

ADHD
and
Executive Control

On the Merits of an Executive Control Theory of
Attention-Deficit/Hyperactivity Disorder

UNIVERSITEIT VAN AMSTERDAM
Department of Psychology

Arjan Berkeljon

ADHD

and

Executive Control

On the Merits of an Executive Control Theory of
Attention-Deficit/Hyperactivity Disorder

by

Arjan Berkeljon

Studentnumber 9906509

A thesis (*doctoraalscriptie*) submitted in partial
fulfillment of the requirements of the degree

Master of Science in Psychology
(Doctorandus in de Psychologie)

Supervised by

dr. W.P.M. van den Wildenberg

Second reading by

Dr. H.M. Geurts

UNIVERSITEIT VAN AMSTERDAM

Department of Psychology

Zaandam, June 2006

Contents

Contents	i
1 Introduction	1
1.1 The nature of ADHD	2
1.2 Executive functioning	7
2 Executive Control Theories of ADHD	13
2.1 Executive functioning and ADHD	13
2.2 Barkley's behavioral inhibition model of ADHD	15
2.3 Predictions of the behavioral inhibition model	21
3 On the Merits of Executive Control Theories of ADHD	27
3.1 Criteria for a primary executive control deficit in ADHD	28
3.2 Applying the primary deficit criteria to ADHD	29
3.3 The nature of executive control deficits in ADHD	30
4 Conclusion	37
References	40

1

Introduction

The purpose of this thesis is to evaluate theories of Attention-Deficit/Hyperactivity Disorder (ADHD) in which executive control plays a central role. Such theories emphasize the involvement of deficits in executive functions, such as directing and maintaining attention, and appropriate behavioral/response inhibition, in the symptoms associated with ADHD [e.g. Pennington and Ozonoff, 1996; Barkley, 1997a,b]. In recent years however, a number of researches have questioned the proposed central role of disorders of executive functioning in ADHD [e.g. Sergeant, Geurts, and Oosterlaan, 2002; Boonstra, Oosterlaan, and Buitelaar, 2005; Willcutt, Doyle, Nigg, Faraone, and Pennington, 2005]. In this thesis, both sides of this argument will be discussed and their claims evaluated.

Attention-Deficit/Hyperactivity disorder is thought to affect a sizable percentage of school-age children (numbers between 3 and 10% are reported [Barkley, 1997a,b; Faraone et al., 2003]). Children with ADHD are characterized by an inability to sustain attention for longer periods of time, over-activity, and an inability to persist in activities or tasks when compared to their peers [Barkley, 1997a].

Of course not every highly active child or a child that has lapses in concentration has ADHD. A diagnosis of ADHD is only given if a cluster of symptoms persists for a prolonged period of time. The standard diagnostic criteria for ADHD are given in the Diagnostic and Statistical Manual (DSM) issued by the American Psychiatric Association (the current edition, from 2000, is the DSM-IV-TR, a text revision of the DSM-IV from 1994). These criteria are printed in table 1.1.

As useful as these criteria are in diagnosing and describing ADHD, in themselves they say preciously little about the origin(s) and cause(s) of ADHD. This is, of course, true of any pathology or disorder, a description of symptoms is not

a theory of its cause(s). It is the symptoms instead, that require an explanation. Thus, if we recognize ADHD as a distinct and well-demarcated disorder, a search for its origin(s) and cause(s) should aim to find what explains each of the behavioral patterns symptomatic of ADHD, as well as why they coexist in the way they do.

This is complicated by the fact that, at least in some patients, the symptoms of hyperactivity appear to occur without the symptoms of inattention and vice versa. Thus in table 1.1 three subtypes of ADHD are recognized: (1) ADHD of the combined type (what is most commonly understood to be ADHD); (2) a predominantly inattentive type; (3) and a predominantly hyperactive type.

It is not clear however, if the predominantly inattentive type and the predominantly hyperactive type are proper subtypes of ADHD at all [Barkley, 1997a; Nigg, 2005]. Both alleged subtypes are quite rare and thus under-investigated. Since it is unclear how these alleged subtypes relate to ADHD in which both inattention and hyperactivity are present, it is unclear how they could be incorporated in a theory of the cause(s) of ADHD. Therefore, in the present article and evidence reviewed therein, only ADHD of the combined type shall be considered unless otherwise indicated.

1.1 The nature of ADHD

Barkley, in his influential work [Barkley, 1997a] describes the core symptoms of ADHD as: (1) inattention and (2) hyperactive-impulsive behavior (disinhibition) [*ibid.* p. 9].

Inattention

Inattention manifests itself as an inability to sustain attention in tasks or play for the same duration as their peers and to follow through on instructions and rules as well as their peers [Barkley, 1997a; Biederman, 2005]. Children who suffer from inattention symptomatic of ADHD are described as disorganized, distracted and forgetful when compared to other children of the same age. Parents and teachers often report the child's inability to follow instructions, their being easily distracted and frequently daydreaming, and their switching to a new activity without first completing the previous one more so than other children. Research using measures of attention corroborate these reports [Barkley, 1997a].

It is interesting to note that some research suggests that children with ADHD are mainly distracted by irrelevant stimuli within the task at hand.

Table 1.1: DSM-IV-TR Criteria for ADHD

A. Either (1) or (2):

- (1) **inattention:** six (or more) of the following symptoms of inattention have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:
- (a) often fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities
 - (b) often has difficulty sustaining attention in tasks or play activities
 - (c) often does not seem to listen when spoken to directly
 - (d) often does not follow through on instructions and fails to finish school work, chores, or duties in the workplace (not due to oppositional behavior or failure to understand instructions)
 - (e) often has difficulty organizing tasks and activities
 - (f) often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (such as schoolwork or homework)
 - (g) often loses things necessary for tasks or activities (e.g., toys, school assignments, pencils, books, or tools)
 - (h) is often easily distracted by extraneous stimuli
 - (i) is often forgetful in daily activities
- (2) **hyperactivity-impulsivity:** six (or more) of the following symptoms of hyperactivity-impulsivity have persisted for at least 6 months to a degree that is maladaptive and inconsistent with developmental level:

Hyperactivity

- (a) often fidgets with hands or feet or squirms in seat
- (b) often leaves seat in classroom or in other situations in which remaining seated is expected
- (c) often runs about or climbs excessively in situations in which it is inappropriate (in adolescents or adults, may be limited to subjective feelings of restlessness)
- (d) often has difficulty playing or engaging in leisure activities quietly
- (e) is often "on the go" or often acts as if "driven by a motor"
- (f) often talks excessively

Impulsivity

- (g) often blurts out answers before questions have been completed
- (h) often has difficulty awaiting turn
- (i) often interrupts or intrudes on others (e.g., butts into conversations or games)

(cont.)

Generally they do not appear to be more distracted by events external to the task than are children of the same age without ADHD [Barkley, 1997a].

Hyperactive-impulsive behavior (disinhibition)

Hyperactivity in children with ADHD manifests itself as an increased tendency to move around, run, climb, etc. than children of the same age without ADHD

Table 1.1: continued

-
- B. Some hyperactive-impulsive or inattentive symptoms that caused impairment were present before age 7 years.
 - C. Some impairment from the symptoms is present in two or more settings (e.g., at school [or work] and at home).
 - D. There must be clear evidence of clinically significant impairment in social, academic, or occupational functioning.
 - E. The symptoms do not occur exclusively during the course of a Pervasive Developmental Disorder, Schizophrenia, or other Psychotic Disorder and are not better accounted for by another mental disorder (e.g., Mood Disorder, Anxiety Disorder, Dissociative Disorders, or a Personality Disorder).

Code based on type:

314.01 Attention-Deficit/Hyperactivity Disorder, Combined Type: if both Criteria A1 and A2 are met for the past 6 months

314.00 Attention-Deficit/Hyperactivity Disorder, Predominantly Inattentive Type: if Criterion A1 is met but Criterion A2 is not met for the past 6 months

314.01 Attention-Deficit/Hyperactivity Disorder, Predominantly Hyperactive-Impulsive Type: if Criterion A2 is met but Criterion A1 is not met for the past 6 months

Coding note: For individuals (especially adolescents and adults) who currently have symptoms that no longer meet full criteria, "In Partial Remission" should be specified.

American Psychiatric Association [2000]

[Barkley, 1997a; Biederman, 2005]. Parents and teachers report them to be more active than their peers which is also corroborated by research findings.

These problems with hyperactive and disinhibited behavior appear to arise around 3–4 years of age. This is notably earlier than the problems associated with inattention, which usually arise around 5–7 years of age or later still. Subsiding of these symptoms shows a reverse pattern, hyperactivity declines as the child gets older while inattention persists at least up and until adolescence [Barkley, 1997a].

Prevalence and persistence of ADHD

Although usually regarded as a children's disorder, ADHD is known to persist into adolescence and adulthood in a substantial number of cases [Barkley, 1997a,b; Biederman, 2005]. Barkley [1997b] reports that in 50–80% of clinically diagnosed cases of ADHD the disorder persists from childhood into adolescence. Faraone et al. [2006] reports that in 15–65% of children with ADHD the disorder persists into adulthood, depending on if the full DSM-IV criteria for ADHD or only the criteria for partial remission are used (see 1.1 for these criteria).

Estimates of the prevalence of adult ADHD are between 3 and 5% [American Psychiatric Association, 2000; Biederman, 2005].

Biederman [2005] reports certain stable, yet not perfect, predictors of ADHD persistence, including family history of ADHD, psychiatric comorbidity, and psychosocial adversity. However, as Biederman also notes, persistence of ADHD is not necessarily associated with a complete and severe persistence of dysfunction. As was noted above with regard to a subsiding of the symptoms of hyperactivity and inattention, it appears that at least a partial remission of symptoms is possible. The role of treatment is not entirely clear in this regard.

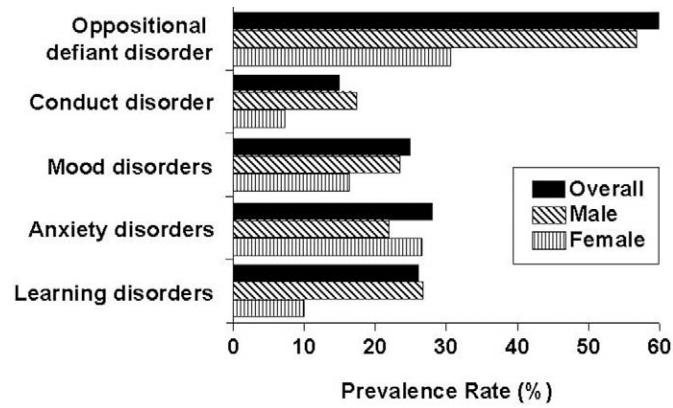
With regards to the familial influence it should be noted that ADHD is believed to have a significant genetic component. Studies on familial influence report a two- to eightfold increase in the risk for ADHD in parents or siblings of children with ADHD [Biederman, 2005]. Aggregate results from a number of twin studies reported by Biederman show a high mean heritability factor of .77. In an adoption study by Sprich et al. [2000] it was found that among the adoptive relatives of ADHD patients, rates of ADHD and comorbid disorders were lower than among the biological relatives of the ADHD patients in question. This also supports the hypothesis that ADHD has a strong heritable component.

Cognitive and academic effects

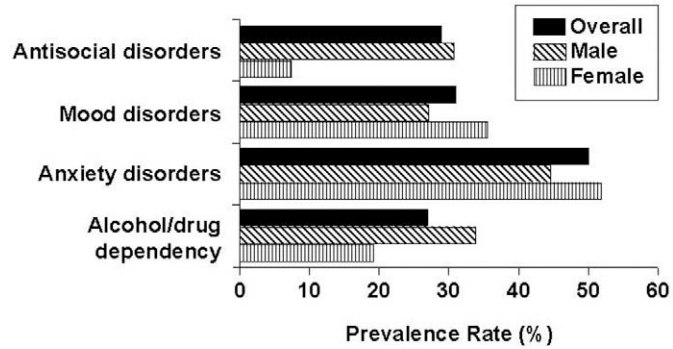
Barkley [1997a] reports the impact of the core ADHD symptoms in the cognitive domain. These include difficulties with motor control and sequencing behavior, digit span and mental computation, planning and anticipation, verbal fluency, effort allocation, applying organizational task strategies, internalization of self-directed speech, following restrictive instructions and self-regulation of emotion. These factors manifest themselves in the academic as well as social domain. Children with ADHD are thus at high risk for academic failure and social dysfunction [Biederman, 2005].

Comorbid disorders

Although ADHD (of the combined type) is recognized as a distinct developmental disorder it often appears alongside other disorders. As evident from the previous subsection one would expect difficulties in academic and social domains. In children with ADHD comorbidity of reading/learning disorder and mood and conduct disorder are regularly reported (see figure 1.1(a)). In adults with ADHD social and mood disorders are also reported (see figure 1.1(b)).



(a) Children



(b) Adults

Figure 1.1: Comorbid diagnoses in children and adults with ADHD [from: Biederman, 2005]

There is a relationship between comorbid disorders of ADHD and the interesting fact that an ADHD diagnosis is far more common in boys than it is in girls [American Psychiatric Association, 2000]. Biederman, Mick, Faraone, Braaten, Doyle, and Spencer [2002] reports that girls with ADHD are at less risk for comorbid disruptive behavior disorder than boys with ADHD (see also 1.1). Since clinical referral is often driven by disruptive behavior, this finding might explain the high discrepancy in the male/female ratio between clinically referred (10:1) and community referred (3:1) samples of children with ADHD.

Conclusion

The cognitive and academic effects outlined above have been interpreted as deficits in, or related to, executive functioning or control [e.g. Pennington and

Ozonoff, 1996; Barkley, 1997a,b]. The proposal then, is that ADHD could be symptomatic of a disorder of executive control. Before discussing the details and merits of such a proposal an introduction to the domain of executive functioning/control is given. Executive function and executive control are used somewhat interchangeably for the remainder of this chapter although the terms carry a different connotation. Executive functioning is a broad term covering a wide range functions involved in planning and problem-solving. Executive control, on the other hand, seems more narrowly concerned with those aspects of executive functioning that structure and control behavior. Executive functioning therefore, seems more apt to be used as a description of general cognitive functions while executive control, with its more narrow definition, seems better suited for a discussion of the specific cognitive functions that are thought to be disturbed in ADHD patients.

1.2 Executive functioning

Executive functions describe higher cognitive functionality such as working memory, planning, inhibition, integration of information, and task switching/attention [Fuster, 1997; Luciana, 2003]. Mainly through the work of Fuster [1997] these higher cognitive functions have come to be associated with the prefrontal cortex (PFC), the anterior area of the frontal lobe (e.g. the area of the brain directly behind the forehead). This interpretation of broadly defined functions and crude localizations has not gone without criticism but it is generally accepted as a correct, although general, model. That is, most everyone acknowledges the broader implications and predictions of this account while at the same time it is understood it is a coarse approximation not without caveats. For example Pennington and Ozonoff [1996] mention the “frontal metaphor”, a way of describing particular behaviors as “appearing to be frontal” because they resemble the behavior of documented frontal patients. This is of course not meant as a diagnosis or proper theoretical prediction but more as a heuristic in hypothesis formation and theory construction.

Development of executive functioning

On the basis of measurements through experimental tasks hypothesized to tap into executive functions and neuroanatomical development we can say several things about the development of executive functioning and the PFC. Broadly speaking the PFC and the executive functions correlated with it gradually develop during childhood until mature levels of development and function are reached in adolescence [Fuster, 1997; Luciana, 2003].

Cell-development in the PFC appears to be completed in the early postnatal period Luciana. Neurogenesis is achieved during pregnancy (rather later in development compared to other developmental trends), as is the formation of synapses that follows neurogenesis. This formation continues in infancy and levels off during later childhood and into puberty. After puberty gradual synaptic elimination begins which is thought to consolidate functionally stable neural circuits that have been acquired through behavioral experiences.

Behavioral evidence of the development of the PFC and executive functioning can be inferred from various experimental tasks, the one most often used being the spatial delayed response task. In such tasks, which have been tested on a variety of subjects (human and animal), the subject is presented with a stimulus in a particular location of the visual field. After a delay the subject must indicate the location of the stimulus without any aid from recognition memory. Thus the subject is thought to rely solely on an internal representation of the stimulus location kept in working memory. In non-human test subjects (primates) as well as human subjects the capacity to perform this task correctly emerges during late infancy, i.e. around the time that independent motor ability is achieved.

Performance on increasingly complex tasks gradually develops throughout childhood. For example in sorting tasks, such as the Wisconsin Card Sorting Task (WCST), children around ages 3-4 can reliably sort stimuli (cards with a varying number of geometric shapes in different colors in the case of the WCST) according to one criterion of stimulus attributes (e.g. shape only). As they get older children can switch between sorting according to one criterion to sorting according to another (between ages 4 and 5). Around age 6 children are able to sort according to three different criteria.

It has been observed that children make mistakes on the WCST similar to those frontal patients make. These so-called perseveration errors, which manifest themselves as the inability to switch to using a new sorting rule and continuing to sort according to the previous rule, are common to both groups. It has been suggested a lack of inhibition of the previously learned sorting rule (a prime example of an executive function) underlies these errors [Crone, Ridderinkhof, Worm, Somsen, and van der Molen, 2004].

Similarly, on tasks of behavioral inhibition tasks such as the Stroop task. In this task subjects are required to read aloud color names. Difficulty is created by printing the color names in colored fonts that are different from the name printed (e.g. BLUE is printed in green). For young children a variant of this task is used called the Day-Night Stroop task. In this task children must respond to two cards, one showing a picture of the sun and the other of the moon. Difficulty is created by requiring the children respond by saying "Night"

in response to a picture of the sun, and “Day” in response to a picture of the moon. This task is difficult for 3–4-year-olds but fairly easy for 6–7-year-olds. Thus performances on frontally guided tasks improves considerably during the preschool and early school period. Adequate performance on complex frontally guided tasks and behavioral patterns is not reached until after puberty however.

To follow the progression of development and performance across age groups a battery of test measuring frontal functions was developed that could be administered to subjects in age groups ranging from pre-schoolers to adults. This battery of tests, the Cambridge Neuropsychological Testing Automated Battery (CANTAB), consist of of subtasks in the following three domains: (1) working memory/planning; (2) visual memory; and (3) visual attention. Luciana [2003] mentions several studies using the CANTAB to assess the development of frontal lobe function. Results of these studies corroborate the earlier mentioned finding that performance on frontally guided tasks increases during childhood but does not reach a adult performance levels until after puberty [*ibid.*].

Executive function and dysfunction

Findings of executive functioning deficits related to disturbances of the frontal areas of brain further substantiates the correlation between these areas and executive functioning. As the development of executive functions follows the maturation of the frontal cortex and thus provides a positive indication of the involvement of the frontal cortex in executive functioning, the pattern of dysfunctional behavior following frontal lesions provides a reverse indication of the role of the frontal cortex.

Fuster [1997] describes seven broad functional disorders related to PFC dysfunction: (1) attention and perception; (2) motility; (3) Memory; (4) planning; (5) intelligence; (6) temporal integration; and (7) affect and emotion. These dysfunctions are observed in frontal patients in varying degrees of severity and are therefore hypothesized to be functionally correlated with this area of the brain.

Attention and perception

Deficits in these areas result from abnormalities of attention. Fuster distinguishes seven subtypes: (1) low alertness in which a patient appears generally less alert and aware of the world around him; (2) sensory neglect in which a patient lacks full awareness of one side of the body including any stimulation of this area; (3) distractibility in which a patient is abnormally attracted to irrelevant stimuli and finds it hard to not let these stimuli interfere in ongoing

task performance; (4) disorders of visual search and gaze control manifested by an inability to direct attention and unsystematic visual scanning; (5) difficulty sustaining attention manifested by an inability to maintain concentration for any given period of time; (6) internal inference manifested by a sensitivity to interfering internal impulses; and (7) defective motor attention manifested by a sensitivity to internal interference on motor tasks (lack of motor inhibition).

Fuster also notes that frontal lesions are known to cause deficits of perception that cannot be related to deficits in attention. These are mostly disorders in perceiving spatial relationships in one's environment or performing tasks that require guidance by visual information.

Motility

Disorders of motor behavior, not resulting from lesions in the (pre)motor cortex, can result from damage to the PFC. Two kinds of disorders can be distinguished: (1) disorders of general spontaneous motor behavior; and (2) disorders of goal-directed motor behavior.

Disorders of spontaneous motor behavior fall into two categories, hypokinesia and hyperkinesia. Hypokinesia is diminished spontaneous motor activity which can vary greatly in degree, ranging from mild asponaneity to severe apathy. Hyperkinesia on the other hand is characterized by excessive and aimless motor behavior (hyperactivity).

Disorders of goal-directed motor behavior often exist alongside the disturbed motor behavior just mentioned, but are not caused by them. They are thought to result from cognitive deficits in initiation, planning, and organization of action which in turn are manifestations of PFC lesions in their own right.

Memory

PFC patients are capable of forming and retrieving long-term memory episodes. Any problems associated with long-term memory are most often attributable to deficits in organization and monitoring of the material to be committed to memory. There thus appears to be a deficit in applying so-called mnemonic strategies most probably because of a lack of attention and drive.

A typical, and more structural problem is that of a working memory deficit. Frontal patients exhibit problems on tasks that require keeping in memory certain operations required for adequate task performance. These effects appear to be related to deficits in sustained attention and susceptibility to interference but are completely explained by them. There are patients who fail on tests of working memory even if attention and interference are controlled for.

Planning

Fuster notes that while deficits in memory lead to problems with hindsight, forethought is hindered by planning difficulties. This lack of “prospective memory” is closely related to difficulties in formulating and applying plans. As with working memory deficits, here too inattentiveness and lack of drive appear to play a role in this aspect of the disorder. Also the heightened sensitivity to internal interference (competing plans or ideas) most frontal patients exhibit may pose especially disruptive in executing plans.

Intelligence

Setting aside the semantic mess that plagues intelligence research a few general remarks about intelligence in relation to the PFC can be made. One striking finding is that most frontal patients have normal IQ scores and are capable of marked intellectual achievements. However, as the previous suggest, certain aspects of cognitive function are most definitely impaired. Upon closer inspection it appears that so-called ‘fluid intelligence’, or the capacity to solve novel problems, is indeed impaired in frontal patients. This is not necessarily completely detrimental to cognitive functioning but it is a definite impairment nonetheless. It speaks for the versatility and persistence of those patients that are able to achieve quite normal performance and behavior in spite of their cognitive impairments.

Temporal integration

Temporal integration concerns the integration of (novel) behavioral routines. Frontal patients have no trouble carrying out previously learned behavioral routines but learning new ones is an almost impossible challenge. Fuster remarks that the synthesis of new behavioral routines, acts or plans is perhaps the most quintessential aspect of ‘executive function’. This deficit of temporal integration then, appears to be the cumulative effect of the previously mentioned functional disorders.

As with the deficits in intelligence the deficits in temporal integration do not preclude normal functioning. They are only evident in challenging situations and not necessarily in simple, daily activities. Thus frontal patients can lead relatively normal lives, albeit with much routine and habit and without much creativity or imagination.

Affect and emotion

These are among the most elusive of disorders associated with PFC lesions since it is difficult to disentangle these emotional problems from the cognitive disorders they may be secondary too. Be this as it may, two emotional manifestations are observed, often dissociated: apathy and euphoria.

Apathy is manifested by the same symptoms as inattention and hypokinesia (diminished motor activity). Patients show low awareness and lack of initiative and motility. Affect and emotional responses are most aptly described as blunted. Note that this is not the same as clinical depression although Fuster reports that PFC lesion can result in clinical depression. In both cases it is possible that the changes in affective behavior follows cognitive deficits as a reaction to the deterioration of cognitive functions.

Euphoria, as it occurs in frontal patients, is characterized by a sporadic but recurrent elevation of mood and resembles pathological mania. It is often associated with two symptoms of frontal dysfunction discussed earlier, distractibility and hyperactivity.

Social effects

As should be evident from the discussion of major symptoms of frontal dysfunction above, a lesion in the frontal lobes can have varying effects on cognitive functioning and behavior. As such, quite often lesions in this area of the brain lead to changes, be that subtle or dramatic, in the way lesioned patients interact socially. Some cognitive effects will be less noticeable than others. The effects on affect and emotion are usually among the most dramatic changes. Such changes can and do have adverse effects on the frontal patient's personality and social behavior.

Conclusion

Integrating the neurophysiological and behavioral data suggests that functional maturation of the PFC and its correlated functions occurs as the PFC matures structurally. As a corollary to this we might suppose that particular functional disturbances in the development of frontal lobe functions are attributable to certain structural developmental disturbances. In the case of several developmental disorders, such as autism but also ADHD, this is exactly what several theorists have proposed. These executive functioning/control theories of ADHD are the topic of the next chapter.

2

Executive Control Theories of ADHD

The diverse set of symptoms observed in ADHD patients, including hyperactivity, impulsivity, and distractability, has led theorists to consider a deficit in executive (control) functioning as the root cause for ADHD. As mentioned in the previous chapter, executive functioning refers to a broad range of higher cognitive functions such as planning, inhibition, integration of information, and task switching/attention. Evidence for deficits on tasks measuring these aspects of cognitive function in ADHD patients is diverse and fairly well-documented.

2.1 Executive functioning and ADHD

Numerous studies of impairments in executive functioning in ADHD patients have been conducted. Almost exclusively these studies find definite impairments on measures of executive (control) functioning in patients with ADHD.

Reported impairments of executive functioning in ADHD patients

A synthesis of the results from three meta-analyses of studies of executive functioning deficits in ADHD patients, gives what appears to be a stable subset of impaired executive functions in patients with this disorder.

In a key study of executive functioning impairments in ADHD, Pennington and Ozonoff [1996] conducted a meta-analysis of 18 studies using measures of executive function administered to ADHD patients. These measures include tasks that test behavioral inhibition, task switching ability, sustained attention, and problem solving capacity (e.g. the Wisconsin Card Sorting Task and the Stroop task mentioned in the previous chapter). In 15 out of 18 studies a

significant difference between ADHD subjects and control subjects on one or more measures of executive functioning was found. Furthermore, on 40 of the 60 (67%) measures of executive functions used in total, performance of ADHD subjects was significantly worse than in the control group. By contrast, on none of the 60 measures did the ADHD subjects perform significantly better than the control subjects.

The most prominent effects on executive functioning are found in the domain of motor inhibition, and working memory and planning. Tasks that appear especially sensitive to ADHD are the Stroop task (inhibition) mentioned before, and Tower of London/Hanoi type tasks (planning; subjects have to arrange a number of colored balls/rings on three wooden pegs according to rules pertaining to the order of the balls/pegs). Tasks for pure motor inhibition such as the Go No-Go task and the Stop task (both tasks that require inhibiting a motor response) also show group differences between ADHD patients and controls.

In a similar but more recent analysis Sergeant, Geurts, and Oosterlaan [2002] found that in 23 out of 34 (67%) studies that measured various executive functions in patients with ADHD, performance of the ADHD patients was significantly impaired compared to performance of non-ADHD subjects. These results support those reported by Pennington and Ozonoff including the curious finding that ADHD subjects sometimes do, and sometimes do not show a significant impairment on the WCST. It is interesting to note that the WCST is a task on which frontally lesioned patients almost consistently show impaired performance.

These results are corroborated by a large meta-analysis conducted on 83 studies measuring executive functioning in ADHD patients [Willcutt, Doyle, Nigg, Faraone, and Pennington, 2005]. In 50–82% of studies, depending on the measure of executive functioning used, performance of ADHD patients was impaired compared to control subjects. To further investigate the prevalence of deficits in executive functioning in ADHD patients effect sizes¹ were computed for different measures across the 83 different studies. Of the 83 studies, in 29 studies a large effect size ($d \geq .8$) was reported. In an additional 33 studies a medium effect size ($.5 \leq d \leq .8$) was reported.

Comparing studies by category of administered task we find predominantly studies that measure inhibition and planning in which significant differences (at $p < .05$) between ADHD patients and control groups are reported. If the effect

¹The effect size (Cohen's d), or magnitude of the experimental manipulation, is calculated by dividing the difference between the means of experimental group and the control group by either of their standard deviations (if the variance is homogenous) or by their pooled standard deviation. Effect sizes are commonly defined as "small" if $d = .2$, "medium" if $d = .5$, and "large" if $d = .8$ [Cohen, 1988].

sizes reported above are averaged over categories of administered executive function, such as working memory, response inhibition, and planning, we find moderately high effect sizes ($.43 \leq d \leq .69$) with the highest values again within the domain of inhibition and planning.

Conclusion

Taken together these results strongly suggest an impairment in inhibitory control, planning (possibly included in, and related to, a working memory deficit) in ADHD patients. It is unclear how specific these results are to ADHD however, as they have been observed in frontally lesioned patients as well [Fuster, 1997]. Even if they are necessarily impaired in ADHD, they most probably do not reflect a sufficient cause of the disorder.

Two major problems plague the interpretation of these results: (1) most effect sizes of the measured impairments in executive functioning fall in the moderate range; and (2) it is unclear how specific the impairments found are for ADHD. Recall from the first chapter that ADHD is often accompanied by comorbid disorders such as Learning Disorder, Conduct Disorder and mood disorders. Also, it is known that executive functioning deficits accompany other pathologies such as frontal lesions.

These problems cast doubt on any theory that proposes a direct connection between a deficit in executive functioning and ADHD. Nonetheless, the finding of executive functioning impairments is reliable and stable enough that any theoretical account of ADHD should at least provide an explanation for the implied correlation between the two.

The next section discusses a theoretical account that does just that, Barkley's behavioral inhibition model [1997a; 1997b]. With this model in place the discussion can return to executive functioning deficits in ADHD, this time with the predictions that follow from Barkley's model in mind. Subsequently, in chapter three, the problems in interpreting the relation between executive functioning deficits and ADHD alluded to above, are discussed in more detail.

2.2 Barkley's behavioral inhibition model of ADHD

Among the most influential theoretical accounts of ADHD, Barkley's behavioral inhibition model of ADHD [1997a; 1997b] is an attempt to provide a unifying theory of ADHD by positing a core deficit in behavioral inhibition which is responsible for the observed behavioral and cognitive symptoms of this disorder. The model presumes that the essential impairment in ADHD is a deficit in response inhibition. This primary deficit leads to secondary deficits in abilities

of executive functioning which are dependent on inhibitory control for proper operation.

Behavioral inhibition, self-control, and executive functioning

Barkley defines behavioral inhibition as comprising of three interrelated processes: (1) inhibiting the initial prepotent response to an event; (2) stopping an ongoing response or response pattern, thereby permitting a delay in the decision to respond or continue responding; and (3) protecting this period of delay and the self-directed responses that occur within it from disruption by competing events and responses (interference control) [Barkley, 1997a, p. 47]. Thus behavioral inhibition does not directly cause executive or self-directed actions but rather facilitates their occurrence.

Self-control or self-directed behavior is defined as the responses by an individual directed at himself distinguished from the environmental events that initiate such an action. These actions are designed to alter the probability of subsequent behavioral responses by the individual (Barkley uses the example of rehearsing a telephone number: this is self-directed behavior because it is triggered by an external need, namely to remember a telephone number for later use, and rehearsing it increases the probability of subsequent recall).

Barkley distinguishes self-regulatory behaviors as a subset of self-directed behaviors. Self-regulatory behaviors are those behaviors intended to maximize the outcomes of responses over the long-term rather than the short-term. The benefits of long-term and short-term behavioral outcomes are often at odds, therefore it is important an individual learns to appreciate the value of long-term gains of behavior over short-term gains in cases where this is beneficial. If this is not learned the occurrence of self-control is unlikely.

The inherent delay involved in appreciating the long-term gains of behavioral responses requires cross-temporal organization of behavior, specifically a cross-temporal persistence of the link between behavior and its outcomes. This, in turn, requires a temporal sense, a sense for the passing of time. Such a capacity has been implicated in self-regulatory behavior before and has been linked to executive functioning and presumed neural correlates thereof in the frontal lobes [for example see Fuster, 1997, and also chapter one].

It is in this domain of self-regulatory functions that Barkley places the executive functions. Executive functions describe those self-directed actions that are used to self-regulate behavior. The occurrence of these actions depends on certain capacities often ascribed to executive functioning which make possible the cross-temporal integration of behavioral responses and their outcomes. However, these executive functions can only take place if external, as well as and

perhaps more importantly, internal interference can be inhibited sufficiently. It is quite often interference from competing behavioral patterns (or plans) which interferes with the decision to execute or delay execution of a certain behavioral response. Therefore it is vital that interference control can sufficiently inhibit competing internal influences in order for proper self-regulation of behavior to occur.

Defining executive functioning in this manner, it is perhaps unsurprising that certain authors prefer to use the term executive control instead of executive functioning. It appears that what Barkley means to emphasize about executive functioning is focused primarily on behavioral control. Executive control thus seems a more apt term for a theory of ADHD in which the proposed deficiency is mainly one of behavioral control.

The behavioral inhibition model of ADHD

Barkley's behavioral inhibition model of ADHD has six components: one behavioral inhibition component; four executive (control) function components; and one motor control component which can be seen as the (local) output of the system. Each of these components will be discussed in turn. The model is depicted in its totality in figure 2.1.

Behavioral inhibition

The first component of the model is behavioral inhibition. This component, inhibits, interrupts behavioral responses and protects goal-directed behavior from disruption by external and internal interference. This component of the model has a direct influence on motor control and indirect influence on motor control through four executive (control) functions: (1) nonverbal working memory; (2) verbal working memory; (3) self-regulation of affect, motivation and arousal; and (4) reconstitution.

Executive (control) functions

Nonverbal working memory Nonverbal working memory refers to the capacity to retain information across time (during delays in responding) and to manipulate this information online organizing it to perform complex sequences of behavior. Deficits in nonverbal working memory in patients with ADHD should manifest themselves as an inability to hold in mind relevant information and thus a diminished capacity to have and utilize internally represented information. This, in turn, leads to behavior that is more contextually driven than it is internally driven.

Verbal working memory Verbal working memory, by contrast, refers to the capacity to use self-directed (or internalized) speech to regulate behavior. The ability to follow and keep in mind instructions but also the ability to explicitly verbalize and generate rules to guide behavior, are an important part of this capacity (so-called rule-governed behavior). Self-reflection and self-questioning are also included in this capacity. This self-directed verbal behavior is hypothesized to be deficient in ADHD patients. So not only should there be an explicit delay of internalized speech in patients with ADHD compared to their peers, there should also be deficits in rule-governed behavior guided by this internalized speech. Together with the deficits mentioned under nonverbal working memory one would thus expect ADHD patients to exhibit less rule-governed behavior and more contingency-shaped (context-dependent) behavior than their peers. Also, internalizing a rule in the course of a rule-governed task should be absent or occur much less frequently and more slowly in patients with ADHD compared to their peers.

Self-regulation of affect/motivation/arousal Self-regulation of affect, motivation, and arousal refers to the internal motivation or drive that is necessary for behavior to occur in the absence of adequate external reinforcement. Persisting in a behavioral pattern to acquire long-term benefits often requires an emotional and motivational commitment that, in the absence of external reinforcement, has to be driven by internal factors. Emotional, motivational, and arousal-level regulation can help to preserve the proper incentive to persist in the desired behavioral pattern. Such deficits should be especially evident in ADHD patients and reflects their inability to motivate themselves and thus to bridge delays between reinforcers. Their behavior thus remains dependent on constant external and immediate reinforcement, more so than in non-ADHD subjects.

Reconstitution Reconstitution is the capacity to reorganize and re-sequence complex behavioral sequences. It allows the creation of novel behavioral structures in response to unfamiliar problems. The capacity to self-generate rules (which can later be verbalized through the verbal working memory component) follows from this capacity. Reorganizing behavioral sequences will almost always require a recombination of several simple behavioral sequences into a more complex one. Obviously some sequences of behavior are more instrumental in achieving a particular goal than others. Barkley proposes there is a syntax of behavior which guides this assembly of complex behavioral sequences from simpler ones. An important aspect of generating novel behavioral sequences has to do with their so-called fluency. Behavioral fluency refers to the capacity

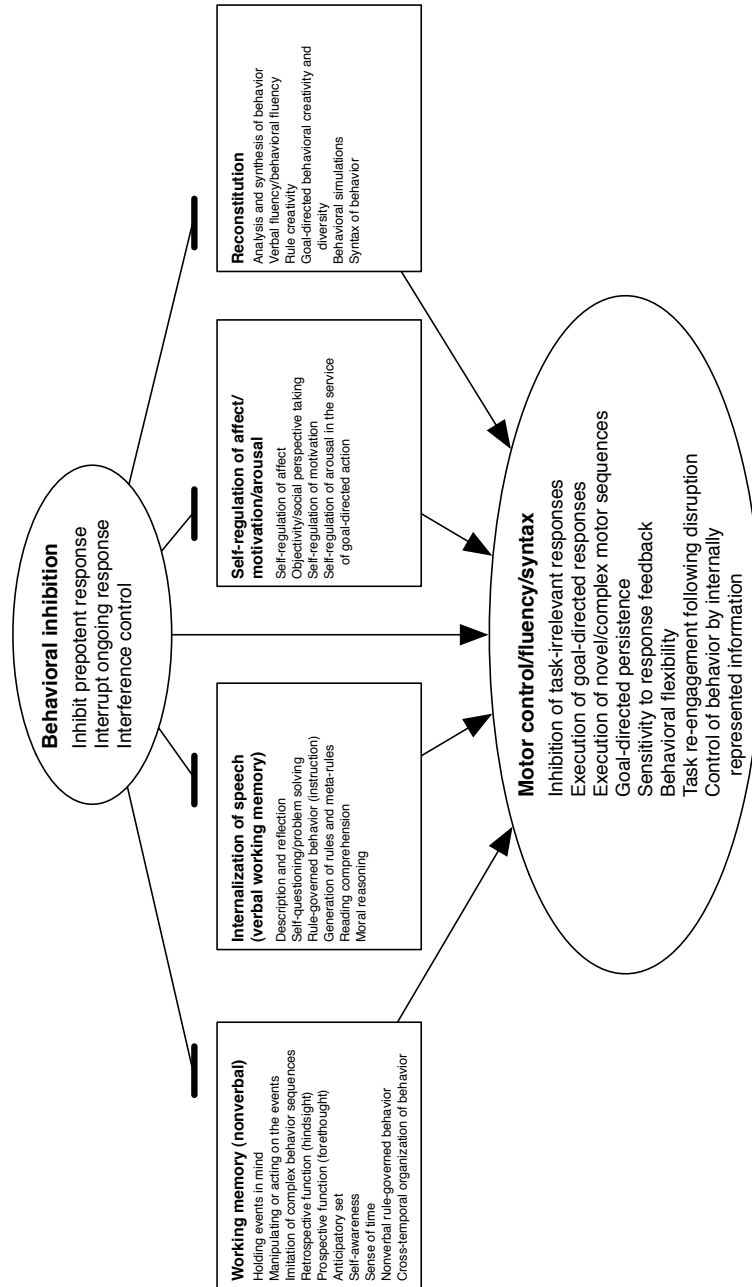


Figure 2.1: Behavioral inhibition and executive functioning model of ADHD [Adapted from Barkley, 1997a.]

to generate fluent, i.e. coherent and productive, novel behavioral sequences. Deficits in reconstitution should be evident in ADHD patients not only in the synthesis of speech but also in motor behavior, i.e. recombining old behaviors into novel behavioral sequences should be especially complex for patients with ADHD. These deficits should thus be especially noticeable in situations that demand novel and complex behavioral sequences in order to achieve a particular goal.

Motor control/fluency/syntax

The final component of the behavioral inhibition model represents the effect of behavioral inhibition and the four mentioned executive (control) functions on motor control. It thus represents the outcome of behavioral inhibition, self-regulation and behavioral responses. Fluency and syntax are part of this component as well but not at the level of (re)sequencing behavior but at the level of execution of (newly) synthesized behavioral sequences. These deficits should thus be evident in patients with ADHD as distinct from deficits of reconstitution but as deficits of motor execution. Motor behavior in ADHD patients is expected to be generally less inhibited or controlled by internal behavioral monitoring than in non-ADHD subjects. It is also expected to be more sensitive to immediate contextual influences and therefore more easily disrupted. Because of associated deficits in working memory and motivation goal-directed motor responses should occur less frequently and are more easily disrupted in patients with ADHD than in their non-ADHD peers. Behavioral performance should be especially affected by such disruptions in ADHD patients. Because of their sensitivity to contextual influences, working memory deficits, and decreased ability to self-motivate ADHD patients are less likely to restart a motor response following a disruption than non-ADHD patients. This sensitivity to disruption and its effects together with deficits associated with reconstitution (planning of behavioral sequences) should lead to a general disorganization of motor behavior observable in ADHD patients.

Behavioral activation and non-specific arousal

The model described in the previous section is not meant to be taken in isolation. Obviously it functions within the broader range of human cognitive capacities and, to a large extent, relies on cognitive abilities outside the scope of the model. External input into the components of the model is left unspecified, the capacity for language and utilization and evaluation of rules are taken for granted, and input-output relations between functions implicated in the model such as arousal, motivation and motor control are missing. It is obvious

then, that this model should not be taken as general model of human cognition, or even as a general model of human executive function. The model pertains to executive functioning insofar it is concerned with self-control, self-regulation and goal-directed behavior. It is at this level of cognitive processing where the deficits observed in ADHD patients are thought to arise.

Two relevant aspects of the model omitted from the description given in the previous paragraph are worth noting. As Barkley remarks, the model's mention of a behavioral inhibition system and linkage of self-control to drive and arousal begs the question where in the model a behavioral *activation* system and a *non-specific* arousal system should be incorporated.

It stands to reason that a system in which behavioral inhibition is a component, behavioral activation has a role to play as well. This behavioral activation component would represent behavioral activation through immediate and short-term reinforcement; precisely the activation the behavioral inhibition system would seek to inhibit.

Modification of drive and arousal require an interface with a system of non-specific arousal. Furthermore, it is natural to suppose a non-specific arousal system would be relevant to the functioning of the proposed model at all levels. Efficient and useful inhibition of behavior possibly mediated by executive (control) functions is obviously influenced by non-specific arousal or activation.

Barkley correctly raises and acknowledges these concerns. He remarks that while both a behavioral activation component and a non-specific arousal component are relevant to his behavioral inhibition model, they are not to be included in this model proper since they are not forms of self-directed behavior. Both components certainly facilitate self-directed behavior but are themselves not forms of it.

While these components should not be incorporated in the behavioral inhibition model per se, their relation to the model can be illustrated. Figure 2.2 depicts this relationship.

2.3 Predictions of the behavioral inhibition model

With the behavioral inhibition model of ADHD established, it is worthwhile to reexamine the deficits in executive (control) functioning observed in ADHD patients but now in light of the predictions that follow from this model. From each of the executive (control) functions mentioned in the model we can generate a set of predicted deficits that should be observable in patients with ADHD.

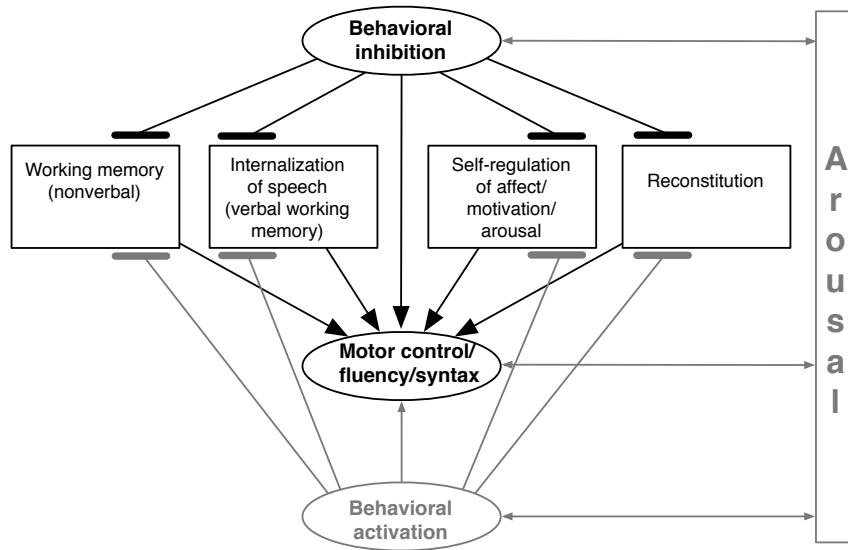


Figure 2.2: Relationship between the behavioral inhibition model (in black) and non-specific arousal and behavioral activation systems (in grey) [Adapted from Barkley, 1997a.]

Nonverbal working memory

Deficits of nonverbal working memory should be evident in patients with ADHD compared to control subjects. In experimental studies we would expect impaired performance on such tasks as the delayed-response task, the self-ordered pointing task, and tasks that measure counting and digit spans.

From the meta-analytical studies reviewed in the current paper, evidence for a specific nonverbal working memory deficit in ADHD is sparse. Studies of spatial working memory, digit span, self-ordered pointing show moderate impairments of ADHD patients compared to control subjects [Pennington and Ozonoff, 1996; Sergeant, Geurts, and Oosterlaan, 2002; Boonstra, Oosterlaan, and Buitelaar, 2005; Willcutt, Doyle, Nigg, Faraone, and Pennington, 2005].

Deficits of this nature have also been found in patients with Oppositional-Defiant disorder and Conduct disorder [Sergeant et al., 2002]. The comorbidity of these disorders with ADHD stressed earlier makes it that much more difficult to interpret these findings as deficits specific to ADHD.

Verbal working memory

Deficits in measures of verbal working memory have been found in ADHD patients albeit with moderate effect sizes [Sergeant et al., 2002; Boonstra et al., 2005]. As such, these findings would support the same conclusions as were

drawn for nonverbal working memory; the findings are relevant but neither striking nor do they appear to be specific to ADHD.

An interesting prediction that follows from Barkley's model is the prediction that there should be delayed internalization of speech in ADHD. The second executive (control) functioning component in the model is described as facilitating internal speech and verbalization of rules for self-questioning and reflection. Thus it may be that an impairment on traditional experimental tasks used to measure verbal working memory in ADHD patient's is more readily explained by a learning disorder or a non-specific impairment in academic achievement attributable to ADHD. This would plausibly explain the moderate but persistent deficits observed.

The case for a verbal working memory impairment, at least as defined Barkley as the internalization of speech, then might still be supported if a delay in internalization of speech can be demonstrated in ADHD patients. Barkley cites several studies that investigated the nature of self-directed speech in children with ADHD. Although here too, a learning disorder was a likely confounding influence, the results support the prediction that self-directed speech was less mature in children with ADHD compared to their non-ADHD peers. Overall the amount of self-directed speech does not differ significantly between groups. However, children with ADHD do use significantly more task-relevant externalized private speech (describing one's actions, self-directed comments etc.) and significantly less task-relevant inner-speech (evidenced by external manifestations such as mouthing of task-related words and inaudible muttering of task-related words) compared to their non-ADHD peers. These results support the prediction of the behavioral inhibition model that internalized speech is delayed in children with ADHD. If self-directed speech is taken, as it is in the model, as a significant contribution to self-regulating actions this provides a possible explanation for the impairments in self-regulation that are assumed to occur in ADHD patients within the context of the model.

Barkley relates a delay in self-directed speech to the ability to engage in rule-governed behavior. He presents some rather tentative evidence suggesting that children with ADHD are less able to resist forbidden temptations (in the context set by an experimenter) than their non-ADHD peers. This then, according to Barkley would support a deficit in the ability to engage in rule-governed behavior. A more parsimonious explanation however, is to interpret this result as direct evidence of a lack of behavioral inhibition (see figure 2.1 for the direct link between behavioral inhibition and motor control) rather than an impairment in the ability to follow rules. This explanation is even more likely when it is noted than on a task of implicit rule-following, the WCST,

there is no clear evidence of an impairment in ADHD patients [Pennington and Ozonoff, 1996; Willcutt et al., 2005].

Self-regulation of affect/motivation/arousal

The behavioral inhibition model predicts a lack of self-regulation of affect, motivation and arousal. For affect it is difficult to determine whether or not the model's predictions are correct in specific relation to ADHD as mood and anxiety disorders are often comorbid with ADHD (see figure 1.1).

Findings associated with lack of motivation, measured as decreased productivity on experimental tasks, are frequently reported in children with ADHD. These results are difficult to interpret however, since it is unclear whether the decreased productivity reflects a deficit regarding the task requirements or a lack of intrinsic motivation. Barkley offers some tentative evidence to support his deficient motivation hypothesis. Evidence to the contrary is given in Van Meel, Oosterlaan, Heslenfeld, and Sergeant [2005] where it was shown that ADHD children's impaired performance on a motor timing task was due to a generic motor timing deficit rather than motivational factors. Of course this does not entail that Barkley's assumption about the involvement of motivational factors in task persistence is unfounded. McInerney and Kerns [2002] examined time reproduction in children with ADHD in order to investigate if these children have a true deficit in their subjective sense of time or if their impaired performance reflects a motivational deficit. The authors report that increased motivation (manipulated by having a version of the task with extended reinforcement and one with regular reinforcement) did lead to significantly better performance for the ADHD children. In spite of this increase however, performance of ADHD patient was never better than control subject's performance, children with ADHD performed significantly worse than control children on both versions of the task. For control subjects an increase in performance resulting from increased motivation was not observed. Their performance was stable across conditions. Integrating these results suggests that the effect of motivation on task performance in children with ADHD is present yet subtle. This, in turn, suggests that the role of motivation in ADHD is not as prominent and specific as Barkley's model suggests but rather that it has a more general influence on performance [McInerney and Kerns, 2002].

In the case of self-regulation of arousal there is little support for the model to be reported. There is evidence from neuroimaging studies that brain activity is diminished in ADHD patients [Barkley, 1997a], specifically in the frontostriatal structures of the brain (lateral prefrontal cortex, dorsal anterior cingulate cortex, caudate, and putamen; roughly a brain circuit which includes the

prefrontal cortex and basal ganglia) [Bush et al., 2005]. As Barkley remarks, this reduced activity observed in those with ADHD in brain areas relevant to executive (control) functioning is indicative of impaired activation/arousal in these areas of brain. However, the connection between the proposed deficits in self-regulation of arousal is not plausibly inferred from these findings. This hypothesized component of the model thus remains tentative.

Reconstitution

In the domain of reconstitution measures of verbal fluency are reviewed as likely deficits in ADHD patients. A moderate deficit in letter and category fluency tasks is reported [Pennington and Ozonoff, 1996; Barkley, 1997a; Sergeant et al., 2002]. Sergeant et al. report similar deficits in studies of comorbid disorders such as oppositional-defiant disorder and conduct disorder. This again calls into question the specificity of reported findings for ADHD.

Barkley cites a number studies reporting impaired performance on the WCST as evidence for deficits in response flexibility, and thus of reconstitution, in ADHD. The combined findings of the meta-analyses in Pennington and Ozonoff [1996], Sergeant et al. [2002], and Willcutt et al. [2005] suggests only a moderate effect size of differences in WCST performance between ADHD patients and controls, however.

Taken together these findings do not seem to support a specific deficit of reconstitution in ADHD patients. It is more likely the observed findings reflect a general cognitive inefficiency related to other aspects of the disorder, such as lack of inhibition, and possibly disorders comorbid with ADHD.

Conclusion

Barkley's behavioral inhibition model [1997a] aims to give a unified account of ADHD in which the primary deficit is a lack of behavioral inhibition which results in deficits in executive (control) functioning and motor control and behavior.

Reported findings in the key domains of executive (control) functioning that Barkley defines, nonverbal working memory, verbal working memory, self-regulation of affect/motivation/arousal, and reconstitution, suggest significant yet moderate deficits in these domains are associated with ADHD. Furthermore, executive (control) functioning deficits of a similar nature are found in patients with disorders comorbid with ADHD such as, Oppositional-Defiant Disorder, Conduct Disorder, and Learning Disorder, as well as frontally lesioned patients.

This lack of universality and specificity of executive (control) functioning deficits in patients with ADHD raises serious doubts concerning the primacy executive (control) functioning receives in Barkley's behavioral inhibition model. Rather, the findings presented in this section suggest, as Willcutt et al. [2005] remark, that executive (control) functioning deficits are neither necessary nor sufficient causes of ADHD in all individuals with this disorder.

In the next chapter the merits of executive (control) functioning deficits in theorizing about ADHD are discussed in light of the findings present in this chapter. Possible implications for future research and theoretical accounts of ADHD are considered.

3**On the Merits of Executive Control Theories of ADHD**

In the previous chapter claims for the primacy of an executive control deficit in ADHD were evaluated in terms of a particular executive (control) functioning theory of ADHD [Barkley, 1997a]. It was found that support for executive (control) functioning deficits in ADHD is substantial but neither universal nor specific enough for such deficits to be regarded as primary and defining impairments in ADHD.

In the current chapter empirical findings concerning deficits of executive control in ADHD will be evaluated, not in terms of a theory which emphasizes a primary causal role of such deficits, but in terms of their general involvement in ADHD. A primary deficit of executive control in ADHD may not be supported by empirical findings, the specific weaknesses of executive control consistently associated with ADHD nonetheless do warrant an explanation.

Explanations for the observed weaknesses of executive control in ADHD have recently been suggested by models which emphasize the heterogeneous symptomology of ADHD. Such models emphasize the involvement of deficits in multiple neuropsychological mechanisms of cognition, motivation, and arousal, in accounting for the heterogeneous symptomology of ADHD. In these multiple-deficit models weaknesses in executive control are thus only one part of the complete spectrum of symptoms associated with ADHD. Two such models, the dual pathway model of ADHD [Sonuga-Barke, 2002, 2003] and the cognitive-energetic model of ADHD [Sergeant et al., 1999], are discussed at the end of this chapter.

3.1 Criteria for a primary executive control deficit in ADHD

In order to adequately evaluate the evidence presented in the previous chapter concerning executive control deficits in ADHD it is useful to posit criteria with which empirical findings can be evaluated. Willcutt et al. [2005] provide four such criteria which need to be met if an impairment in executive control is to be considered a primary deficit in ADHD:

1. Groups with ADHD must consistently exhibit deficits on measures of executive control. Some argue that such deficits should persist even if confounding variables such as, age, language, general intelligence, reading ability, and symptoms of other psychopathologies are controlled for. Willcutt et al. remark this last claim is much disputed, however.
2. Executive control deficits must account for a substantial proportion of the variance in ADHD symptoms in the population.
3. Executive control deficits must be present in most individuals with ADHD.
4. Executive control deficits and ADHD symptoms must be attributable to common etiologic influences; because ADHD is highly heritable one would expect executive control deficits to be coheritable with ADHD.

For the present discussion I will consider only the first two criteria. The third criterion is an extension of the first two and the fourth criterion is secondary to establishing a primacy of an executive control deficit in ADHD and hence should be discussed only if this primacy has in fact been established.

The first and second criteria basically require executive control deficits to be *specific* and *universal* to ADHD in relation to possible comorbid disorders. They also require executive control deficits to be *necessary* and *sufficient* causes of ADHD. Evidence presented in the previous chapter casts doubt on whether or not these requirements can be met. However, this evidence is fully dependent on the validity of its measures, i.e. on the validity of the measures of executive control. A conclusion about whether or not the requirements for executive control deficit as a cause of ADHD can be met is only as strong as the evidence for executive control deficits in ADHD patients. The latter is completely dependent on the validity of measures of executive control.

Pennington and Ozonoff [1996] raise important methodological points in this regard. One problem is that tasks measuring executive control are often complex. For example, the WCST is commonly thought of as a task that measures executive control, more specifically, working memory and rule-shifting

behavior. Unfortunately, for our purposes, the WCST most probably also measures several other cognitive abilities such as, knowledge of categories (number, shape and color) as well as a basic understanding of rule-following behavior, for example. This complexity of tasks that measure executive control is on the one hand required since such functions are cognitively complex functions. On the other hand the complexity also entails a lack of specificity which is difficult to reconcile with the specificity one would like to have in order to determine if a particular executive function is disturbed or not.

A related problem concerns the theoretical underspecification of many of the executive control constructs. As mentioned in the introduction, executive control is a commonly accepted and useful *general* theory. It is not very specific, however. This need not be detrimental to the theory in general, broad models can and do serve a useful, guiding purpose. It is problematic, however, when very specific claims and predictions are being made as is the case with executive control theories of ADHD.

Concerning task complexity and associated measurement issues, it should be noted that convergent performance (i.e. similar correct answers and errors are observed) on particular tasks can provide compelling evidence for similar functional deficits across subjects. On the WCST for example, it has been observed that frontal patients and children make similar perseveration errors, that is they persist in a previously learned, yet currently incorrect rule. Even if it is not completely clear which specific processing deficit underlies these errors, the convergent performance provides compelling support for a similar, and in this case frontal, deficit in processing (be that a deficit caused by immaturity, disturbed development, or injury) [Crone et al., 2004].

Thus, measures of executive functions, even though complex, can provide useful convergent support for a similar deficit or functional cognitive (sub)system impairment in different subject groups. This can be used to amend or revise hypotheses and direct and further specify ongoing research. These considerations also meet the objections of underspecification of executive constructs mentioned above, if the proper scope of the conclusions obtained from measuring such constructs is taken into account. That is, the conclusions can only be as specific as the measured executive control construct in question and its measurement are.

3.2 Applying the primary deficit criteria to ADHD

With these criteria and considerations in mind the results from the three meta-analyses discussed in section 2.1 [e.g. Pennington and Ozonoff, 1996; Sergeant et al., 2002; Willcutt et al., 2005] can be reevaluated. It was concluded there

that the results, interpreted in terms of Barkley's [1997a] behavioral inhibition model, were neither universal nor specific enough for a deficit of executive control to be regarded as the primary deficit in ADHD as Barkley claims.

In terms of the first two criteria from among the four put forth in the previous paragraph a similar picture arises. The first of the two criteria put forth in the previous paragraph appears to be met to a certain extent by the results reported in the four meta-analyses. That is, in a predominance of studies analyzed ADHD patients show impairments on measures of executive control compared to control subjects. Pennington and Ozonoff report that on 40 of the 60 (67%) measures of executive control used in the studies included in their meta-analysis ADHD patients performed significantly worse than control subjects. Similarly, Sergeant et al. report that in 23 out of 34 (67%) studies measuring executive control performance of ADHD patients was significantly worse than that of control subjects. Also, in an extensive analysis performed by Willcutt et al., it was found that in 50–82% of studies, depending on the measure of executive functioning used, performance of ADHD patients was impaired compared to control subjects. These results suggest a consistent, albeit moderately strong, association between ADHD and deficits of executive control.

Concerning the second criterion, the amount of variance accounted for by executive control deficits in ADHD, the effect sizes of group differences between ADHD patients and control subjects on measures of executive control reported by Willcutt et al. are worth mentioning. As discussed in section 2.1 in roughly a third of the studies included in their analysis a large effect size ($d \geq .8$) was found for differences between ADHD patients and controls on executive control tasks. In an additional third of the studies moderate effect sizes were found. If, however, these effect sizes from individual studies are averaged over categories of administered executive function, such as working memory, response inhibition, and planning, we find only moderate effect sizes ($.43 \leq d \leq .69$). This suggests that although executive control deficits are associated with ADHD, as the support for the first criterion proves, this association is not strong enough for the impairment to be considered a primary deficit in ADHD.

3.3 The nature of executive control deficits in ADHD

From the preceding sections it is clear that theoretical accounts which emphasize an impairment of executive control as the primary deficit in ADHD cannot hold. However, there is a rather consistent association between deficits of executive control and the occurrence of ADHD. Based on findings from the meta-analyses discussed in the previous section, the next section is about those

domains of executive control in which deficits associated with ADHD occur. Subsequently, the nature of the executive control deficits in ADHD is discussed.

Executive control deficits associated with ADHD

Patients with ADHD often exhibit certain weaknesses on measures of executive control. These weaknesses are not evenly distributed among the full range of executive functions. Table 3.1 lists four broad categories of executive (control) functioning domains on which ADHD patients often show impaired performance (with typical experimental measures used to test performance on each domain listed in parentheses). Reported in the right column of table 3.1 are the number of studies—from the total number of studies reported in the meta-analyses by Sergeant et al. [2002] and Willcutt et al. [2005]¹ for a particular executive control domain—that found a significant group difference between ADHD patients and non-ADHD subjects on the domain of executive control under consideration.

Unsurprisingly, considering the way ADHD is commonly understood, measures in the executive control domain of inhibition often find impaired performance for ADHD patients compared to non-ADHD subjects. In 87 % of studies measuring inhibition analyzed in Sergeant et al. and Willcutt et al., performance of ADHD patients was found to be significantly impaired compared to non-ADHD subjects. By contrast, only in 50 % of the studies analyzed in these meta-analyses an impaired performance was found in ADHD patients compared to non-ADHD patients for studies investigating set shifting. Both in the domains of planning and working memory there is a substantial, though less pronounced than for inhibition, impairment of performance in ADHD patients compared to non-ADHD subjects. In 69 % and 58 % of studies, respectively, an impairment is found.

Of course this interpretation is subject to the methodological caveats raised by Pennington and Ozonoff [1996] relating to task complexity and specificity and underspecification of executive control constructs. Also, as was reported in the previous section the association between measures of executive control in patients with ADHD reported is weak. Therefore, based on the results reported here and given the present state of theorizing about executive control, one cannot conclude that ADHD is primarily a disorder of executive control in general or specifically of inhibitory executive control and planning.

What can be concluded is precisely what the data suggest: deficits of executive control occur in some ADHD patients but not in others. That is, deficits of

¹Duplicate studies are only considered once; results reported by Pennington and Ozonoff [1996] are left out because the studies these authors report are all included in Sergeant et al. and Willcutt et al.

Table 3.1: Domains of executive control impaired in ADHD

Domain (measures)	Number of studies that found a significant group difference
<i>Set shifting</i> (Wisconsin Card Sorting Task)	22/44 (50 %)
<i>Working memory</i> (Delayed response task)	17/12 (58 %)
<i>Planning</i> (Tower of London, Tower of Hanoi)	16/28 (69 %)
<i>Inhibition</i> (Stroop task, Go-NoGo task)	47/54 (87 %)

executive control, instead of being an explanation for the symptoms of ADHD, are to *be explained* by a theoretical account of this disorder.

Multiple-deficit models of ADHD

The occurrence of deficits of executive control in some cases of ADHD and the absence of them in other cases has lead some researchers to propose ADHD is a heterogeneous psychopathology instead of a single-deficit disorder. This is a radical change from previous thought on ADHD and marks a departure from simple causal models of ADHD. According to this recent interpretation multifaceted models of ADHD are necessary to account for the cognitive as well as motivational deficits observed in this disorder.

Two prominent and distinct approaches attempting to account for the proposed heterogeneity of ADHD have emerged [Willcutt et al., 2005]. On the hand there is the so-called dual (developmental) pathway model of ADHD [see Sonuga-Barke, 2005, for an overview an appraisal], which posits that ADHD is a heterogeneous disorder with discrete neuropsychological subtypes that arise from dysfunction in substrates of distinct psychopathophysiological pathways. On the other hand there is the Cognitive-Energetic Model of ADHD which posits that the heterogeneity of this disorder arises from additive or interactive effects of dysfunction at different levels of information processing [see Sergeant, 2005, for an overview and appraisal]. Each of these models is discussed in turn.

The dual pathway model of ADHD

The dual (developmental) pathway model [Sonuga-Barke, 2002, 2003] proposes that ADHD is the developmental outcome of disturbances in two independent pathways or neural circuits, namely an executive and a motivational/reward circuit (see figure 3.1). The rationale for this model comes from the common observation that ADHD patients have deficiencies in inattention/overactivity (related to maintaining attention in case of delayed reward, and motivation) and executive control (predominantly inhibition), and that these deficits account for the diverse set of symptoms observed in ADHD.

In the first pathway ADHD is characterized as predominantly a disorder of motivational style mediated by the emergence of so-called delay aversion during childhood. The delay aversion hypothesis states that a negative emotional reaction to imposed delays (of reward) is a developmental consequence of the failure of an impulsive child to cope effectively with delays (of reward). Delay aversion manifests itself behaviorally as either delay avoidance, and thus choosing settings or behaviors that provide immediate rewards (impulsiveness), or, if delay cannot be avoided, delay time reduction, whereby the delay time is subjectively reduced by acting on the current environment to make it more interesting and engaging (inattention/overactivity).

Emergence of delay aversion during childhood is hypothesized to proceed through five steps. First, impaired neural circuitry involved in the signaling of delayed reward leads to impulsiveness. Second, impulsiveness leads to failures in coping effectively with delays. Third, the failure to cope with delays leads to negative/punitive responses from parents, teachers etc., which in turn leads to a negative emotional reaction associated with delays, i.e. delay aversion. Fourth, the failure to cope with delays also leads a lack of experience with delays and thus a lack of development of the necessary skills to facilitate coping with delays. Finally, the impulsiveness and lack of effective skills to handle delays associated with delay aversion, over time reinforce a pattern of behavior in which delay aversion is persistent and causes significant impairments in everyday behavior and self-organizational skills as associated with ADHD.

In the second pathway ADHD is characterized as predominantly as a disorder of executive control and inhibition. Impairments associated with inhibitory deficits are thought to arise along the same lines as motivationally-based impairments. In this case a disturbance in executive neural circuitry leads to inhibitory deficits. Again negative/punitive responses from parents, teachers etc., may lead to ‘executive-task aversion’, which in turn could lead to avoidance of such tasks and settings which require executive control. Also, failure on executive tasks may reduce the extent in which these tasks are intrinsically

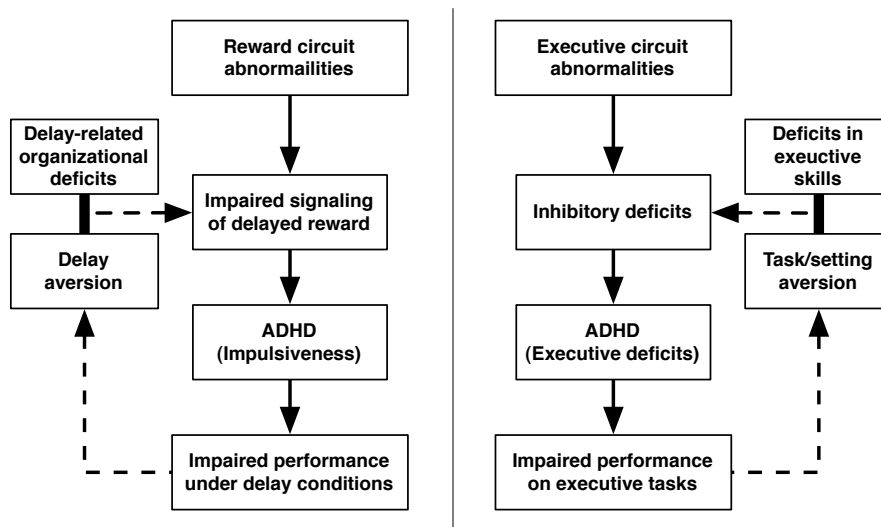


Figure 3.1: The dual pathway model of ADHD. On the left the developmental influences of the motivational/reward pathway on ADHD. On the right the developmental influences of the cognitive/executive pathway on ADHD. [Adapted from Sonuga-Barke, 2003, 2005.]

motivating. This may lead to decreased task motivation and effort adding to the ongoing process of avoidance of executive tasks and settings. Avoidance of executive task and settings because of one or both these processes leads to a lack of necessary exposure to such tasks in order to form effective executive skills. This perpetuates the neurologically based problems of executive control already present and thus facilitates emergence of the apparent lack of executive skills in ADHD patients.

The cognitive-energetic model of ADHD

The cognitive-energetic model [Sergeant et al., 1999] characterizes ADHD as a lack of efficiency in information processing determined by the interaction between three processing levels: computational mechanisms of attention, state factors, and management/executive function (see figure 3.2). The model encompasses both top-down and bottom-up processes and posits that ADHD is associated with defects in these processes at all three levels of the model.

The first level of the model, computational mechanisms of attention, comprises the following four stages: encoding, search, decision, and motor organization. These stages are associated with basic stimulus processing.

The second level of the model, state factors, comprises three distinct energetic pools: effort, arousal, and activation. Effort is defined as the energy

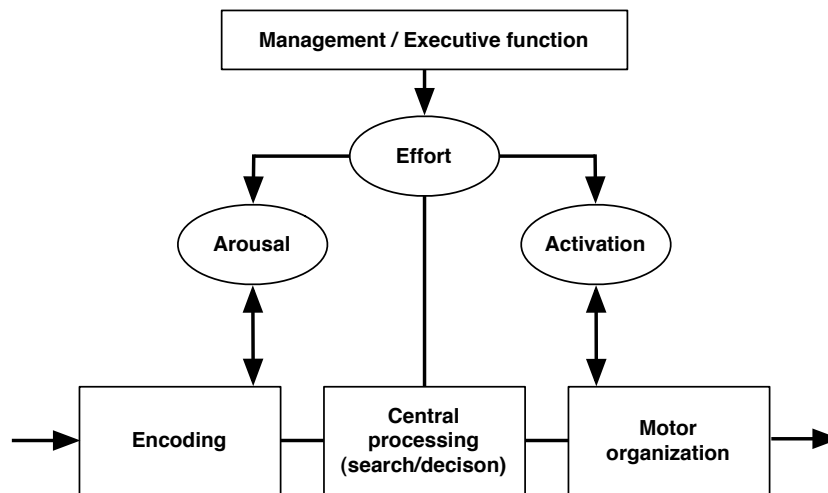


Figure 3.2: The cognitive-energetic model of ADHD. The upper level contains the executive control system. The middle level contains three distinct energetic pools: effort, arousal, and activation. The lower level contains attentional mechanisms for encoding, central processing, and motor organization. [Adapted from Sergeant, 2005.]

necessary to meet task demands. It is required when the current state of the organism does not match the task demands and is thus affected by cognitive load. It is also linked to motivation. Arousal is defined as phasic responding and occurs simultaneously with stimulus processing. It is typically influenced by stimulus intensity and novelty. Activation, finally, is defined as the tonic physiological readiness to respond and is typically affected by preparation, alertness, time of day, and task-relevant processing time.

The third level, management/executive function, is defined as planning, monitoring, detection of errors, and error correction. This is the level where overseeing of basic processing takes place. The inhibitory deficits associated with ADHD are thought to arise from possible defects at all three levels of the model, but predominantly from the energetic (second) level and the cognitive (third) level. Deficits in motor organization observed in ADHD [also mentioned by Barkley, 1997a, and discussed in brief in chapter 2] are thought to arise from defects in first level of the model, that of computational mechanisms of attention.

Conclusion

The central purpose of this chapter has been to evaluate the empirical findings concerning executive control deficits in patients with ADHD. It was concluded that support for a primary executive control deficit is not supported by em-

pirical studies investigating this matter, neither in a preconceived theoretical model [i.e. Barkley, 1997a], nor in an analysis based primarily on experimental tasks known to tap into executive control functions.

Nevertheless, the persistent finding of associations between executive control deficits and ADHD warrants an explanation. Two recent theoretical proposals were reviewed in which ADHD is not regarded as a single deficit disorder (with a primary executive control deficit), but as a multiple deficit, heterogeneous disorder in which deficits in the cognitive, attentional, and motivational domain all contribute to the diverse symptomology of ADHD.

In the dual pathway model [Sonuga-Barke, 2002, 2003] two distinct developmental pathways are proposed to be involved in ADHD. Disturbances in pathways that mediate delayed reward appraisal and executive control lead to disrupted behavior in tasks that require coping with delayed rewards or executive control. Negative internal (task aversion and avoidance) and external (negative/punitive responses from others) factors perpetuate these disturbed behavioral patterns and further reinforce the symptoms characteristic of ADHD, impulsiveness and lack of inhibitory control.

In the cognitive-energetic model [Sergeant et al., 1999] disturbances at three levels of information processing are thought to lie at the heart of the symptoms of ADHD. At the first level, that of computational mechanisms of attention and motor organization, the lack of motor behavior organization symptomatic of ADHD is proposed to originate. At the second and third levels, state factors such as effort/arousal/activation, and executive management respectively, the lack of inhibitory control characteristic of ADHD is thought to originate.

Although different with regards to certain assumptions and structural organization connecting the causes and symptoms of ADHD, the dual pathway model and the cognitive-energetic model both recognize and attempt to explain the multifaceted symptomology of this disorder. This marks an important shift in theorizing about ADHD, one in which the role of executive control in this complex disorder is neither under- nor overemphasized.

4

Conclusion

The diverse set of symptoms observed in ADHD, most importantly deficits of inhibition and impaired performance on certain experimental measures of executive (control) functioning, have led some to propose that this disorder essentially consists of a primary deficit in executive control of behavior [e.g. Barkley, 1997a,b].

The prevalence and persistence of ADHD—it is thought to affected a sizeable number of school-age children [3–10%; Barkley, 1997a,b; Faraone et al., 2003] and persist into adulthood in a substantial number of these cases [15–65%, depending on if DSM-IV criteria for full or partial remission are used; Faraone et al., 2006]—provide a strong incentive to search for a theoretical account of this disorder. Such an account, if supported by empirical findings, may very well prove useful in evaluating and amending the diagnostic criteria for ADHD currently in use [DSM-IV-TR American Psychiatric Association, 2000].

The suggested executive control deficit in ADHD patients is supported by empirical evidence from tasks that are thought to measure a number of executive functions, predominantly in the domain of response inhibition and planning. The main proponent of an executive control theory of ADHD is Barkley [1997a,b]. In his behavioral inhibition model of ADHD Barkley posits that a deficit in behavioral (response) inhibition disturbs the capacity for self-control and self-directed behavior aimed at modifying and correcting ongoing behavioral responses. This inhibitory deficit then leads to the observed impaired performance of ADHD patients on experimental measures of executive functioning and impaired motor control (manifested by over-activity).

The interpretation of the empirical findings supporting an executive control deficit in patients with ADHD is hampered by the lack of universality and specificity of these findings. In a review of a large number of studies investigating executive functioning deficits in ADHD patients [Pennington and

Ozonoff, 1996; Sergeant et al., 2002; Willcutt et al., 2005], performance of this group was found to be impaired compared to the performance of non-ADHD subjects on particular measures of executive functioning. However, these findings are not universal among ADHD patients, i.e. not all studies find impaired performance for patients with ADHD, and furthermore they lack specificity concerning ADHD, i.e. similar findings are reported for patients with pathologies distinct from ADHD although some of these pathologies are known to be comorbid with ADHD.

These findings support the conclusion that there is a moderately strong connection between ADHD and a deficit of executive control. This connection is too weak, however, to support an account in which a deficit of executive control is regarded as a primary factor in the symptomology of ADHD. Thus Barkley's behavioral inhibition model [1997a] is not supported by the empirical findings reviewed in the current paper.

On the other hand, the connection between an executive control deficit and ADHD is substantial enough to warrant an account of how such a deficit arises in patients with ADHD in the cases where it in fact does arise. This is an important conclusion since it places empirically motivated constraints on any theoretical account of ADHD. Note that these constraints do not require parting with the view that ADHD is a unitary and distinct psychopathological disorder. That is, none of the above requires radically distinct theoretical accounts of ADHD for individuals who appear to have a deficit in executive control in combination with ADHD symptoms and those who do not appear to have a deficit in executive control in combination with ADHD. What is required is a theoretical model that allows and accounts for the heterogeneous symptomology of ADHD while maintaining a unitary account of the disorder. This means that explaining, and not merely accommodating, the heterogeneity of symptoms observed in ADHD should be a primary goal for any such model.

Acknowledging that ADHD is not a single-deficit disorder (such as the behavioral inhibition model [Barkley, 1997a] proposes) but a multiple-deficit disorder, marks a shift in theorizing about the underlying mechanisms of ADHD. Recent theoretical models of this disorder emphasize the interaction between multiple cognitive and motivational neuropsychological mechanisms, throughout development as well as with regards to current efficiency of information processing and behavioral responding, in accounting for the diverse symptomology of ADHD.

Two such models, the dual pathway model of ADHD [Sonuga-Barke, 2002, 2003] (in which ADHD is proposed to arise from a developmental interaction between disturbances in two neural circuits, an executive and motivational/reward circuit) and the cognitive-energetic model of ADHD [Sergeant

et al., 1999] (in which ADHD is proposed to arise from additive or interactive dysfunctions at different levels of information processing), were discussed in chapter 3 as possible implementations of a multiple-deficit approach to ADHD. Since these models are fairly recent direct empirical support is still lacking and thus further research is necessary to establish their validity [Sonuga-Barke, 2005; Sergeant, 2005].

Note that in both these models (for a full description see section 3 of chapter 3) a system or neural mechanism for executive control is included. This underlines the importance of such a system for a complete account of the disturbances associated with ADHD. In no way, however, is this executive system proposed to be primary in accounting for the deficits found in ADHD. This is in marked contrast with Barkley's behavioral inhibition model [1997a] in which an executive system is the primary component of the model only secondarily connected to a system of arousal regulation (see figure 2.2). From the studies reviewed in the current paper it can be concluded that such an emphasis is unwarranted in the case of ADHD. Rather, it is likely the emphasis on interactions between systems in multiple-deficit models of ADHD is nearer to the truth and will lead to more fruitful research and theorizing about this disorder.

In conclusion, it must be remarked that although the discussed multiple-deficit models of ADHD are still tentative, the assumption they rely on, namely that a theoretical model of ADHD should account for the heterogeneous symptomology of this disorder by positing multiple neuropsychological mechanisms in which deficits can arise, is sufficiently established [Willcutt et al., 2005; Sergeant et al., 2002]. The findings presented in the current paper suggest a deficit of executive control in ADHD should be considered as an important, although not primary, component of any such model. For both these reasons *single-deficit* executive control theories of ADHD do not appear to have much merit. As is evident from the present discussion however, this does not mean that the role of executive control in models of ADHD is played out. It merely means that its role is no longer that of a single leading character, but instead, consists in a more modest interaction with the other characters. For those who regard modesty as a virtue, I take this to be a favorable conclusion.

References

- AMERICAN PSYCHIATRIC ASSOCIATION [2000]. *Diagnostic and Statistical Manual of Mental Disorders*. Washington, DC. 4th edition, text revision.
- BARKLEY, R.A. [1997a]. *ADHD and the Nature of Self-Control*. The Guilford press, New York, NY.
- BARKLEY, R.A. [1997b]. Behavioral Inhibition, Sustained Attention, and Executive Function: Constructing a Unified Theory of ADHD. *Psychological Bulletin*, **121**:65–94.
- BIEDERMAN, J. [2005]. Attention-Deficit/Hyperactivity Disorder: A Selective Overview. *Biological Psychiatry*, **57**(11):1215–1220.
- BIEDERMAN, J., MICK, E., FARAONE, S.V., BRAATEN, E., DOYLE, A., & SPENCER, T. [2002]. Influence of Gender on Attention Deficit Hyperactivity Disorder in Children Referred to a Psychiatric Clinic. *American Journal of Psychiatry*, **159**:36–42.
- BOONSTRA, A.M., OOSTERLAAN, J., & BUITELAAR, J.K. [2005]. Executive Functioning in Adult ADHD: A Meta-Analytic Review. *Psychological Medicine*, **35**:1097–1108.
- BUSH, G., VALERA, E.M., & SEIDMAN, L.J. [2005]. Functional Neuroimaging of Attention-Deficit/Hyperactivity Disorder: A Review and Suggested Future Directions. *Biological Psychiatry*, **57**(11):1273–1284.
- COHEN, J. [1988]. *Statistical Power Analysis for the Behavioral Sciences*. Lawrence Erlbaum Associates, Hillsdale, NJ, 2nd edition.
- CRONE, E.A., RIDDERINKHOF, K.R., WORM, M., SOMSEN, R.J.M, & VAN DER MOLEN, M.W. [2004]. Switching Between Spatial Stimulus-Response Mappings: A Developmental Study of Cognitive Flexibility. *Developmental Science*, **7**(4):443–455.

- FARAONE, S.V., SERGEANT, J., GILLBERG, C., & BIEDERMAN, J. [2003]. The Worldwide Prevalence of ADHD: Is it an American Condition? *World Psychiatry*, **2**:104–113.
- FARAONE, S.V., BIEDERMAN, J., & MICK, E. [2006]. The Age-dependent Decline of Attention Deficit Hyperactivity Disorder: A Meta-analysis of Follow-up Studies. *Psychological Medicine*, **36**:159–165.
- FUSTER, J.M. [1997]. *The Prefrontal Cortex: Anatomy, Physiology and Neuropsychology of the Frontal Lobe*. Lippincott-Raven, New York, NY, third edition.
- LUCIANA, M. [2003]. The Neural and Functional Development of the Human Prefrontal Cortex. In DE HAAN, M., & JOHNSON, M.H., editors, *The Cognitive Neuroscience of Development*. Psychology Press, New York, NY.
- MCINERNEY, R.J., & KERNS, K.A. [2002]. Time Reproduction in Children with ADHD: Motivation Matters. *Child Neuropsychology*, **9**(2):91–108.
- NIGG, J.T. [2005]. Neuropsychologic Theory and Findings in Attention-Deficit/Hyperactivity Disorder: The State of the Field and Salient Challenges for the Coming Decade. *Biological Psychiatry*, **57**(11):1424–1435.
- PENNINGTON, B.F., & OZONOFF, S. [1996]. Executive Functions and Developmental Psychopathology. *Journal of Child Psychology and Psychiatry*, **37**: 51–87.
- SERGEANT, J., OOSTERLAAN, J., & VAN DER MEERE, J. [1999]. Information Processing and Energetic Factors in Attention-Deficit/Hyperactivity Disorder. In HERBERT, C., & HOGAN, A.E., editors, *Handbook of Disruptive Behavior Disorders*, pages 75–104. Kluwer Academic Publishers, Dordrecht, Netherlands.
- SERGEANT, J.A. [2005]. Modelling Attention-Deficit/Hyperactivity Disorder: A Critical Appraisal of the Cognitive-Energetic Model. *Biological Psychiatry*, **57**(11):1248–1255.
- SERGEANT, J.A., GEURTS, H., & OOSTERLAAN, J. [2002]. How Specific is a Deficit of Executive Functioning for Attention-Deficit/Hyperactivity Disorder? *Behavioural Brain Research*, **130**(3–28).
- SONUGA-BARKE, E.J.S. [2002]. Psychological Heterogeneity in AD/HD—A Dual Pathway Model of Behaviour and Cognition. *Behavioural Brain Research*, **130**:29–36.

- SONUGA-BARKE, E.J.S. [2003]. The Dual Pathway Model of AD/HD: An Elaboration of Neuro-developmental Characteristics. *Neuroscience and Behavioral Reviews*, **27**:593–604.
- SONUGA-BARKE, E.J.S. [2005]. Causal Models of Attention-Deficit/Hyperactivity Disorder: From Common Simple Deficits to Multiple Developmental Pathways. *Biological Psychiatry*, **57**(11):1231–1238.
- SPRICH, S., BIEDERMAN, J., CRAWFORD, M.H., MUNDY, E., & FARAONE, S.V. [2000]. Adoptive and Biological Families of Children and Adolescents with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry*, **39**(11):1432–1437.
- VAN MEEL, C.S., OOSTERLAAN, J., HESLENFELD, D.J., & SERGEANT, J.A. [2005]. Motivational Effects on Motor Timing in Attention-Deficit/Hyperactivity Disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, **44**(5):451–460.
- WILLCUTT, E.G., DOYLE, A.E., NIGG, J.T., FARAONE, S.T., & PENNINGTON, B.F. [2005]. Validity of the Executive Function Theory of Attention-Deficit/Hyperactivity Disorder: A Meta-Analytic Review. *Biological Psychiatry*, **57**(11):1336–1346.