

# Cutting Corners: Neuropsychological Research into the Energetics of ADHD

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## 1. Introduction

People with ADHD are often subject to normative judgments by others for not doing their best, or being lazy. At school, the teachers' urge that they should put more effort into school work, may often be phrased as a question of *will* rather than lacking *ability*. Figuratively they could be said to cut corners, in the sense that they often perform tasks the easiest way. Even though they may know the long term advantages of hard work or of performing dull tasks, they may nonetheless follow the desires of the moment. This deficit in volition or self control has been construed as an aspect of behavioural inhibition or executive functioning (EF) (Barkley, 1997) considered as a core deficit in ADHD. Executive functions exert a top top-down mediating role on other cognitive processes, such as attention, learning and motor function. An ongoing discussion is whether impaired top-down processing is sufficient to explain ADHD, or sufficient to explain the heterogeneity of ADHD symptomatology. For some time, most neurocognitive research has focused on EF functions, clearly showing that subjects with ADHD are deficient in such tasks. However, there are four problems with considering this EF impairment as the cause of disordered behaviour.

### 1.1 Problems with EF impairment as a core deficit of ADHD

First: Although the research following this line has shown unrefutable results, effect sizes are at best moderate and only account for a part of the diagnosis related variance (Banaschewski et al., 2004; Toplak et al., 2009).

The second problem is related to specificity: other neurocognitive disorders also display dysfunctions of EF while clearly displaying other behavioural problems (Salimpoor & Desrocher, 2006). Zelazo and Müller (2002) suggest that EF deficits are a common outcome of many different perturbations of the epigenetic process, rather than a cause. They suggest that deficits in planning and inhibition, often related to a dysfunctional dorsolateral prefrontal cortex (DL-PFC), may be secondary to a developmental older dysfunction in orbitofrontal cortex (OFC) in autism, while dysfunctions in regulations of emotions and social relations in ADHD often associated with OFC, is secondary to their dysfunctions in DL-PFC. The main point of interest to this chapter, is that EF dysfunction is considered a symptom rather than a mechanism. These symptoms may be caused by a

mixture of bottom-up and top-down processes or both types of processes can account for the same result.

The third problem related to the primacy of inhibition or EF dysfunction, is related to ADHD subgroups. Although described by parents and teachers as clearly different, many studies have failed to find EF differences between subjects with the combined and the inattentive subtype of ADHD (Geurts et al., 2005; Nigg et al, 2002), suggesting that other mechanisms may underlie this division.

The fourth problem is related to variability in reward contingencies. External immediate reward has been found to increase performance quality or even normalize otherwise deficient functioning among subjects with ADHD (Liddle et al. 2011; Luman et al, 2008) even though external structure and need for internal structuring i.e. EF demand, remains the same.

### **1.2 Bottom-up mechanisms in ADHD**

That ADHD can be caused by both bottom-up and top-down mechanisms are the central axiom of the Dual Pathway Model (Sonuga-Barke, 2002). Similarly to Barkley, it posits inhibitory dysregulation as one pathway leading to ADHD symptoms. The other pathway has to do with motivational style and is associated with alterations in reward mechanisms. According to this view, inattention, overactive and impulsive behaviour can be functional expressions of delay aversion. When faced with a choice between immediacy and delay, ADHD children will choose the former (Sonuga-Barke et al, 1992). When no choice is available, they will reduce the perception of time by engaging in task irrelevant or incompatible (hyperactive) behaviour or being inattentive. In this model the cognitive deficits such as impaired planning and working memory, are seen as secondary to the bottom-up effects of delay aversion for some subjects with ADHD. For other children with ADHD poor inhibitory control underlie much of the same symptoms. Sonuga-Barke (2002) considers the motivational and the regulation dysfunction as independent, thus the dual pathway. In a study of cognitive impairments in probands with ADHD and their relatives, Kuntsi et al. (2010) found indications of the existence of two familial distinctive patterns. Another study indicated the independence of three pathways, namely temporal processing as well as inhibitory control and delay aversion (Sonuga-Barke et al., 2010).

Recent research on default mode network (DMN) in ADHD gives support to the emphasis on bottom-up mechanisms in ADHD. DMN is a distributed brain system comprising medial prefrontal cortex and medial and lateral parietal regions that are anticorrelated with attentional networks. While attentional networks are activated by goal-directed behavior (Liddle et al., 2011), activity in the DMN is related to mindwandering and resting state. When performing a task, the DMN must be deactivated. Both fMRI and EEG studies have found impaired attenuation of the DMN from rest to mentally demanding tasks among subjects with ADHD (Helps et al., 2010; Liddle et al., 2011; Peterson et al., 2009).

The emphasis on heterogeneity of mechanisms giving rise to the symptoms of ADHD is less clear in the Cognitive Effort Model (CEM) compared to the Dual Pathway Model. Similarly to the Delay Aversion pathway, it emphasizes the impact of bottom up motivational processes (Sergeant et al., 1999). Sergeant et al. (2003) considers delay aversion and aberrant reward systems as one expression of deficient energetics. Sonuga-Barke et al.(2010) agrees that the two theories overlap, but that it is possible to deduct different predictions that can be tested in future research.

Sergeant et al. (2003) present the CEM model as the most comprehensive model, incorporating also the top-down processes emphasized by Barkley (1997) and by Pennington & Ozonoff (1996), and extending the view of energetics in larger detail. The model divides the energetics of ADHD into different resource pools, i.e. effort, activation and arousal (Sergeant, 2005).

This chapter will present clinical research on the energetics of ADHD. First, the neurobiological basis and the psychopharmacological evidence will be briefly outlined, before proceeding to our own clinical neuropsychological findings. The energetic resource pools are in this connection considered latent variables difficult to operationalize in ways that differentiate them from aspects of executive function. Based on our own previous research, we nevertheless think it is possible to deduct measures from neuropsychological examinations that can serve as approximate operationalizations, making it possible to extract their contributions to some of the problems affecting subjects with ADHD.

## 2. Energetic resources

The first level of CEM comprises different stages in information processing: Information must be detected and encoded, be subjected to some type of processing and responses must be organized. The three energetic resources modulate these processes. *Effort* is required whenever the current state of the organism does not match that required to perform the task (Sergeant, 2005). Effort encompasses factors such as motivation and response to contingencies. The claim that effort seems to be impaired in ADHD is based on findings of variability of performance. Under specific circumstances that increase intrinsic motivation or under high extrinsic reinforcement, subjects with ADHD increase their performance more than controls (Luman et al., 2007). If reinforcement and motivation contingencies rather than task complexity are decisive, the typical impaired performance of ADHD subjects on effortful executive function tasks, would better be described as impairment in effort than in executive function per se. The research on the DMN is relevant as a possible physiological mechanism, indicating that subjects with ADHD fail to down-regulate the resting state and up-regulate the necessary effort related activation necessary for effective performance.

The other energetic pool, *arousal* is related to the alerting effect of sensory activity. Signal intensity and novelty increases arousal. By the same token, decreased intensity of stimulation leads to falling arousal. Arousal is the time-locked phasic physiological response to input and is regulated from the frontolimbic forebrain and the by basal ganglia. The primary neurotransmitters are noradrenaline and serotonin (van der Meere, 2002). The third energetic resource pool, *activation*, is related with the tonic or long lasting physiological readiness to respond and is affected by task variables such as preparation, alertness, time of day and time on task (Sergeant, 2005). While stimulation increases arousal, activity increases activation. The primary neurotransmitters are dopamine and acetylcholine.

### 2.1 Neuropsychological operationalizations of energetic resources

An operationalization of effort allocation in behavioral or neuropsychological terms needs to refer to a task in which the subject can *choose* how much effort to put into the task, in the sense that the environmental constraints on task performance must be minimal. If it is too easy, we would not see any potential effect of a failure of effort allocation. If it can only be solved by putting in high effort, we will not know whether a failure to perform successfully was due to task complexity or the proposed deficit in state regulation or effort allocation.

In order to interpret an ADHD related deficit in terms of effort allocation, it is also necessary to avoid testing effort with measures of executive function or attention. Such tasks are generally effortful, but impaired performance is difficult to interpret as impairment in motivation and not in executive function per se. In a recent study applying Evoked Response Potentials (ERP) and Skin Conductance Level (SCL) Johnstone et al. (2010) tried to disentangle the effect of effort and EF. As in many other studies of neuropsychological function in ADHD, they contrasted the behavior and ERP/SCL of subjects with ADHD to controls on attention to congruent and incongruent stimuli. Withholding response to incongruent stimuli would require interference control and is considered effortful. However, intending to make a critical test of the predictions from CEM and the interference control model of Barkley, they reasoned that degrading stimuli both in congruent and incongruent stimuli presentations would make the task more effortful while not increasing demand for interference control. They found an effect of stimuli degradation on skin conductance level, indicating that degrading stimuli increased arousal level. Also ERP findings were interpreted as suggesting impaired resource allocation (i.e. effort) rather than interference control per se. However, contrary to what we found, they did not find any significant group effects on the performance level.

The third condition that must be met in designing a performance measure of effort, is that it must not be a test of general ability in disguise. In our own research on learning and memory in ADHD, to which I soon will turn, we think that we constructed measures of effort that satisfy these three criteria (Egeland et al., 2010).

Research on the effects of event-rates on continuous performance tests (CPTs) has shown that subjects with ADHD tend to be impaired with large inter-stimulus intervals (van der Meere, 2002). This has been interpreted as an effect of underactivation (Sonuga-Barke et al., 2010), but might as well be related to underarousal. It is however, difficult to discern the effect of stimulus intensity from activation effects as long as measures demanding motor responses are the dependent variable. Behaviorally we cannot register reactions to stimuli the subject have to attend to, but not respond to. The finding of Drechsler et al. (2005) that ADHD subjects responded slower to targets not preceded by a warning signal, indicates an effect of arousal rather than activation. In the same vein, Benikos and Johnstone (2009) found that the length of the interstimulus-interval not only had effect on performance to targets, but also to ERP responses to a warning signal *preceding* the target that was responded to.

In the author's opinion previous interpretations of CPT performance have not sufficiently differentiated between the input or output aspects of the information processing, i.e. between stimulus and response intensity. In older CPTs, stimulus intensity have either been fixed or not analyzed in standard scoring systems. In these so called "low stimulus to noise" tests the subjects are to watch stimuli appearing on the computer screen and respond to about 10 or 20 % of all stimuli. The Conners' Continuous Performance test (CCPT-2: Conners, 2002), however, has a high stimulus to noise ratio. Subjects are supposed to respond to *all* letters appearing on the screen, *except* to X's, which amount to 10 percent of the exposures. At the same time, the inter-stimulus intervals (ISI) vary between 1, 2 and 4 seconds. This makes it possible to differentiate between the phasic effects of high or low stimulus intensity while at the same time assess the long term activation effects, since the subject has to respond more actively than in the former generation of CPTs.

In their seminal paper differentiating different aspects of attention, Mirsky, Anthony, Duncan, Ahearn & Sheppard (1991) factor-analyzed results from a series of attention tests, including a low signal to noise CPT. They identified four sub-functions of attention. The scores from CPT loaded on what was termed a vigilance factor. Vigilance was defined as the attentional capacity to remain alert also when less stimulated, i.e. similarly to losing arousal, but was considered synonymous to sustained attention. As was the custom of the time, the sum of CPT omissions and commissions was analyzed with no analysis of time on task changes or changes after different interstimulus-intervals. Since the Conners's CPT offers a lot of subtle measures not available in the older CPTs we hypothesized that it would discern different patterns of attention deficit among different patient populations. Thus, we sat out to perform the new factor analysis to which we now will turn.

### 2.1.1 The factor structure of Conners' CPT-II.

CPTs are widely used in neuropsychological assessment of subjects with ADHD (Wasserstein, 2005) as well as in schizophrenia research. Many studies have shown that children (Root & Resnick, 2003) and adults (Hervey et al, 2004) with ADHD are impaired compared to normal controls on this type of test, but also other clinical groups show impairments. In fact, the lack of differences between clinical groups, represent a problem when using the test as part of an ADHD assessment. Studies have failed to find differences between clinical groups such as ADHD and reading disorder (McGee et al, 2000), ADHD and schizophrenia (Øie & Rund, 1999) and ADHD and internalizing disorders (Solanto et al, 2004). These studies have, however, analyzed overall measures of attention from the CPT, such as  $d'$ , i.e. signal detection that is derived from both omissions and commissions and have not taken into account whether the errors are performed initially or late in the test, or subsequent to short or long stimulus-intervals. The reason for not being able to differentiate between clinical groups may then not be a lack of reliability, but may rather represent an accurate description that all these groups also suffer from a deficit in attention. Although they fail in attention for different reasons. While hyperactivity and impulsivity are considered to mediate the attention deficit of subjects with ADHD (Epstein et al, 2003), subjects with schizophrenia may have an impairment initially focusing attention, but may profit from exercise (Egeland et al., 2003, Egeland et al., 2007). Finally, lack of effort or fatigue may underlie the attention deficit in depression (Egeland et al., 2003). It is reasonable that the different mechanisms underlying the attention deficit will also be reflected in different patterns of CPT performance. The factor-analysis described below (Egeland & Kowalik-Gran, 2010a) was performed on CCPT-II protocols from a mixed clinical sample of 376 adolescent and adult participants with either ADHD-C, ADHD-I, affective disorders, schizophrenia spectrum, mild mental retardation or mild neurocognitive disorder, nonverbal learning disorder, learning disorder, different mild psychological disorders and subjects using analgetics. A normal group was included as well. In a follow up study to validate the factors (Egeland & Kowalik-Gran, 2010b), hypotheses were formulated as to the how subjects with ADHD, schizophrenia, affective disorders, language disorders and brain injury should perform in order to consider them different sub-processes. As part of the validation procedure, correlations with other tests of attention were also computed.

The result of the factor analysis is presented in Table 1. The first factor was coined *focused attention* since it was an overall measure of being able to focus on the task, namely to

respond quickly whenever target stimuli appeared. Previously, studies had shown problems with focusing attention among all the analyzed patient groups, and thus it was expected that they could not be differentiated on this factor. It was also expected that this factor should correlate with other tests of controlled attention such as the Stroop Color-Word interference Test, the Trail Making Test and the Paced Auditory Serial Addition Test, but not with Digit span or Knox Cubes measuring more automatic attention span. These hypotheses were all confirmed.

	I Focus	II H/I	III Sustain	IV Vigilance	V Change in control
Variability <sup>1</sup>	<b>.870</b>	.086	.133	.194	-.010
Hit RT SE <sup>2</sup>	<b>.843</b>	.265	.145	.334	.056
Perseverations <sup>3</sup>	<b>.768</b>	.612	.019	-.093	-.033
Omissions <sup>4</sup>	<b>.747</b>	.255	.224	.058	.072
Commissions <sup>5</sup>	.335	<b>-.804</b>	.048	-.022	-.123
Hit RT <sup>6</sup>	.363	<b>.760</b>	.049	.251	.185
Response style <sup>7</sup>	.206	<b>.688</b>	.089	-.012	-.087
Block change SE <sup>8</sup>	.162	-.064	<b>.842</b>	.128	.020
Block change <sup>9</sup>	.068	.047	<b>.731</b>	.073	<b>.469</b>
$\Delta$ omissions <sup>10</sup>	-.137	-.131	<b>-.707</b>	.042	.245
Hit RT ISI <sup>11</sup>	.097	.021	.014	<b>.847</b>	.163
Hit RT ISI SE <sup>12</sup>	.135	.103	.099	<b>.842</b>	-.042
$\Delta$ commissions <sup>13</sup>	.197	.086	-.022	.088	<b>.904</b>
Eigenvalue	3.94	1.93	1.49	1.31	0.99
% variance explained	22.92	15.13	14.28	12.94	9.17

<sup>1</sup> Variability of standard errors i.e. a measure of within respondent change in consistency of reaction time. <sup>2</sup> Hit reaction time standard error (i.e. consistency of response time). <sup>3</sup> Perseverations: responses without preceding stimuli. <sup>4</sup> Omissions: missed targets, <sup>5</sup> Commissions: responses to non-target stimuli. <sup>6</sup> Hit reaction time. <sup>7</sup> Response style ( $\beta$ ): cautious response style aimed at minimization of commission errors, or impulsive style minimizing omission errors. <sup>8</sup> Hit reaction time standard error over time-blocks (change in consistency as the test progresses) <sup>9</sup> Block Change: the slope of change in reaction time over six time blocks. <sup>10</sup>  $\Delta$  omissions: Changes in omissions over time (numbers of omissions in last third of test subtracted from first third). <sup>11</sup> Hit reaction time ISI: decrease in reaction time with longer interstimulus-interval. <sup>12</sup> Hit RT ISI SE: Standard Error of Hit RT ISI, i.e. whether reaction time becomes more variable with longer interstimulus intervals). <sup>13</sup>  $\Delta$  commissions: Changes in commissions over time.

Table 1. **Factor structure of Conner's CPT** (from Egeland & Kovalic-Gran, 2010a)

The next factor was termed *hyperactivity-impulsivity* (H/I) since it received loadings from commissions and reaction time. To validate such a term for this factor, we would expect that only the ADHD-C group would perform impaired. None of the other groups were expected to have a H/I problem. To qualify as a measure of H/I scores it should also not correlate with any of the other tests of attention. Also these hypotheses were confirmed.

The remaining three factors measured attributes of attention that had to do with changes over time or stimulus contingency. The two block-change-measures, as well as  $\Delta$  omissions (the difference between number of omissions in the first and the last third of the test), were

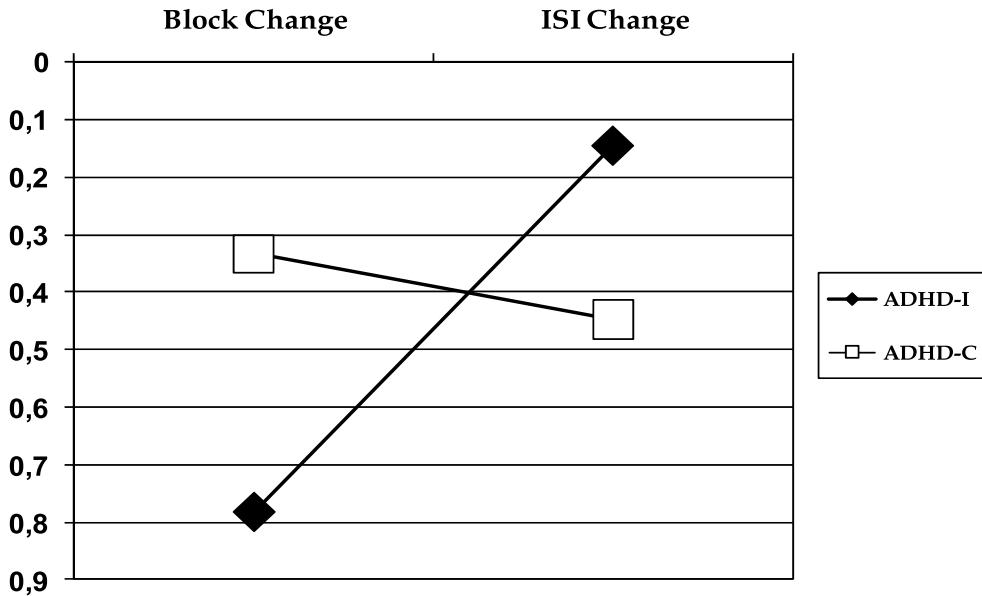
named *sustained attention*. The ADHD-I group scored below all other groups on this measure. Differential validity came from the finding that the factor did not correlate with any other attention test intended to measure other aspects than sustaining attention per se. The two ISI measures (changes in reaction time or increased variability of reaction time as a function of increased interstimulusinterval) loaded on the fourth factor named *vigilance*. Contrary to expectations, there was only a non-significant tendency for the ADHD-C subjects to score below the normal group. The fifth factor received a high loading from  $\Delta$  commissions, and a mediocre loading from block change. The factor seemed then to measure whether subjects became more impulsive as a function of time on task, and it was thus termed *change in control*. Both ADHD- groups scored below the normal group as well as the schizophrenia spectrum group and subjects using analgetics. That also subjects with ADHD-I lost control was contrary to expectations, but as a change measure, it could be due to different initial level of commissions. Interestingly, since previous studies had shown difficulties to differentiate between subjects with LD and ADHD when using overall measures, this process measure showed that LD subjects gained control, whereas ADHD-I subjects lost control. This is reasonable if the LD subjects had commission errors due to difficulties with letter differentiation, whereas commission errors reflect impulsivity among ADHD subjects.

Overall, the study showed that different mechanisms mediate the attention deficit in different groups, and that H/I is specific to ADHD-C, while impaired sustained attention is specific to ADHD-I. It also differentiated sustained attention from vigilance, thus possibly giving the clinician a tool to distinguish between arousal and activation.

### 2.1.2 Differentiating arousal and activation

This was examined further in a study of Conners' CPT performance comparing children and adolescents with ADHD-C and ADHD-I and healthy controls (Egeland et al., 2009). Sixty-five healthy controls and 67 subjects with ADHD between nine and 16 years of age participated in the study. The ADHD-I group performed below control children on Hit Reaction Time Block Change, considered to measure sustained attention. The ADHD-C group scored below controls on Hit Reaction Time Inter-Stimulus-Interval, considered to measure vigilance.

As illustrated in Figure 1, comparison of the two clinical groups showed a test by group interaction, with ADHD-I subjects performing below ADHD-C subjects with regard to sustained attention and above ADHD-C subjects with regard to vigilance. Sustained attention on the CCPT correlated specifically with parent and teacher ratings of inattention, but not with ratings of H/I, while vigilance correlated with all symptom ratings. Although correspondence between general findings of attention deficit in neuropsychological laboratory tests and daily life ratings tend to be significant but mediocre (Toplak et al, 2009), it is seldom that specific measures intended to measure underlying mechanisms mediating the deficit, also correlate with daily life ratings. In this case, the measure possibly reflecting insufficient activation correlated only with inattention scores and not with hyperactivity. However, the measure intended to quantify low arousal correlated also with H/I. This could be an indication that H/I is a way for the ADHD-C subjects to compensate for low arousal, but that prolonged activation leads to the fatigue more typical of the ADHD-I subjects. Contrary to the expectation from Barkley's interference control model, numbers of commissions neither differentiated between the ADHD subgroups nor between ADHD and healthy controls in this



Effect size in z-scores derived from age matched healthy controls

Fig. 1. **Double dissociation between presumable arousal-mediated vigilance (ISI Change) and sustained attention in ADHD-C and ADHD-I** (from Egeland et al., 2009)

study. However, Barkley et al. (2001) also used the CCPT-II and found no group difference between ADHD-C subjects and healthy controls with regard to commission errors. They commented that their results contradicted previous findings and could have to do with the type of CPT applied. That the difference between low and high signal to noise CPTs alter what was previously considered a main finding regarding ADHD performance on CPTs testifies to the moderate and variable effect size of this EF measure (Banaschewski et al., 2004; Toplak et al., 2009).

### 2.1.3 The impact of effort

The next study that will be discussed here concerns the effort construct (Egeland et al., 2010). As mentioned previously, to differentiate the bottom-up process of allocating effort to difficult tasks, from the top-down process of controlling attention, one has to construct effort measures that are not at the same time measures of executive function. Our point of departure was the observation that subjects with ADHD underperform at school (Ek et al., 2011). In formal tests of memory, they tend to be impaired in free recall, but often not impaired to the same degree in recognition memory. How could this be explained? If they are generally inattentive, how could they then be able to recognize items that they not had attended to in the first place? Could it be that encoding of new information was insufficient due to insufficient effort rather than inattention? All subjects were tested with a verbal list learning test. Four measures were computed that were considered to demand effort, without placing an equivalent load on executive functions (i.e. not demanding flexibility, inhibition, willful focusing on some information and ignoring other). The four measures were:



*Semantic organization of recall:* This was a measure of whether the subjects organized the haphazardly presented learning list in a thematically organized way when reporting back what they remembered. Such organization requires elaborative encoding considered to be specifically effortful (Hasher & Zacks, 1979).

*Buildup of proactive interference (PI):* PI refers to a normal process in which previous learning interferes with new learning. It is the price one pays for deep level effortful encoding.

*Degree of retroactive interference (RI):* RI refers to the phenomenon that new learning interferes with effective retrieval of previous learning. While lack of effective learning would prevent the build-up of PI, the same low-effort learning would be expected to increase the potential for retroactive interference. While PI depends on the original learning material still being remembered, RI is in fact forgetting due to new learning overriding the effect of old learning.

*Overriding the primacy and recency effect:* When presented with a learning list, the easiest items to remember are the first and last ones. They are often remembered “for free” or automatically while remembering the middle items demand some kind of organization of the stimuli, again considered to be an effortful process.

The study showed that the ADHD-C subjects differed from the healthy controls on all four effort indices, while the ADHD-I group differed on three of them, but showed a normal proactive interference. The effects were significant even when controlling for IQ that otherwise is related to effective learning strategies. Most effects were significant also when analyzing only ADHD subjects with no comorbid conduct or oppositional defiant disorder (CD & ODD). As in most other studies, the ADHD groups were impaired in free delayed recall, which of course is most important for school learning. Regression analyses of the explanatory power of the effort measures on such delayed recall, showed that they accounted for 39 % of the variance in the ADHD-I/control-analysis, and 35 % in the analysis of the ADHD-C and controls. When IQ was entered into the regression analysis after the four effort indices, the total variance explained rose to 45 % in the ADHD-I group. When entering diagnosis after the effort-indices, the total explained variance increased only marginally. In fact diagnosis did not contribute significantly beyond the effect of effort. Such statistical analyses do not disclose causative effects, but they show that impaired effort could be a sufficient explanation of impaired learning among subjects with ADHD.

#### **2.1.4 Effort and arousal effects on motor performance**

In the last study that will be discussed here, we tried to integrate both effort and arousal variables as possibly underlying motor impairment in ADHD (Egeland et al., 2011). Impaired motor function was one of the criteria for diagnosing Minimal Brain Dysfunction (MBD) which was a precursor of the present ADHD-diagnosis. Although deficits in writing, drawing and fine motor function still is considered typical of ADHD, the prevailing view today is that such impairments are merely secondary to the core ADHD symptoms, be that impulsivity/hyperactivity or energetics. ADHD subjects cut corners when drawing squares not because they lack motor skills, but because of a central processing deficit, i.e. a deficit that transcends the motor domain.

The same subjects that participated in the effort-study, also took part in this study. The subjects were tested with the Visual Motor Integration test (VMI: Beery, 1997) in which they

were required to draw 27 drawings as neatly as possible. Difficulties with this, which is typically found (Geurts et al, 2005), could be due to lacking motor function or to not allocating sufficient effort to the task. The reason for this, was that they themselves could choose how long time they would spend on the test and decide on their own level of accuracy within the limits of their capabilities. If motor problems mediated impaired performance, VMI performance should correlate primarily with other tests of motor function where energetics is not probable to influence results. Fingertapping speed and dexterity measured in a pegboard test were chosen as such tasks. If energetics as an example of a central processing deficit influenced VMI performance, then performance on this test should correlate with effort measures and arousal even when these measures are derived from a completely different behavioral domain. A summed effort measure was computed from the four separate effort measures from the previously cited memory function study (Egeland et al., 2010) and the interstimulus-interval effect from Conners' CPT study was used as the arousal-measure.

The results of partial correlations controlling for age and IQ showed that the simple motor tests did not correlate with any of the energetic-measures, indicating that they represented different sources of variance. Despite this, both energetics-measures correlated significantly with VMI performance in the ADHD-C group as did the simple motor tests. In the ADHD-I group the correlation between energetics and VMI performance was close to zero. This was interpreted to mean that when subjects with ADHD-C are more impaired in a complex fine motor test such as VMI, this is due to an impairment both in simple fine motor control and energetics, while only impaired fine motor control contributed to the impairment in ADHD-I.

### **2.1.5 Critical appraisal of the performance based research on energetics**

The studies presented above show that attention over time can fail for different reasons. This is not a trivial statement, as impaired attention is reported in several neuropsychiatric conditions, as well as in conditions traditionally considered to be mostly psychogenic in nature. If we want to understand ADHD, a prerequisite is a thorough understanding of the specific mechanisms mediating the attention deficit of that disorder. Whereas persons with different conditions can be impaired in focusing attention on a descriptive level, they nevertheless differ as to whether the attention deficit is characterized by hyperactivity-impulsivity, by presumably reduced arousal under low-intense external stimulation, or reduced ability to sustain activation, as found in ADHD.

An important challenge is to bridge the gap between experimental research and clinical practice. The research reported here uses standard neuropsychological tests commercially available to the clinician, while also supporting previous findings using other methods that have indicated a role for energetics. Extending the research beyond the core symptom of attention deficit, the presented research on the effect of impaired effort allocation gives a possible explanation of the learning and motor impairments accompanying ADHD. Again the findings are derived from clinical tests but nicely fit the notion of impaired attenuation of default mode processing in ADHD.

Although the research described here suggests a role for energetics, does it represent a critical test between the interference model of Barkley and the Cognitive Effort Model?

The finding of an interstimulus effect and the lack of a between group difference in commissions in the Egeland et al study (2009), at least makes the arousal interpretation more parsimonious. However, the effect sizes indicating low arousal in ADHD-C and loss of activation in ADHD-I are small, and can by far be called a defining characteristic. Heterogeneity is typical of all neurocognitive research on ADHD. Examining the frequency of individuals with clinically significant attention deficits in ADHD and schizophrenia Egeland (2010) found that only a minority of adult subjects with ADHD-I were impaired in sustaining attention. The reason for the heterogeneity may be problems with measuring a phenomenon that nevertheless is the underlying mechanism, i.e. the problem of sensitivity. In the Egeland (2010) study, however, the difference between different cut-off levels for impairment was small. Applying a cut-off level for mild impairment showed that somewhere between 35 % and 45 % percent, depending on what measure of sustained attention was used, were impaired in sustained attention. However, 30% to 35 % were impaired also when using a strict level for severe impairment. Although it is generally found that neuropsychological tests are less sensitive than rating scale information regarding behaviour related to the same phenomenon (Toplak et al., 2009), the relatively small impact of changing from the severe to the mild impairment cut-off level indicates that differences in sensibility at best explains only part of the heterogeneity. Another possibility is that there are *several* underlying mechanisms mediating typical ADHD behaviour. These mechanisms may only partly be related to the present subdivision into combined, inattentive and hyperactive-impulsive subtypes. Sonuga-Barke et al. (2010) found that a substantial proportion of ADHD subjects were only impaired in one of three behavioural domains and that their cognitive profiles correlated with that of unaffected siblings. Inhibition was the least common impairment pattern. This research points to the validity of sub-classifying, but challenges the present division.

It may also be that the phenomenon we examine is merely an often occurring symptom more than a measure of the underlying deficit. This is what we claim is the case regarding the findings related to EF and inhibition specifically. An additional source of methodological noise is related to making the diagnosis. Presently, the diagnosis is set by collecting information about function in daily life, indicating attention deficit and/or hyperactivity/impulsivity evident in at least two areas in life, for instance school and at home. It is clear that environmental conditions such as where survey information is collected (Rescorla et al., 2007) and class size (Havey et al., 2005) influence the informants interpretations of children's behaviour. This may contribute to over-inclusion of subjects with ADHD, that again will confound studies looking for underlying mechanisms mediating functional deficits.

Turning back to the presented studies, it is fair to state that although the finding of an interstimulus-effect in ADHD must now be considered established knowledge, the differentiation between the two subgroups of ADHD with regard to arousal, are new. Thus, this must be replicated preferably with imaging or brain activation-measures that could corroborate the findings from the performance based measures, and give validity to the interpretations of them as measures of arousal and activation respectively. The specificity of the arousal and activation measures must also be examined, i.e. whether the same findings characterize other neuropsychiatric conditions that otherwise display executive dysfunction, such as Tourette syndrome and autism.

While the arousal and activation effects were small, the finding of an effort effect on learning, was large. A critical question, however, is whether the operationalizations of effort could be interpreted as executive dysfunction as well. Although impaired executive function cannot be equated to frontal lobe dysfunction, the two are clearly related to the extent that frontal damage are often used as a metaphor for executive dysfunction (Pennington & Ozonoff, 1996). Imaging studies localize proactive interference to the left inferior frontal gyrus (Feredoes et al., 2006), while lesion studies generally find that subjects with frontal brain damage display a *larger* PI effect than controls. Based on a presumption of executive dysfunction one could therefore expect that subjects with ADHD should display an *increased* PI. Instead the *lack* of the normal PI effect indicates low effort processing.

The possible differentiated expectancies from Barkley's interference control model and CEM regarding the other three measures of effort, namely retrograde interference, middle list responses and semantic organization are less clear. Searching the research literature shows no linking of these organizational phenomena to interference control. However, that may reflect that researchers interested in interference control use tests designs directly involving conflict resolution, and represent no direct evidence that executive functions do not play a role in choice of learning strategies. Imaging studies show frontal activation during deep level encoding, so if one adhere to the frontal metaphor for executive dysfunction (Pennington & Ozonoff, 1996) that will indicate a role for EF, although perhaps not for interference control as a more limited EF sub-process, in such organizational processes. However, localization of mental processes is giving way for network models of cognitive function. A large metastudy of fMRI studies of memory (Kim, 2011) showed differential activation patterns between items subsequently failed and items successfully encoded. A distributed network of five brain areas constituted the activation pattern associated with successful encoding, whereas failure of encoding was related to activity in the Default Mode Network. This indicates that inability to change into the effortful task related network underlie failure of encoding.

The hypothesis that insufficient effort constitutes a behavioural correlate to impaired ability to shift from Default Mode processing to task-specific processing, must be tested in studies that apply both behavioural measures (i.e. neuropsychological tests) and imaging techniques.

Future research must also study the relations between the dopaminergic thalamo-cortico-striatal dopaminergic reward system and effort. A dysfunctional reward system in ADHD leads to weaker conditioning, faster extinction of behaviour, and a weaker influence of reinforcers on behaviour in the sense that they are controlled by immediate rather than distal reinforcers (Stark et al., 2011). The dysfunctional dopamine system can be the neurochemical mechanism underlying impaired energetics. A study by Söderlund et al. (2010) showed that white noise normalized memory performance in inattentive school children and worsened performance among attentive school children. The authors speculate that simultaneous noise in inattentive children with low tonic dopamine level, increase stimulus dependent phasic response to stimulation, while a high tonic level suppresses the phasic release. Although not designed as a critical test comparing the interference and the energetic model, one could have expected that having to suppress background task-irrelevant noise should instead lead to distraction.

### 3. Conclusion

The chapter outlines the energetics of ADHD posited by the Cognitive Energetic Model (CEM). Arousal, activation and effort are considered bottom-up processes mediating attention, response and processing capacity among subjects with ADHD. The author's neuropsychological research tries to go beyond global measures of attention, learning and motor function, and to operationalize clinical available measures of the energetics of ADHD. The presented research shows subgroup differences between ADHD combined and inattentive subtypes, indicating a role for effort allocation in both. Impaired arousal may be most typical of the combined subtype, while deficient sustained attention is more typical of the inattentive subtype. Impaired effort allocation may explain impaired memory in both subtypes, whereas impaired motor function may be secondary to impaired energetics only in the combined subtype. Generally, effect sizes are small and heterogeneity large in clinical research on ADHD. This may be due to methodological problems such as inadequate sensitivity of measures or incorrect threshold levels for diagnosis. Also more substantial reasons such as measuring the wrong construct or genuine heterogeneity with regard to what is the core deficit in the disorder, may cause variability. Integrating neuropsychological methodology with research using both time and space distributed brain imaging techniques on the DMN and dopaminergic reward systems will probably lead to a better understanding of the role of bottom-up processes in ADHD, and to what extent they represent additional or alternative explanations of symptomatic behaviour.

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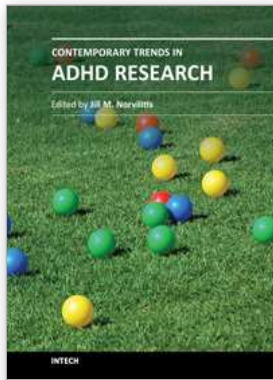
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