samples, including not only comorbidity and gender differences but also variability in neuropsychologic response; 3) continuing to update conceptual models in line with emerging models in the neurosciences; and 4) providing more detailed developmental accounts that can integrate the early precursors of ADHD in the realm of emotion and affect regulation and effortful control with childhood data on such cognitive functions as state regulation, executive functioning, and motivation.

Such developmental accounts must, yet to date have not,

more deeply address the interplay of socialization and interpersonal process in early childhood along with development of the self-regulatory abilities assessed in neuropsychologic studies. Such integrated hypotheses—and accompanying data—will make an important contribution when they appear. The neglect of this integration might in part be due to a misguided belief that because ADHD is highly heritable, socialization processes do not require intensive study. This belief is misguided because heritable effects are likely to be mediated at least in substantial part by socialization, through genotype–environment correlations or other mechanisms.

Overall, support is converging for understanding ADHD-C from a neuropsychologic perspective as often related to atypical development in cognitive control operations along the frontal–striatal networks that involve dopaminergic and noradrenergic innervation. Characterizing these problems in terms of cognitive or neuropsychologic endophenotypes now features some combination of "top-down" executive control processes (e.g., suppressing competing responses) and "bottom-up" motivation or regulation processes (e.g., arousal, activation, or delay–reward gradient). It is unlikely that a single-process theory can account for either the range of phenomena to be explained in groups of children with ADHD or for within-group variation in ADHD expression. Whether these competing models can be integrated into a consensus, parsimonious two-process model that explains at least definable subgroups of children with ADHD remains to be seen. Doing so in a convincing fashion will require integration with broader developmental conceptions of how self-regulation consolidates in context. Attempting to address these challenges likely will occupy the ADHD theory and research community for some time. Along the way, new subtypes, anchored in measures of cognitive and affective response, should become clear, along with new insights into the developmental etiology of ADHD in its various manifestations.

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