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Attention-deficit disorder (attention-deficit/hyperactivity disorder without hyperactivity): A neurobiologically and behaviorally distinct disorder from attention-deficit/hyperactivity disorder (with hyperactivity)

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Abstract
Most studies of attention-deficit/hyperactivity disorder (ADHD) have focused on the combined type and emphasized a core problem in response inhibition. It is proposed here that the core problem in the truly inattentive type of ADHD (not simply the subthreshold combined type) is in working memory. It is further proposed that laboratory measures, such as complex-span and dual-task dichotic listening tasks, can detect this. Children with the truly inattentive type of ADHD, rather than being distractible, may instead be easily bored, their problem being more in motivation (underarousal) than in inhibitory control. Much converging evidence points to a primary disturbance in the striatum (a frontal–striatal loop) in the combined type of ADHD. It is proposed here that the primary disturbance in truly inattentive-type ADHD (ADD) is in the cortex (a frontal–parietal loop). Finally, it is posited that these are not two different types of ADHD, but two different disorders with different cognitive and behavioral profiles, different patterns of comorbidities, different responses to medication, and different underlying neurobiologies.

I join the growing chorus of those who argue that attention-deficit/hyperactivity disorder (ADHD) of the "truly" inattentive subtype (what I will call "attention-deficit disorder" [ADD]) is a different disorder from ADHD where hyperactivity is present (e.g., Barkley, 2001; Cantwell, 1983; Carlson, 1986; Carlson & Mann, 2000; Goodyear & Hynd, 1992; Hynd, Lorys, Semrud–Clikeman, Nieves, Huettner, & Lahey, 1991; in particular see the outstanding paper by Milich, Balentine, & Lynam, 2001). Not only is "ADHD without hyperactivity" (ADHD of the predominantly inattentive type) an awkward locution, but it also tries to squeeze ADD into a box in which it does not belong. The term ADHD should be reserved for when hyperactivity is present (as the term implies), regardless of whether inattention is also present.

The points I make in this paper include the following: many individuals currently diagnosed with the inattentive subtype of ADHD appear to be misdiagnosed. ADD appears to be an instance of childhood-onset "dysexecu-

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tive syndrome." ADD and ADHD are characterized by dissociable cognitive and behavioral profiles, different patterns of comorbidities, different responses to medication, and different underlying neurobiological problems. The core cognitive deficit of ADD is in working memory. Contrary to what many have claimed (that laboratory tests cannot capture the core cognitive deficits in ADD), I argue that complex-span and dual-task dichotic listening tasks can indeed capture them. The working memory deficit in many children with ADD is accompanied by markedly slowed reaction times, a characteristic that covaries with poorer working memory in general. Individuals with ADD are not so much distractible as easily bored and underaroused. I hypothesize that the *DAT1* gene will be found to be more closely linked to ADHD than to ADD, whereas the *DRD4* gene will be found to be more closely linked to ADD than to ADHD, and that the primary neural circuit affected in ADHD is frontal–striatal, whereas the primary neural circuit affected in ADD is frontal–parietal.

The current *DSM-IV* (American Psychiatric Association [APA], 1994) diagnostic guidelines list three subtypes of ADHD: (a) primarily inattentive, (b) primarily hyperactive and impulsive, or (c) both combined. ADHD conceived in this manner is by far the most commonly diagnosed psychological/behavioral disorder of childhood (e.g., Barkley, DuPaul, & McMurray, 1990; Szatmari, 1992; Weiss & Hechtman, 1979).

Individuals with ADHD of the inattentive subtype tend to be disorganized, easily pulled off course, forgetful, and inattentive (*DSM-IV*; APA, 1994). They tend to be disorganized mentally and physically. They tend to make careless mistakes, and are not good at paying close attention to detail. They have difficulty organizing their work, setting priorities, planning out a strategy, and remembering to do all required tasks. They have difficulty organizing their things and tend to be sloppy. They have trouble keeping track of their belongings and forget where they have put them, in part because they rarely put things away. If multiple items are needed for an assignment or task, they will typically forget one or more. They have trouble keeping track of multiple things held in mind, which can make arithmetic calculation, reading, or abstract problem solving difficult.

Individuals with ADHD of the inattentive type also tend to have a hard time sustaining focused attention on a task or activity. They are quite poor at following through on something to completion. They tend to get bored with a task fairly quickly and often abandon a task unfinished, bouncing from one partially begun project to another. They may have a hard time keeping their mind on any one thing at a time. When doing homework or reading, they often find their minds wandering. Because focusing deliberate, conscious attention on completing a task is so arduous or aversive for individuals with the inattentive subtype of ADHD, they tend to try to avoid beginning a task, procrastinate, may forget to write an assignment down, forget to bring home the materials needed to complete an assignment, or lose materials needed for an assignment.

In 1986 Baddeley coined the term dysexecutive syndrome to refer to adults who seem to have a deficient "central executive" and who thus appear to be disorganized, easily pulled off course, forgetful, and inattentive. As far as I know, dysexecutive syndrome has always been used with reference to adults. I would like to suggest that children with ADHD of the inattentive subtype provide an instance of the dysexecutive syndrome in children. Dysexecutive syndrome patients may go off on tangents or lose their train of thought. Individual skills, such as encoding an item into memory or retrieving an item from memory, are intact. However, dysexecutive patients "have problems in initiating [a chore], monitoring their performance, and . . using such information to adjust their behavior. As most tests concentrate on the building blocks or component skills and are less concerned with the integration of these skills into real-life tasks, many [dysexecutive] patients . . . perform adequately on frontal lobe tasks . . . In contrast, many everyday activities involving executive abilities require patients to organize, or plan their behavior over longer time periods or to set priorities in the face of two or more competing tasks" (Wilson, Evans, Emslie, Alderman, & Burgess, 1998, p. 214). It is on such everyday activities that the dysexecutive def-

icit is most evident. Dysexecutive patients often start out performing a task well, but quickly become sidetracked. It is hard for them to stay focused on the task at hand, and they commonly must be reminded of what it was they were supposed to be doing.

The *DSM-IV* cutoffs for the inattentive, hyperactive, and combined subtypes of ADHD were derived largely from research with young males, who are more prone to hyperactivity and impulsivity than are girls or older males or females. Hence, some individuals get miscategorized as inattentive-type ADHD, despite being hyperactive for their gender or age, because they are not significantly more hyperactive or impulsive than young boys (e.g., Carlson & Mann, 2002; deHaas & Young, 1984; Milich et al., 2001; Weiss, Worling, & Wasdell, 2003). Such individuals should be considered the combined type. In this article, I focus on individuals with ADD (those who meet the criteria for inattentive-type ADHD and who are not hyperactive, excluding those with significant hyperactivity even if subthreshold for a combined-type diagnosis according to current *DSM* criteria). There is considerable overlap between what I am calling "ADD" and what others have called "slow cognitive tempo" (SCT; e.g., Milich et al., 2001), but SCT includes additional features that characterize only a subset of children with ADD. I reserve use of the term ADHD for ADHD that includes prominent hyperactivity (which for all practical purposes means combined-type ADHD because predominantly hyperactive ADHD is so rare after the age of 6).

Children with combined-type ADHD have many of the above symptoms, but they also have great difficulty sitting still (APA, 1994). They are overactive (motor and verbal), restless, and always on the go. They are overly talkative, fidgety, and squirmy. They often do repetitive motions like wiggling their feet or tapping their pencil. They get up when remaining seated is expected. They can talk incessantly and have difficulty playing quietly.

They also tend to be impulsive (APA, 1994) and are inclined to be very disorganized and sloppy, because they are often too impatient to carefully attend to detail or to put things away. They can have trouble waiting their turn, may blurt out an answer before hearing the whole question, and may interrupt others. They may intrude on others' conversation or game, without considering beforehand that it might be inappropriate. Because they tend to act impulsively, they may run into the street without looking or grab a toy from another child.

ADD and ADHD That Includes Hyperactivity Are Dissociable Disorders

Whereas children with ADHD are frenetic and hyperactive, a significant proportion of children with ADD are exactly the opposite. A significant subset of children with ADD are hypoactive, sluggish, and very slow to respond (see Table 1). Children with ADHD are often insufficiently self-conscious; children with ADD tend to be overly self-conscious. Both groups tend to have social problems, but for different reasons. An ADHD child is likely to have social problems because he/she alienates other children by butting in, taking their things, failing to wait his or her turn, and in general, acting without having first considered the feelings of others (e.g., Lahey, Schaughency, Hynd, Carlson, & Niever, 1987). On the other hand, a child with ADD is likely to have social problems because of being too passive, shy, or withdrawn (e.g., Goodyear & Hynd, 1992; Hinshaw, 2002; Maedgen & Carlson, 2000). His or her quietness or slowness to respond may be misinterpreted by others as aloofness, disinterest, or unresponsiveness. Children with ADHD tend to be extroverted; children with ADD do not.

Because of their disruptive behavior, children with ADHD are more likely to be suspended or expelled from school (Weiss et al., 2003). Conduct disorder and aggressivity are far more commonly comorbid with ADHD than with ADD (e.g., Barkley et al., 1990; Barkley, DuPaul, & McMurray, 1991; Faraone, Biederman, Wever, & Russell, 1998; Edelbrock, Costello, & Kessler, 1984; Goodyear & Hynd, 1992; Lahey et al., 1987; Morgan, Hynd, Riccio, & Hall, 1996; Nigg, 2000; Weiss et al., 2003). Conversely, children with ADD are somewhat more prone to internalizing disorders such as anxiety or depression (or at least show a marked absence of externalizing disorders) and tend to be more socially isolated

Table 1. *A comparison of the characteristics of ADHD and ADD*

ADHD With Hyperactivity[a]	ADD[b]
Hyperactive, always on the go, impulsive	A significant subset are hypoactive and sluggish and have slow response speeds.
Primary deficit in response inhibition	Primary deficit in working memory, especially prominent in auditory processing because of the demands it places on working memory
Often insufficiently self-conscious	Tend to be overely self-conscious
Social problems because too assertive and impulsive: butt in, take things belonging to others, fail to wait their turn, and act without first considering the feelings of others	Social problems because too passive, shy, or withdrawn
Tend to be extroverted	More likely to be introverted
Externalizing behaviors, such as conduct disorder, aggressivity, disruptive behavior, and even oppositional defiant disorder are far more commonly comorbid with ADHD than with ADD.	Internalizing disorders, such as anxiety or depression, are somewhat more common in children with ADD than those with ADHD. ADD children tend to be socially isolated or withdrawn. Reading and language deficits and problems with mental mathematical calculations are more commonly comorbid with ADD than with ADHD.
Respond positively to methylphenidate (Ritalin)	A significant percentage are not helped by methylphenidate.
Most respond positively to methylphenidate in moderate to high doses.	Those who are helped by methylphenidate often do best at low doses.
Those with ADHD are more likely to smoke than are those with ADD. (There are marked similarities in the neurobiological and psychological effects of nicotine and methylphenidate.)	A significant subset are helped by amphetamines rather than methylphenidate. Amphetamines affect both the reuptake and release of catecholamines. A marked deficit in the release of DA and NE might cause sluggishness and underarousal.
Methylphenidate adresses catecholamine reuptake. Addressing reuptake appears to be sufficient to help individuals with ADHD.	People with ADD are not so much easily distracted as easily bored. Their problem lies more in motivation than in inhibition.
	Challenge or risk, something to literally get their adrenaline pumping, can be key to keeping their attention and to eliciting optimum performance. Individuals with ADD, although typically shy, may engage in risk-taking and thrill-seeking activities as ways to experience a level of engagement they have difficulty sustaining in their daily lives.
Converging evidence for a primary disturbance in the striatum	A primary disturbance in prefrontal cortex is implicated.
The primary neural circuit that is affected may be a frontal–striatal one.	The primary neural circuit that is affected may be a frontal–parietal one.
Polymorphisms in the *DAT1* gene are associated with ADHD. This is consistent with the centrality of the striatum in ADHD because DAT plays a particularly important role there. It is also consistent with the efficacy of methylphenidate because DAT is the primary target for the clinical action of methylphenidate.	The 7-repeat allele polymorphism of the *DRD4* gene is more strongly linked to ADD than to ADHD. This is consistent with the centrality of prefrontal cortex in ADD because the D4 DA receptor is present in prefrontal cortex but not in the striatum in humans.

[a]Children with combined-type ADHD or hyperactive-type ADHD.
[b]Children with inattentive-type ADHD, excluding those with significant hyperactivity, even if they fail to meet the criteria on seven hyperactivity items (they are really combined-type ADHD).

or withdrawn than are children with ADHD (Barkley et al., 1990, 1991; Faraone, Biederman, Wever, & Russell, 1998; Edelbrock, Costello, & Kessler, 1984; Goodyear & Hynd, 1992; Lahey et al., 1987; Morgan, Hynd, Riccio, & Hall, 1996; Nigg, 2000; Weiss et al., 2003). Reading and language deficits are more commonly comorbid with ADD than with

ADHD (Faraone et al., 1998; Warner–Rogers, Taylor, Taylor, & Sandberg, 2000; Weiss et al., 2003; Willcutt & Pennington, 2000) as are problems with mental mathematical calculations (Carlson, Lahey, & Neeper, 1986; Hynd et al., 1991; Marshall, Hynd, Handwerk, & Hall, 1997; Morgan et al., 1996).

Most children with ADHD (perhaps as high as 90%; Barkley, 2001; Barkley et al., 1991; Milich et al., 2001; Weiss et al., 2003) respond positively to methylphenidate (Ritalin) and over two-thirds of such children respond positively to methylphenidate in moderate to high doses (Barkley, 2001; Barkley et al., 1991; Milich et al., 2001; Weiss et al., 2003). In contrast, a significant percentage of children with ADD are not helped by methylphenidate and those who are helped often do best at low doses (Barkley, 2001; Barkley et al., 1991; Milich et al., 2001; Weiss et al., 2003). Many individuals with ADD are helped by amphetamines, such as Adderall. There is considerable overlap in the mechanisms of action of methylphenidate and amphetamines, but there is a significant difference. Although both methylphenidate and amphetamines inhibit reuptake of dopamine and norepinephrine, only amphetamines also promote *release* of those neurotransmitters. Recent research also suggests that low doses of methylphenidate (the dosages likely to be efficacious in treating ADD) preferentially release norepinephrine in the rat brain (Ishimatsu, Kidani, Tsuda, & Akasu, 2002). Possible problems with the neural release of norepinephrine in ADD are relevant to motivational issues discussed later.

There is also some evidence for differential responsivity to nicotine. There are marked similarities in the neurobiological and psychological effects of nicotine and methylphenidate (e.g., Pomerleau, 1997). It has been hypothesized that individuals with ADHD who are not taking stimulant medication may try to self-medicate by smoking. Certainly, unmedicated adolescents with ADHD smoke far more than do their medicated ADHD peers and their non-ADHD peers (Whalen, Jamner, Henker, Gehricke, & King, 2003). Krause, Dresel, Krause, la Fougere, and Ackenheil (2003) report that individuals with ADHD are far more likely to smoke than are individuals with ADD: "It was striking how many of the 20- to 40-year-old patients in our group, who had shown symptoms of hyperactivity and impulsivity in childhood, were smokers: nine smoked and only three were non-smokers . . . The opposite was shown in the patients with only inattentive symptoms throughout their whole life: only two smoked, seven were non-smokers" (pp. 610–611; although note that Tercyak, Lerman, & Audrain, 2002, report the opposite).

Filipek, Semrud–Clikeman, Steingard, Renshaw, Kennedy, and Biederman (1997) report that individuals with ADHD who respond favorably to stimulant medication have the smallest caudate nuclei. There is now converging evidence for a primary disturbance in the striatum in children with ADHD. Both structural and functional neuroimaging studies report striatal abnormalities in children with ADHD. Structural magnetic resonance imaging studies consistently find smaller caudate volumes and reversed caudate asymmetry in those with ADHD (Aylward, Reiss, Reader, Singer, Brown, & Denckla, 1996; Castellanos, Elia, Kruesi, Gulotta, Mefford, Potter, Ritchie, & Rapoport, 1994; Castellanos et al., 1996; Filipek et al., 1997; Hynd, Hern, Novey, Eliopulos, Marshall, Gonzalez, & Voeller, 1993; Mataro, Garcia–Sanchez, Junque, Estevez–Gonzalez, & Pujol, 1997; Schrimsher, Billingsley, Jackson, & Moore, 2002), although the laterality of the differences and direction of left–right asymmetry have not always been consistent across studies. Functional neuroimaging studies report less striatal activity in ADHD children while they are performing response–inhibition tasks compared to age-matched controls (Durston, Tottenham, Thomas, Davidson, Eigsti, Yang, Ulug, & Casey, 2003; Lou, Hendriksen, & Bruhn, 1984; Lou, Hendriksen, Bruhn, Borner, & Nielsen, 1989; Teicher, Ito, Glod, & Barber, 1996; Vaidya, Austin, Kirkorian, Ridlehuber, Desmond, Glover, & Gabrieli, 1998; Zametkin, Liebenauer, Fitzgerald, King, Minkunas, Herscovitch, Yamada, & Cohen, 1993). Hyperactivity is more typically found after structural damage to the striatum than after structural damage to frontal cortex. Motor hyperactivity is not a prominent characteristic of frontal patients, although an impulsive, manic type of activity (such as marked verbosity) can sometimes be

seen in frontal patients. In contrast, patients with Parkinson disease (where the primary disturbance is in the striatum) can show a kind of motor restlessness (called "akathisia"; Lang & Johnson, 1987).

Dopamine transporter (DAT) is the principal mechanism for reuptake of released dopamine. DAT is abundant in the striatum (Garris & Wightman 1994), where it is widely distributed and strategically located (Sesack, Hawrylak, Matus, Guido, & Levey, 1998). It is far less abundant and less well situated in the prefrontal cortex (Sanchez–Gonzalez & Cavada, 2003; Sesack et al., 1998). Hence, it plays a more important role in striatal function than in prefrontal function. DAT is the product of the *DAT1* gene. Several studies report that commonly found polymorphisms in the *DAT1* locus are associated with ADHD (Barr, Wigg, Bloom, Schachar, Tannock, Roberts, Malone, & Kennedy, 2000; Cook, Stein, Krasowski, Cox, Olkon, Kieffer, & Leventhal, 1995; Daly, Hawi, Fitzgerald, & Gill, 1999; Gill, Daly, Heron, Hawl, & Fitzgerald, 1997; Swanson et al., 2000; Waldman, Rowe, Abramowitz, Kozel, Mohr, Sherman, Cleveland, Sanders, Gard, & Stever, 1998). In a meta-analysis of 11 family-based studies, Cook (2000) found the association between the *DAT1* gene and ADHD to be highly significant ($p < .0001$). It is important that levels of hyperactive–impulsive symptoms are correlated with the number of *DAT1* high-risk alleles but levels of inattentive symptoms are not (Waldman et al., 1998). A role for polymorphisms of the *DAT1* gene in ADHD is consistent with the centrality of the striatum in ADHD because DAT plays a particularly important role in the striatum. It is also consistent with the efficacy of methylphenidate in treating ADHD, because methylphenidate acts directly on DAT function (Dresel, Krause, LaFougere, Brinkbaumer, Kung, Hahn, & Tatsch, 2000; Seeman & Madras, 1998; Shenker, 1992; Volkow, Gur, Wang, Fowler, Moberg, Ding, Hitzemann, Smith, & Logan, 1998). Further, there is evidence that nicotine may act directly on DAT in a way similar to that of methylphenidate (Krause et al., 2003; Krause, Dresel, Krause, Kung, & Tatsch, 2000; Krause, Dresel, Krause, Kung, Tatsch, & Ackenheil, 2002). Indeed, DAT binding specifically in the striatum has been found to be related to motor hyperactivity but not to inattentive symptoms (Jucaite, Fernell, Halldin, Forssberg, & Farde, 2005).

If the striatum is potentially the primary site for neurobiological dysfunction in ADHD, what is the primary site for dysfunction in ADD? There has been far less research on ADD, especially excluding individuals misdiagnosed as having ADD rather than subthreshold combined-type ADHD. However, the neurobiological, cognitive, and behavioral profile of children with ADD strongly implicates a primary disturbance in prefrontal cortex. Whereas polymorphisms in the *DAT1* gene are hypothesized to be more strongly linked to ADHD than ADD, the 7-repeat allele polymorphism of the *DRD4* gene is more strongly linked to ADD then to ADHD (Rowe, Stever, Giedinghagen, Gard, Cleveland, Terris, Mohr, Sherman, Abramowitz, & Waldman, 1998). The dopamine receptor subtype, DRD4, is present in prefrontal cortex in humans, but not in the striatum (Meador–Woodruff, Damask, Wang, Haroutunian, Davis, & Watson, 1996). Hence, a link with the *DRD4* gene implicates prefrontal, rather than striatal, involvement. In the human prefrontal cortex, mRNAs for the dopamine receptor subtypes (DRD1 and DRD4) are the most abundant, although the other dopamine receptor subtypes are present. In the human striatum, in contrast, receptors are abundant for, and limited to, D1, D2, and D3 (Meador–Woodruff et al., 1996). Similarly, in the rhesus monkey, DRD4 is densely localized to prefrontal cortex and the hippocampus, with significantly lower levels in the striatum (De La Garza & Madras, 2000). Although DRD4 and DRD5 expression is noticeably higher in the cortex than the striatum of the rhesus brain, levels of DRD1 and DRD2 mRNAs are noticeably higher in the striatum than in the cortex. Consistent with an association between DRD4 polymorphism and ADD is Auerbach, Benjamin, Faroy, Geller, and Ebstein's finding (2001) of a significant relation between individual differences in sustained attention and working memory on the one hand and polymorphism of the DRD4 gene on the other hand in normal infants (those with the 7-repeat allele performing worse). Also con-

sistent with this is that *DAT1* gene expression has been found to preferentially affect caudate volume, whereas *DRD4* gene expression preferentially affects prefrontal gray matter volume (Durston et al., 2005). However, the finding that ADHD children with the DRD4 7-repeat allele required higher doses of methylphenidate is inconsistent with this (Hamarman, Fossella, Ulger, Brimacombe, & Dermody, 2004).

No brain region functions in isolation. The striatum has close links with prefrontal cortex and there is considerable evidence that a disturbance in frontal–striatal circuitry is found in ADHD (e.g., Casey et al., 1997; Castellanos, 1997; Hale, Hariri, & McCracken, 2000; Heilman, Voeller, & Nadeau, 1991). Brain regions participate in more than one circuit. The patterns of deficits often seen in ADD (such as problems with math calculation, language, and working memory, and lethargy (not due to depression) implicate a frontal–parietal circuit (e.g., Chochon, Cohen, van de Moortele, & Dehaene, 1999; Peers, Ludwig, Rorden, Cusack, Bonfiglioli, Bundesen, Driver, Antoun, & Duncan, 2005; Ravizza, Delgado, Chein, Becker, & Fiez, 2004; Rivera, Reiss, Eckert & Menon, 2005; Simon, Mangin, Cohen, Le Bihan, & Dehaene, 2002; van Honk, Schutter, Putman, de Haan, & d'Alfonso, 2003).

Several groups have noted deficits in executive functions in children with ADHD (Bayliss & Roodenrys, 2000; Pennington & Ozonoff, 1996; Powell & Voeller, 2004); but, because so many of the children studied were hyperactive, many previous studies and theorists have emphasized deficits in inhibitory control, especially in motor inhibition (Barkley, 2000; Nigg, Blaskey, Huang–Pollock, & Rappley, 2002). I propose that the core executive function deficit in ADD is in working memory, as it is in patients with frontal cortex damage who suffer from a dysexecutive syndrome. If I am correct about the underlying neurobiological bases for ADD and ADHD it would hardly be surprising that executive functions would be compromised in both disorders, despite their being distinct disorders, given that ADHD a frontal–striatal circuit is disrupted and in ADD a frontal–parietal circuit is hypothesized to be most affected.

Children With ADD, Like Adult Dysexecutive Syndrome Patients, Have a Primary Deficit in Working Memory

The term working memory has been defined in a number of different ways. Goldman–Rakic (1987) used it to refer to holding information in mind. Baddeley (1992; Baddeley & Hitch, 1994) defined working memory as holding information in mind while simultaneously manipulating or transforming that information (maintenance + manipulation, or temporary storage + processing). Many have adopted that seminal model of working memory, including D'Esposito, Detre, Alsop, Shin, Atlas, and Grossman (1995), Miyake and Shah (1999), Petrides (1995), and Smith and Jonides (1999). Another prominent model of working memory is that of Engle, who defines working memory as the ability to (a) maintain selected information in an active, easily retrievable form while (b) blocking or inhibiting other information from entering that active state (i.e., maintenance + inhibition; Conway & Engle, 1994; Kane & Engle, 2000, 2002). This shares much in common with the influential thinking of Hasher and Zacks (1988) who have emphasized the inhibitory requirements of gating out irrelevant information from the mental workspace of working memory and deleting no longer relevant information from that limited-capacity workspace.

The perspectives of Baddeley and Engle share in common with my own (e.g., Diamond, 1990, 2002) that simply holding information in mind is not that taxing (unless the number of items becomes very large) and does not generally require involvement of dorsolateral prefrontal cortex (Brodmann's Areas 46 and 9). It is when holding information in mind must be combined with another operation, such as manipulation (which Baddeley emphasized) or inhibition (which Engle and I have emphasized), that cognitive capacity is truly taxed and the dorsolateral prefrontal cortex is required. I have argued that inhibition and holding information in mind are dissociable in that they can be independently varied, although it is their conjunction that requires dorsolateral prefrontal cortex involvement. There is general agreement that the dorsolateral prefrontal

cortex is needed when one must both maintain information in mind and perform another operation (such as working with that information or inhibiting a strong response tendency; for reviews, see D'Esposito, Postle, & Rypma, 2000; Owen, 1997; Petrides, 1996; Smith, Jonides, Marshuetz, & Koeppe, 1998;).

Activation of the dorsolateral prefrontal cortex is more likely to be increased if you are asked to add numbers or repeat them backward (backward digit span) than if you are asked to simply repeat them (forward digit span). Simply repeating back digits in the order in which you have heard them does not require working with the information held in mind; it does not require working memory. Patients with prefrontal damage often show no impairment on forward digit span, although they perform worse than controls on backward digit span (Stuss & Benson, 1986). Mixed groups of children with ADHD and/or ADD also perform worse than controls on backward, but not forward, digit span (Mariani & Barkley, 1997; McInnes, Humphries, Hogg–Johnson, & Tannock, 2003; Milich & Loney, 1979; Shue & Douglas, 1992) and backward, but not forward, spatial span (McInnes et al., 2003).

Similarly, frontal patients and children with ADD have problems when they have to add or manipulate numbers in their head (Barkley et al., 1990; Benedetto–Nasho & Tannock, 1999; Hynd et al., 1991; Welsh & Pennington, 1988; Zentall & Smith, 1993) or when they have to compute two-step problems in their heads, although they can solve each step individually (Barbizet, 1970; Barkley, 1997; Luria, 1973). They can remember an item as well as anyone. Their deficit becomes evident as the number of items increases and as the demands on manipulating those items increase.

Frontal patients and children with ADD perform well on many assessment measures, leading some to argue that tests cannot capture their executive dysfunction (e.g., Barkley et al., 1991). It is easy to see why people would come to that conclusion. The tests used often measure short- or long-term memory rather than working memory. The tests people have used often focus on discrete skills (rather than the conjunction of holding information in mind plus manipulating it or exercising inhibition). Further, tests are often given in situations where there are minimal distractions and the examiner provides the executive functioning, such as repeatedly bringing the test taker back to the task at hand. Finally, frontal patients and children with ADD can have periods of excellent executive functioning; they just cannot perform reliably at that level. A single "snapshot" testing might catch a patient at an unrepresentatively high level of executive functioning. Better accuracy and reliability can be achieved if more than a single testing is administered.

I predict that *complex-span tasks* will prove exquisitely sensitive to ADD and will capture the essence of the cognitive problem of individuals with ADD. Complex-span tasks require transforming information held in mind under high-interference conditions (Dempster, 1981, 1985). When people discuss individual differences, or age-related differences, in working memory, they are often referring to differences in precisely these tasks.

One such complex-span task is the *counting-span task* (Case, Kurland, & Goldberg, 1982). On each trial, the participant is asked to count the number of blue dots, which appear embedded in a field of yellow dots, touching each blue dot and enumerating it. Immediately thereafter, the participant is to give the total number of blue dots for that display and the total number of blue dots for all preceding displays in correct serial order. This requires holding information in mind while executing another mental operation (counting), selectively attending to the blue dots while inhibiting attention to the yellow ones, updating the information held in mind on each trial, and keeping track of the order of the totals computed across trials (temporal order memory).

In the *spatial-span task* (Case, 1992a, 1992b), the participant inspects a 4×4 matrix on each trial, noting which cell is shaded. A filler pattern is then shown, and then an empty 4×4 grid. The participant is to point to the cell that had been shaded on that trial. Over several blocks of trials, the number of shaded cells gradually increases. Interference from prior trials and from the filler pattern is high.

The *pattern-span task* is similar to the spatial-span task. Several cells are shaded. The participant gets a quick look at the pattern. At test, one of the cells that had been shaded is now unshaded and the participant must point to that cell. The number of shaded cells increases until the participant's accuracy falls below criterion. Performance on the pattern-span task, as on the counting and spatial-span tasks, improves greatly between 5 and 11 years of age, when it starts to be asymptotic (Miles, Morgan, Milne, & Morris, 1996; Wilson, Scott, & Power, 1987).

In the *compound stimulus visual information* task (Pascual–Leone, 1970), the participant is taught a different novel response (e.g., raise your hand, clap your hands) for each of several different visual cues (e.g., a square shape or red color). After learning these to criterion, compound stimuli (e.g., a red square) are presented, each for 5 s, and the participant is to "decode the message" by producing every response called for by the stimuli. As on the above complex-span tasks, the number of correct responses increases until about 11 years of age (Case, 1972, 1995).

Two language-related complex-span tasks have been developed by Daneman and Carpenter (1980). The *listening-span* task (Daneman & Carpenter, 1980) requires processing auditorially presented sentences (sometimes being asked to verify the truthfulness of the sentence just read) while retaining, in correct temporal order, the final words of each preceding sentence. Performance on that improves from 6 years until at least 15 years and probably until the early 20s (Siegel, 1994). The *reading-span* task (Daneman & Carpenter, 1980) is similar, but participants read the sentences aloud themselves, rather than hearing someone else read them.

Performance across these complex-span tasks is remarkably consistent and shows exceptionally consistent developmental change. The counting and spatial-span tasks have been normed on large numbers of children over a wide age range. A meta-analysis by Case (1992a, 1992b) of 12 cross-sectional studies shows developmental progressions for these two measures that could not be more comparable (see Figure 1). In typically developing children, continuous and marked improvements are seen from 4.5 to 8 years of age, then continued, more gradual improvement until 10–11 years of age, with much more gradual improvement thereafter. The compound stimulus visual information task has also been administered to large numbers of children and is highly correlated with performance on Case's counting and spatial-span tasks. A mirror image of the close relation between improvements on complex-span tasks during early development is the remarkably similar developmental degradation during aging across letter, reading, spatial, and computation-span tasks (Park & Payer, 2005).

There is no research of which I am aware that looks at complex-span test performance in children with ADD, and almost none looking at this in children with broadly defined ADHD. One of the few studies examining the latter is the outstanding work of Westerberg, Hirvikoski, Forssberg, and Klingberg (2004). They administered a spatial-span task and found a striking difference between children with ADHD broadly defined and controls. The size of the group difference increased markedly with age due to floor effects at the youngest ages (see Figure 2). I predict that mathematical and linguistic complex-span tasks would show similarly striking group differences, that the differences would be even more dramatic if only children with ADD were included, and that marked differences at the youngest ages tested here, and at still younger ages, can be found with complex-span measures more appropriate for younger children.

Verbal presentation of material places a particularly high demand on working memory. Hence, it is proposed that verbal presentation of material is not the best instructional format for children with ADD. Findings of central auditory processing problems in many children with ADD (e.g., Gascon, Johnson, & Burd, 1986; Riccio, Hynd, Cohen, & Hall, 1994) may be largely due to working memory demands. Listening comprehension is highly correlated with both spatial and verbal working memory (e.g., Daneman & Carpenter, 1980; Just & Carpenter, 1992; McInnes et al., 2003). Indeed, a good part of the co-occurrence of language impairment with ADD may be

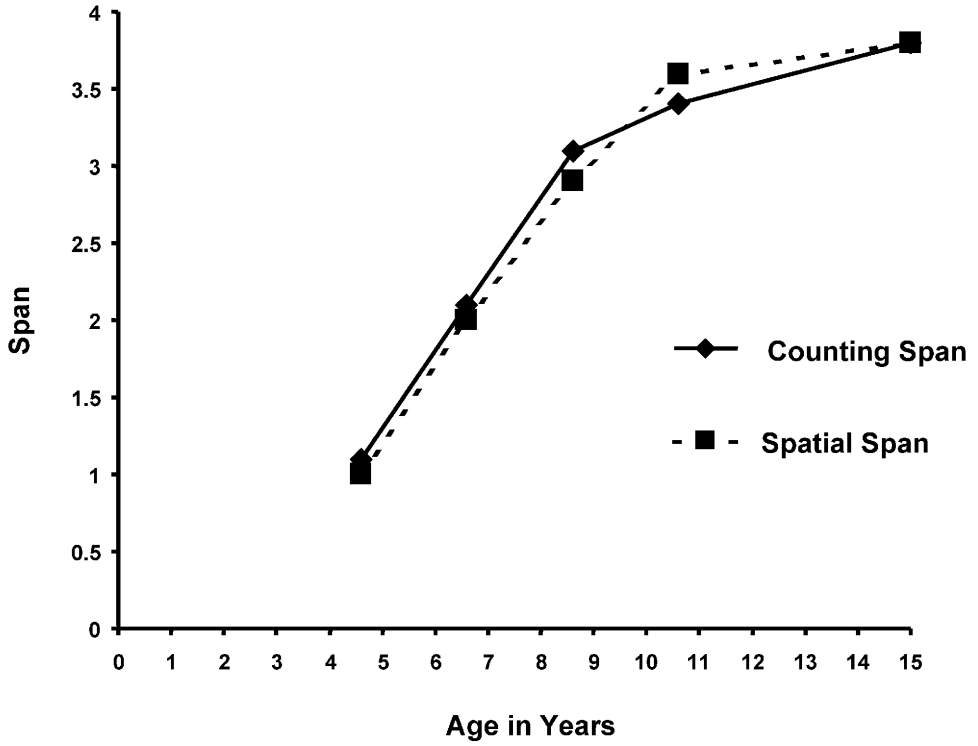

Figure 1. The scores for 4.5- to 15-year-old children on the number of items that can be held in mind (span) on the counting-span and spatial-span tasks. The data for the counting-span task are adapted from Crammond (1992) and those from the spatal-span task are adapted from Menna (1989). From "Normal development of prefrontal cortex from birth to young adulthood: Cognitive functions, anatomy, and biochemistry," by A. Diamond. In *Principles of Frontal Lobe Function*, by D. T. Stuss and R. T. Knight (Eds.), 2002, London: Oxford University Press. Copyright 2002 by Oxford University Press. Reprinted with permission.

due to the working memory demands of much linguistic processing. Children with ADHD broadly defined have no difficulty recalling discrete facts from verbally presented stories (that requires no working memory; Lorch, Milich, Sanchez, van den Broek, Baer, Hooks, Hartung, & Welsh, 2000; Pugzles Lorch, Milich, & Sanchez, 1998; Sanchez, Lorch, Milich, & Welsh, 1999; see also Aaron, Joshi, & Phipps, 2004; Ghelani, Sidhu, Jain, & Tannock, 2004). They show deficits, however, in comprehending complex causal relationships from those same stories (Aaron, Joshi, & Phipps, 2004; Ghelani, Sidhu, Jain, & Tannock, 2004; Lorch, Milich, Sanchez, van den Broek, Baer, Hooks, Hartung, & Welsh, 2000; Pugzles Lorch, Milich, & Sanchez, 1998; Sanchez, Lorch, Milich, & Welsh, 1999). *Dichotic listening tasks*, especially those that require multitasking (reporting what is heard in both ears; Lipschutz et al., 2001), I predict, should be as sensitive to detecting differences in performance between children with ADD and comparison groups as complex-span tasks. Higher working memory span and better performance on dichotic listening are highly correlated (Conway, Cowan, & Bunting, 2001). Indeed, tasks in the auditory domain, whether complex-span or dual-task dichotic listening, should be particularly sensitive to the problems of children with ADD for the reasons discussed above.

In my experience, there is often a trade-off between linguistic and spatial skills. Individuals with ADD are often superior in spatial reasoning and/or artistic drawing. The verbal component of schooling is enormous. Were spatial skills more emphasized in school, and verbal skills less so, children with ADD would show themselves to be far better students.

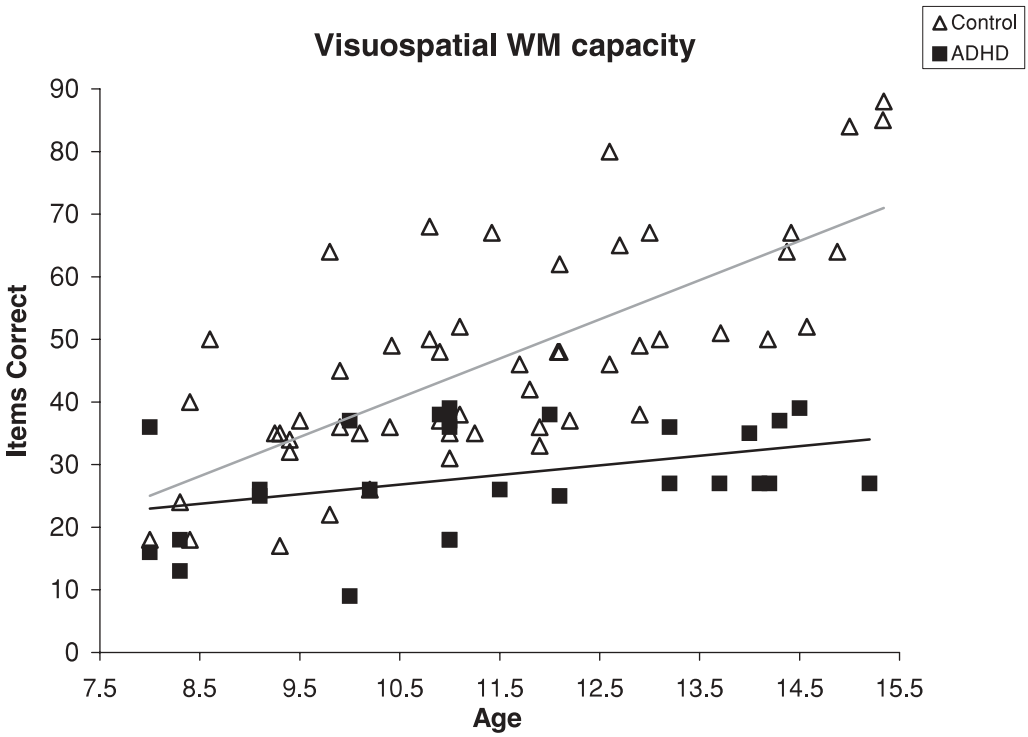

Figure 2. The scores for 8- to 15-year-old children with and without ADHD on the number of items that can be held in mind (span) on a visuospatial-span task. The number of participants in the study was 80. The linear regression lines are for children with ADHD (darker line) and the control group (lighter line). WM, working memory. From "Visuospatial Working Memory Span: A Sensitive Measure of Cognitive Deficits in Children With ADHD," by H. Westerberg, T. Hirvikoski, H. Forssberg, and T. Klingberg, 2004, *Child Neuropsychology*, *10*. Copyright 2004 by Psychology Press (www.psypress.co.uk/journals.asp). Reprinted with permission.

The name ADD implies a primary deficit in attention. It may seem odd, then, that I am proposing a primary deficit in working memory. It is perhaps a bit less odd when the close, intimate relation between memory and attention is appreciated. Focusing on information held in mind for several seconds might as easily be called focused or sustained attention as working memory. Behavioral (Awh & Jonides, 2001; Barnes, Nelson, & Reuter–Lorenz, 2001; de Fockert, Rees, Frith, & Lavie, 2001) and neuroimaging (Awh, Anllo–Vento, & Hillyard, 2000; Casey, Forman, Franzen, Berkowitz, Braver, Nystrom, Thomas, & Noll, 2001; LaBar, Gitelman, Parrish, & Mesulam, 1999) studies converge on the conclusion that the same neural system that is important for working memory is important for selective attention. Individual differences in working memory correspond to individual differences in selective attention (Conway, Tuholski, Shisler, & Engle, 1999; Kane, Bleckley, Conway, & Engle, 2001). The same prefrontal system that helps us selectively attend to stimuli in our environment (tuning out irrelevant stimuli) is the same system that helps us selectively keep our mind focused on the information we want to hold in mind in working memory.

Children With ADD, Like Adult Dysexecutive Syndrome Patients, Often Have Slow Processing Speeds

Another primary characteristic of a large subset of children with ADD is very slow reaction time and speed of processing (e.g., Barkley, Grodzinsky, & DuPaul, 1992; Holdnack, Moberg, Arnold, & Gur, 1995; Weiler,

Holmes–Bernstein, Bellinger, & Waber, 2000; Westerberg et al., 2004). Many ADD children, although not all, appear sluggish, drowsy, spacey, lethargic, and markedly hypoactive (Barkley et al., 1990; Hynd, Nieves, Connor, Stone, Town, & Becker, 1989; Lahey & Carlson, 1991; Stanford & Hynd, 1994). They fit the criteria for having a sluggish cognitive tempo (SCT; Carlson & Mann, 2000, 2002; Frick et al., 1994; Goodyear & Hynd, 1992; Hartman, Willcutt, Rhee, & Pennington, 2004; Milich et al., 2001). The SCT classification, especially its features of daydreaming and drowsiness (not due to medication), which can be separate from slow speed, limits its applicability to only a subset of ADD children (Carlson & Mann, 2002).

Fast speed of processing is not an executive function, yet slow response rates are typical of patients with the dysexecutive syndrome. For reasons not yet fully understood, (a) frontal patients have slowed reaction times and can sometimes perform well on tasks on which they are typically impaired if given more time, (b) there is a strong, well-replicated relation between speed of processing and performance on executive function measures (Duncan, Burgess, & Emslie, 1995; Fry & Hale, 1996; Kail & Salthouse, 1994; Salthouse, 1992), (c) age-related improvements in speed of processing during childhood and adolescence are highly correlated with developmental improvements on complex-span tasks (Case et al., 1982; Hitch, Towse, & Hutton, 2001; Kail, 1992), and (d) age-related decline in the speed of processing from early through late adulthood is highly correlated with age-related decline in performance on complex-span tasks and related measures of executive function (Salthouse, 1992, 1993; Salthouse & Meinz, 1995).

Children's performance on the counting-span task is linearly related to the speed with which they can count the presented dots (Case et al., 1982). Similarly, the faster people can repeat back the word they have just heard, the more words they can hold in mind. As the speed of word repetition improves so too does word-span memory. When the speed at which adults and 6-year-olds can repeat back words is equated (by presenting adults with unfamiliar words), children and adults show equivalent word-span memory (Case et al., 1982). Similarly, when the speed at which adults and children can count is equated (by requiring adults to count in a foreign language), equivalent counting-span memory is found in adults and 6-year-olds.

The empirical relation between performance on complex-span tasks and generalized speed of processing might be due to any number of factors. Faster processing would mean that items do not need to be held in mind as long, reducing the demand on working memory. Faster processing and improved executive function performance may covary because they both reflect more efficient neural processing and improved signal/noise ratios, either because of systemwide improvements in the nervous system or because a better functioning prefrontal cortex improves signal/noise ratios for diverse neural regions, permitting faster and more efficient cognitive processing.

Whereas impaired working memory appears to be ubiquitous in ADD, slower speed of processing is not, although it is quite common. Similarly, although a great deal of the variance in performance on complex-span tasks can be accounted for by processing speed, controlling for speed does not eliminate all age-related differences in complex-span performance (Hitch et al., 2001). Speed and complex-span performance are correlated, but not perfectly so. Indeed, in a study of ADHD broadly defined, poor working memory, poor attentional inhibition, and disorganization were found to load a separate factor from sluggish cognitive tempo (Carlson & Mann, 2002).

Simple choice reaction-time tests would seem a reasonable way to obtain a quick and easy indication of whether a child's response speed is slowed or not. Westerberg et al. (2004) report that choice reaction-time performance differentiated children with ADHD broadly defined almost as well as did complex-span performance, and far better than performance on either a continuous performance task or a go/no-go measure. If one finds that a child with ADD has a slowed reaction rate, it does not necessarily follow, however, that a better instructional format for the child is to present material at a consistently slow rate if that might lead to boredom. Studies have shown that children with ADHD broadly perform poorly when

material is presented at a constant slow rate (e.g., Sykes, Douglas, Weiss, & Minde, 1971), as do frontal patients (Rueckert & Grafman, 1998). However, if rates of presentation are intermixed, children with ADHD broadly defined are able to benefit from the greater processing time available for the more slowly presented items without that being counteracted by their attention wandering because the task is too easy and boring (Conte, Kinsbourne, Swanson, Zirk, & Samuels, 1986).

A Motivational Component to ADD

Although the literature and diagnostic manuals refer to children with ADD as easily distracted, I would like to propose that a more accurate description is that they are easily bored. Their problem lies more in motivation than it does in inhibition. Having lost interest in a project after only a short time, their attention drifts as they look for something else to engage their interest. Bored with the initial task, they abandon it before completion, moving on to the next project. It is not so much that external distraction derails them, as that they go looking for external (or internal) distraction because their interest in what they are supposed to be doing, or had started, has dwindled. (Sergeant, Oosterlaan, and colleagues have proposed a cognitive-energetic model of ADHD, which shares some features in common with what is being discussed here, but they have focused especially on aberrant reactions to reinforcement, which is different from the focus here, e.g., Luman, Oosterlaan, & Sergeant, 2005; Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003.)

Challenge or risk, something to literally get their adrenaline pumping, can be key to keeping their attention and to eliciting optimum performance from persons with ADD. In line with this, adults with ADD sometimes say they can focus better when driving if they speed than if they drive slowly. Children with ADHD broadly defined often perform normally on the continuous performance task when challenged by a fast presentation rate (Chee, Logan, Schachar, Lindsay, & Wachsmuth, 1989; van der Meere, Wekking, & Sergeant, 1991). Individuals with ADD, although typically shy, may engage in risk-taking and thrill-seeking activities, such as bungee jumping or riding roller coasters, as ways to experience a level of engagement they have difficulty sustaining in their daily lives. Computer and video games (which children with ADD can play for hours and hours) are fast paced, often with imminent danger keeping arousal high. Such games often rely on the execution of well-practiced associations between button presses and game features or well-practiced sequences of button presses, which children with ADD have no difficulty retrieving from their intact long-term memory and procedural memory. Executive function is taxed when conscious, top-down control is needed. The execution of any well-practiced skill, such a playing a computer game, is impaired by attempts to exert top-down control and is optimized by allowing older, subcortical systems to guide performance (e.g., Herrigel & Suzuki, 1953; Miller, Verstynen, Raye, Mitchell, Johnson, & D'Esposito, 2003).

Another way of looking at this is that if the neural systems of individuals with ADD have poorer signal/noise ratios, as would be consistent with slower speed of processing, then sustaining focused concentration on all the things that must be remembered and integrated for a task might well be more demanding for individuals with ADD. Hence, they would "burn out" on a task earlier than other folk and would need a greater infusion of adrenaline to fuel the system. Under the right circumstances, when sufficiently motivated, children with ADD (like patients with frontal cortex damage) can perform well, but it is hard for them to sustain that level of performance (*frontal patients*: Fuster, 1989; Stuss & Benson, 1986; *children with ADHD broadly defined*: Corkum & Siegel, 1993; Douglas & Peters, 1978; van der Meere & Sergeant, 1988).

Discussion

The thesis that has been presented here is that (a) ADD (ADHD of the inattentive subtype without hyperactivity) is a different disorder from ADHD that includes hyperactivity. The two differ in their cognitive and behavioral profiles, patterns of comorbidities, responses to medication, and underlying neurobiological disor-

der. (b) ADD provides an instance of childhood-onset dysexecutive syndrome. (c) The core cognitive deficit of ADD is in working memory. Instructional methods that place heavy demands on working memory will disproportionately disadvantage individuals with ADD. Although many have remarked on an executive function deficit in ADHD broadly defined, the overwhelming emphasis has been on a core deficit in inhibition, especially response inhibition, rather than in working memory (e.g., Barkley, 1997; Nigg, 2001; Pennington & Ozonoff, 1996). An emphasis on response inhibition is appropriate for ADHD that includes hyperactivity, but it is argued here that that emphasis is inappropriate for ADD, where the primary deficit is in working memory. (d) The working memory deficit in ADD should be detectable by standardized testing if measures such as complex-span and/or divided-attention dichotic-listening tests are used. Examples of several complex-span working memory measures were provided as were reasons why earlier attempts to capture the cognitive deficit in ADD children using standardized measures were unsuccessful. (e) Language problems often co-occur with ADD, and it is suggested that part of the reason might be that linguistic tasks, especially verbal ones, tax working memory so heavily. Spatial and artistic skills, however, are often preserved or superior in individuals with ADD. (f) The working memory deficit in many children with ADD is accompanied by markedly slowed reaction times, a characteristic that covaries with poorer working memory in general. (g) Individuals with ADD have difficulty maintaining a sufficiently high level of motivation to complete a task and grow bored quickly, perhaps tiring because the working memory demands of the task exhaust them. They go looking for something else to do or think about because they are bored, rather than being unable to inhibit the pull of distractions. Their problem is not so much that are distractible as that they are easily bored. When engaged in an activity they enjoy they are fully able to successfully ignore even potent distractions. To remedy a general lower arousal level, they may seek risks that increase their level of arousal and attentiveness.

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