The top and the bottom of ADHD: a neuropsychological perspective

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Abstract

Five models of attention deficit/hyperactivity disorder (ADHD) are reviewed. It is proposed that the cognitive-energetic model provides a reasonably comprehensive account of ADHD by incorporating the features of both the inhibition and delay aversion models. It is suggested that ADHD can only be accounted for by an analysis at three levels: top-down control, specific cognitive processes and energetic factors. It is argued that a refined and conceptually comprehensive neuropsychological battery is needed to advance research in ADHD. A widely distributed neural network involving frontal, basal ganglia, limbic and cerebellar loci seem implicated in ADHD.

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1. Introduction

Several theoretical accounts of Attention Deficit/Hyperactivity Disorder [ADHD] have emerged over the last 10 years. The purpose of clinical research is to understand, diagnose and treat disorders efficiently. To achieve this goal, theoretical models are used as a systematic guide in conducting research. The current models of ADHD have different emphases. Some stress the frontal cortex [1], which we refer to here as the ‘top’ of the system. Others emphasise subcortical and even brain stem loci, which are referred to here as the ‘bottom’ of the system [2]. This variation in which locus of the brain is thought to be the cause of ADHD reflects, in part, the symptomatic heterogeneity of ADHD and the high degree of comorbidity with other disorders. The variation in the models also reflects, in part, the theoretical bias of the model builder. Models of how ADHD should be explained also differ with respect to which point in the development of the disorder they emphasise. Currently, there is no model which incorporates developmental data and can predict the developmental trajectory of ADHD. In addition, the differentiation of ADHD from highly associated (comorbid) disorders such as oppositional defiant disorder (ODD) and conduct disorder (CD) has proved neuropsychologically more difficult than first expected [3]. Likewise, differentiation of the attentional dysfunction in ADHD from highly associated disorders such as dyslexia and higher function autism (HFA) has met with some, but mixed success [4]. Use of contrasting clinical groups with either specific brain damage or known biochemical disorders, such as Phenylketonuria, that allow...
the comparison of the hypothesised dopamine deficiency in ADHD with conditions with an established dopamine deficiency are currently ongoing. [5,6]. Critical relationships such as the role of punishment and reward in ADHD and their ensuing influence upon higher cognitive processes have only been modestly established. With this in mind, we briefly review the current models and examine issues which require clarification for further advancement in the field.

Currently, there are five models of ADHD: the Delay aversion model [7], the Behavioural Inhibition/Activation model [8], the Inhibition model [1], the executive function (EF) model [9] and the cognitive-energetic model [2]. These five models can be conceived as varying along a continuum of explanatory power: some models are less and others more comprehensive. In this paper the five models are treated as being conceptually nested within the cognitive-energetic model being considered to be the most comprehensive.

The models differ in how far higher, cognitive processes are considered the key to ADHD. A key term in ‘top-down’ models of ADHD is EF. A second term is working memory (WM), a third concept is attention and a fourth is inhibition. These terms are inter-related and this can be a source of confusion. A function of this paper is to discuss the neuropsychological support for these concepts, in particular for WM, and with reference to ADHD. This discussion may help clinicians unfamiliar with the area to appreciate the significance of this work both in terms of its diagnostic and therapeutic significance. For example, some reflection is required on the validity of the concepts; it is not self-evident whether WM and attention are entirely or only partly independent [10], suggesting that the discovery of a specific process deficit in ADHD will require separating attentional and WM effects in task performance.

Both EF, inhibition and WM are concepts which reflect recent cognitive conceptions of brain function. These terms have been used in ADHD research as mechanisms to explain the ADHD deficit [1,11]. Is EF a sufficient explanation of ADHD? This question has been answered elsewhere as ‘no’ and will not be dealt with here extensively [2,12]. It will be concluded that there is evidence of EF deficits in ADHD, although it is unclear whether the EF account of ADHD is process specific enough to differentiate ADHD from other childhood psychopathological disorders, such as HFA.

In contrast to the work focusing on higher cognitive concepts, there is a body of research which has stressed bottom-up processes such as the role of reward-punishment and motivation in ADHD [8,13,14,15]. Barkley [1] noted that several early studies demonstrated that ADHD children become satiated more quickly than controls. Several researchers have suggested that the performance of children with AD/HD is dependent on the presence or absence of response contingencies [16,17]). Although researchers seem to agree about an unusual sensitivity to reward as a characteristic of children with AD/HD, disagreement exists as to whether these children are over-sensitive or under-sensitive to reward. Some researchers argued and showed that children with AD/HD are less sensitive to reward [17–19]. However, others have argued that children with AD/HD show an increased tendency to look for immediate reward [20,21]). There are also studies that have failed to show that reward affects the performance of children with AD/HD differentially [22,23]. In short, although there is disagreement as to whether children with AD/HD are less or more sensitive to reward, results of several studies have suggested that children with AD/HD react differently to reward than do control children.

Of interest for the separation of ADHD from associated disorders is the finding that CD children are sensitive to reward. For example, Shapiro, Quay, Hogan, and Schwartz [24] showed that children with CD were more sensitive to reward than normal control children. Thus sensitivity to reward may not be specific to ADHD but be a mechanism common to both ADHD and CD.

The effects of reward and punishment have been associated in the cognitive energetic model as being critical to the operation of the effort pool [2,25]. The energetic component of that model might be considered to be a ‘bottom-up’ system which registers and gives feedback to the orbital frontal cortex on whether a particular stimulus–response relation is satisfying or aversive for the organism [26].

This emphasis has two justifications. First, in the early to preadolescent stage of ADHD, motor restlessness has historically been considered a key element of the disorder [27]. The mechanism of transition by which overactive children become more normo-active as adults is unclear, although overactivity can still distinguish ADHD persons from controls in adult life [28]. While activity in sleep and sleep pattern in ADHD has a chequered history [27,29], there does seem to be an association of sleep and behavioural overactivity [30]. Consequently, the models that only emphasise higher meta-cognitive dysfunction may be inappropriate for this subgroup of hyperkinetic children. Secondly, the bottom-up system possibly contains interconnections between activity and delay aversion [31]. Schweitzer and Sulzer-Azaroff [32] noted that in contrast to ADHD children, controls adopted a rational strategy of delaying their responses and gaining maximum reward. The ADHD children in that study also showed increased motor activity with task exposure. Various observational studies have shown increased activity of ADHD children in a vigilance task [33,34] which may depend upon energetic allocation [2]. More recently, reward has been shown to activate the nucleus accumbens, suggesting the ‘bottom-up’ systems may be critical to behavioural deficits in ADHD [35].

2. Reward, delay and inhibition

The first term of the above heading clearly refers to a bottom-up process [35–37]. It is unclear whether delay is
Inhibition is considered the dominant EF function in ADHD [1], whereas Quay [13] would also attribute it to a bottom-up role. This illustrates the need for studies which disentangle the conceptual issues addressed here.

Delay aversion is supported by research which suggests that ADHD children are unwilling to delay their need for gratification. Sonuga-Barke et al. [31] found that, when given a choice between a small immediate reward and a large delayed reward, ADHD children chose immediate reward but only when this led to shorter total task duration irrespective of the amount of reward available. When trial length was paced by the experimenter, ADHD children waited for the longer delayed reward.

On the basis of these results (and others), Sonuga-Barke [39] argued that ADHD children are ‘delay aversive’ but do not have a failure of inhibition. More recently, this position has changed. There is evidence that both the delay aversion and the Inhibition models can account for independent variance in ADHD symptoms [40]. This revised dual pathway model recognises two distinct subtypes of ADHD. In the first ADHD the result of the dysregulation of action and thought due to poor inhibitory control associated with the meso-cortical branch of the dopamine system projecting into the pre-frontal cortex [38]. The second ADHD subtype is conceived of as a motivational style characterised by an altered delay of reward gradient linked to the meso-limbic dopamine branch associated with the reward circuits e.g. nucleus accumbens. Note, however, this revised model does not address the issue of how ADHD does or does not differ from ODD or CD.

The behavioural inhibition/activation model accounts for ADHD by arguing that ADHD children have an under-responsive BIS and an overactive BAS [8,13]. According to Gray [37], the BIS involves the septo-hippocampal area and its connections to the frontal cortex. The BIS signals whether the subject should anticipate pleasure or punishment. Evidence for an under responsive BIS has been provided by Iaboni, Douglas and Ditto [41], who showed that ADHD children do not exhibit increased skin conductance level and, hence remain at the same level of arousal, during extinction as healthy controls. An under responsive BIS has been associated with diminished norepinephrine inputs from the locus coeruleus [37]. The BAS signals the state of the organism which is required to meet the task situation. The ADHD children in one study [41] also displayed faster heart rate habitation to signals of reward which is consistent with the prediction that ADHD have an overactive BAS [8,13].

Importantly, the Behavioural Inhibition/Activation model predicts that ADHD, ODD and CD have a common ‘disinhibitory’ deficit. The Behavioural inhibition/activation predicts that on an inhibitory task, such as the Stop Signal Task, these groups cannot be distinguished. This prediction has in fact been confirmed by a meta-analysis of stop-signal task [42]. Thus this model explains disruptive disorders as a group in terms of the balance between the BIS and the BAS systems. As will be seen later, the Cognitive-energetic model follows the behavioural inhibition/activation model in this respect. The BIS/BAS model differs from the Cognitive-energetic model in not accounting for the role of high cognitive processes such as set-shifting in childhood disorders.

The Inhibition model attributes all deficits in ADHD children to a failure of inhibitory control [1]. Barkley claimed that poor behavioural inhibition is the central deficiency in ADHD and argued that the inhibitory deficits are responsible for secondary deficiencies in other EFs. Thus this model places inhibition above all other EFs. His extensive review of the existing literature envisaged inhibition as also regulating arousal and other energetic factors. In contrast, Nigg [3] suggested that cognitive inhibition is necessary to protect WM. Nigg argued that the observed inhibition deficit in children with HFA might be related to deficits in WM. This places WM squarely in the EF terrain. Nigg’s proposal clearly links inhibition to a WM deficit as did an earlier review [9]. Thus following this line of reason, inhibition is fundamentally a top-down process, as opposed to a bottom-up one. Paradigms are required to differentiate the activation patterns which would be predicted to differentiate these two views, followed by their application in distinguishing ADHD from control and other psychopathological groups.

3. Energetics: when top meets bottom

The cognitive-energetic model draws attention to the fact that ADHD has effects at three levels: cognitive mechanisms such as response output, energetic mechanisms such activation and effort and control systems of EF [2]. The model suggests that disruptive disorders have common deficiencies in EF control systems [42], and may be possibly differentiated either at an energetic level or at specific elementary cognitive stages. Thus the cognitive-energetic model is an attempt to encompass both top-down and bottom-up processes. The cognitive-energetic model does not suggest a single EF deficit in ADHD. It suggests that disorders such as HFA will have communality with ADHD in terms of inhibition of responding. Conversely, it assumes that a range of EF-functions can, in principle, differentiate syndromes. The model suggests that differences between ADHD and ODD/CD should be sought in terms of reward mechanisms influencing inhibitory control [23,43]. Anxiety disorders are predicted to be oversensitive to signals of punishment or reward [23,42]. Hence, in specifying the difference between ADHD and say HFA, the cognitive-energetic model predicts that the EF deficits found will be dependent upon both task parameters and the processing state of the child.

The delay aversion and behavioural inhibition/activation models are considered as part of the cognitive-energetic
model in terms of how the ventro-medial frontal cortex influences EF [26,44].

The primary top-down processing, EF, is associated with five principle domains: inhibition, set shifting, planning, fluency and WM. In addition, top-down processing includes emotional and arousal regulation within the EF construct as well as the executive control of attention [45]. Other definitions of top-down processing include what may be synonyms such as goal directed behaviour for planning, further subdivisions such as inhibition of automatic responses and conflict resolution for inhibition, or even cognitive operations that do not appear to be fully captured by the five principle domains such as error detection [46, 47]. Brain networks associated with executive control include the anterior cingulate cortex (ACC) and supplementary motor area, the orbitofrontal cortex, the dorsolateral prefrontal cortex (DLPFC), portions of the basal ganglia and the thalamus [47].

As noted earlier, a primary task of research in ADHD is to establish the relation between inhibition and WM, particularly in response conflict studies, such as, the Stroop and Flanker tasks. The importance of determining the relation between inhibition and WM was noted earlier in relation to Nigg’s [3] proposal that HFA inhibition deficits might be explained by WM, whereas ADHD WM deficits can be better explained by inhibition dysregulation [1]. The idea that response conflict might differentiate ADHD children from children with other disorders and controls raises the question: what is the nature of the response conflict? It might be due to poor mapping of stimulus-response relationships held in WM, although then the question arises whether problems result from stimulus-inherent interference or from deficient organisation of the S–R associations? On the other hand, response conflict could, in part, be explained by an aberrant reward system in ADHD, which fluctuates while performing a task [48].

Thus two levels of explanation may be required for some ADHD deficits. The first is at the level of the maintenance of stimulus-response relationships in WM. This point we will further address in the discussion. The second level of explanation involves the role of the reward-punishment system which has been associated with structures such as the nucleus accumbens and amygdala [26,49]. It cannot be ruled out that these processes co-occur. We will now focus on the level concerned with maintenance of stimulus-response relationships in WM. The way in which inhibition may be one aspect of WM will be described, i.e. be included in the executive control aspect of WM and how the concept of WM may be a better concept than inhibition in explaining outcome of response conflict tasks in different contexts.

4. WM: maintenance, manipulation and monitoring

In this section, we address evidence concerning the functional anatomy of WM and monitoring. Two types of process in WM may be distinguished: ‘maintenance’ and ‘manipulation’ of information on-line, deal respectively, with the hold function versus the manipulation and/or transposing of information e.g., reversing the order of a list of numbers. The maintenance function of WM includes basic mnemonic processes such as active selection, comparison, and judgement of stimuli held in short-term and long-term memory, whereas the manipulation and monitoring function is involved in ‘strategic’ or higher level executive processes [50,51]. Smith and Jonides [52] reviewed a number of neuroimaging dissociations between maintenance and manipulation processes of WM. Spatial maintenance WM is thought to be mediated by a network of predominantly right-hemisphere regions that include areas in the posterior parietal, occipital, and frontal cortices. Smith and Jonides argued that inhibition is mediated by the left-hemisphere prefrontal region and that it can be dissociated from verbal maintenance and rehearsal processes. Petrides [51] argued that the role of the mid-DLPFC in visual WM is not confined to the maintenance of information, but also involves the monitoring of information.

Bunge, Ochsner, Desmond, Glover and Gabrieli [53] found overlapping activations for maintenance and manipulation during a verbal WM task bilaterally at the ventrolateral and DLPFC, anterior insula, ACC and parietal cortex. However, activation of the right middle frontal gyrus and the left inferior gyrus was correlated with the ability to resolve interference (manipulation) efficiently, whereas ACC-activity was correlated with maintenance. Owen et al. [54] found a significant change of blood-flow in the right mid-ventrolateral frontal cortex, but not in the mid-DLPFC, during maintenance in a ‘spatial span’ WM task. During a spatial ‘2-back’ task, requiring subjects to manipulate WM content, blood flow increases were observed in both frontal regions. A direct comparison between the two tasks revealed that only manipulation results in significantly greater activity in the right mid-DLPFC. Thus activation of DLPFC may be dependent on how much a task requires the ‘manipulation’ process as in both the N-back task and the self-order-pointing (SoP) task [50].

The ‘manipulation’ process in WM is also dependent upon the ACC [47,53,55–59]. ‘Manipulation’ seems also to be dependent upon cognitive expectancies. Carter et al. [56] varied subjects’ expectancies for congruent and incongruent stimuli in a variant of the Stroop colour naming task. When expectancy for incongruent stimuli was high, no response-related increase in ACC-activity was present. In contrast, expectancy for congruent stimuli resulted in an increase of ACC-activity when an incongruent stimulus was presented. It was concluded that, when incongruent stimuli were expected, control processes were engaged to overcome the prepotent tendency to read the word. This top-down control seems particularly relevant to the issue briefly noted earlier: delay aversion may be the result of a mismatch between
expectancies and predicted reward, which may be linked to ACC functioning in ADHD children [43,60]. Consequently, delay aversion may be dependent upon top-down processes unaccounted for by the delay of gratification model but can be explained by the cognitive energetic model.

What is the significance of this research for ADHD? It firstly informs us that simple conflict tasks such as the Stroop are more than just inhibition tasks. They also involve WM. The specific form of WM which is used in a conflict task depends upon the precise top-down process(es) tasks require. Thus, from such measures and tasks there is task impurity making it difficult to conclude specifically which process or interaction between WM and inhibition is accounting for the observed results. Since there is some evidence that ADHD adults perform the Stroop by not activating the ACC but bilaterally activating the insula [61], studies of response conflict in children are urgently required in order to determine whether this finding also occurs in children with ADHD. If this were found to be the case, a theoretical account of ADHD would have to incorporate why the insula is active and why the ACC is inactive in ADHD. One possible explanation may be that activation of widely distributed brain networks reflects an inefficient match between required cognitive control and selective firing of the usual specific loci: too much activation is uneconomic; producing too little output for too much brain labour.

5. Distinguishing WM and executive attention

WM evokes the association of a mechanism associated with purely memorial process. However, Baddeley and Logie [62] noted that this understandable association is due to how Baddeley and Hitch [63] approached the issue. Had they approached the concept from an attentional point of view, then it would probably have become termed ‘working attention’. The host of models which describe WM, their differences and similarities have been summarised by Miyake and Shah [64]. For present purposes, a working definition of WM would be active long-term memory, responding to selective attention demands. In a study investigating the relationship between visuospatial search and object WM, Pollmann and von Cramon [65] showed that the right DLPFC is significantly involved when difficulty of the object-recognition process increases and more attention is demanded. This finding suggests that the right DLPFC is invoked in executive control of selective attention/orienting. In support of this, Casey et al. [57] demonstrated in a response conflict task, the flanker task, that activation in both the ACC and DLPFC increased as a function of interference from the response conflict induced by the flanker stimuli with increasing top-down control. This research indicates that there is clearly both specificity of activation but also an overlap in processes subsumed under the concepts of WM and selective attention. Further, the activation observed is dependent upon the controlled processing demands exerted by the task. Consequently, researchers in ADHD will need to account for both processes in the disorder. This may be particularly important in studies with children who not only have ADHD but also either dyslexia or language impairments [66–68].

Selective attention has been defined as serial, effortful controlled processing which can operate either in a focused or in a divided mode [69]. Kane et al [45] refer to executive attention to stress the attentional control component of selective attention. Cowan [70] emphasised the role of activating long-term memory and the temporary nature of this activation in selective attention. It is clear that the concept of WM is not simply attention and it is also not simply memorial. The associated slave systems of the visual scratch pad and the auditory loop reflect the temporary and modality specific nature of activated long-term memory stores [62]. Thus WM may be better conceived of as the selective activation of long-term memory, which requires executive attention. Executive attention appears to have the property of inhibitory control [45], particularly in tasks where shifts of covert and overt attention are required [57]. Executive attention is also required in what is termed monitoring. The monitoring function may vary as a function of the attentional load, or complexity of S–R relationships, and possibly the length of the delay between stimulus presentation and response execution. Since monitoring may be at the heart of the inhibition model [1], clarity concerning this concept and its operationalization would be helpful in future ADHD research. Furthermore, and currently not well-documented, is the fact that tasks commonly called WM tasks may vary along a dimension of controlled-automatic processing and generate different activation patterns. The automatic-controlled dimension may be not only of importance for brain activation patterns but also for the deployment of attentional resources via an effort pool [2].

6. EF and neurotransmitters

EF research in relation to the biochemistry of ADHD constitutes an invaluable contribution to theoretical accounts of cognitive dysfunction in ADHD and may give some future clues to the aetiology of the disorder. In order to delineate exactly what occurs in ADHD, neurotransmitter availability in specific brain regions must be investigated, since some aspects of EF and WM might depend on particular neurotransmitters. Allocation of energetic resources is probably strongly associated with the availability of particular neurotransmitters. Methylphenidate is the current pharmacological treatment of choice in ADHD [71]. Volkow, Fowler, Wang, Ding, and Gatley, [72] have documented that in the human brain therapeutic doses of methylphenidate block more than 50% of the dopamine transporters and significantly enhance extracellular DA, an
effect that appears to be modulated by the rate of DA release. Volkow et al. [72] postulate that methylphenidate’s therapeutic effects are in part due to amplification of DA signals, and that variability in responses is in part due to differences in DA tone. Further, methylphenidate’s effects may also be context dependent. Methylphenidate-induced increases in DA are also associated with its reinforcing effects but only when this occurs rapidly, as with intravenous administration [72].

In order to have some insight into the significance of neurotransmitters in cognitive functioning and its potential for understanding the nature of ADHD, a selective and very brief account is given of EF studies in relation to neurotransmitters relevant for ADHD. Since a hypofunction of dopamine may account for ADHD [72,73], it is important to consider the similarities and dissimilarities in EF between ADHD and PKU, an inherited metabolic disorder which has been established to represent a dopamine deficiency. We briefly discuss the relation of dopamine with EF.

Both dopamine and norepinephrine are important modulators of the attentional system [74]. There is evidence for a relation between acetylcholine and orienting/selective attention, norepinephrine and vigilance/sustained attention, and dopamine and executive control [47]. Dopamine availability has been found to influence some but not all cognitive functions mediated by PFC. McDowell, Whyte, and D’Esposito [75] administered a dopamine agonist, bromocriptine, to patients with lesions in the PFC. Interestingly, on tasks requiring both manipulation and monitoring of WM performance improved with bromocriptine. In contrast, when maintenance of WM was manipulated, performance did not improve with bromocriptine.

One study reported that bromocriptine improved performance on prefrontal measures only in subjects with lower WM capacity, whereas it impaired performance in subjects with higher WM capacity [76]. The results suggest that the effect of a specific neurochemical agent may be dependent on the type of cognitive operation used, the individual differences and possible interactions between task and cortical network affected by the neurotransmitters [77]. Thus, when the effects of methylphenidate on the cognitive performance of ADHD children is investigated, it is important to consider which aspects of cognition are influenced by which component of the medication, i.e. do dopaminergic and noradrenergic enhancements target specific aspects of WM or does noradrenergic enhancement change the level of vigilance which might result in overall cognitive improvement? It has been suggested that inability of ADHD children to delay their need for gratification [31] may be influenced by methylphenidate. In this respect, Mehta et al. [72] were able to dissociate maintenance from manipulation in a spatial WM task of the Cambridge Neuropsychological Test Automated Battery (CANTAB) using methylphenidate. The maintenance function of this task involved the online storage and active response organisation based on the retrieval of information from posterior cortical association systems. In contrast, manipulation involved inhibition of previously learnt, successful S–R associations. Maintenance resulted in drug-induced changes in regional cerebral blood flow in the posterior parietal cortex, whereas manipulation led to methylphenidate induced changes in left DLPFC. Methylphenidate has been shown to normalise CANTAB spatial WM performance in ADHD [78,79]. More studies are required to establish the availability of neurotransmitters and their role in cognition in ADHD.

Comparison of ADHD performance with PKU children is relevant here in view of the well-documented dopamine disorder in PKU children. PKU children have difficulty in sustained attention [5], poorer inhibition of a prepotent responses, and poorer attentional flexibility [6]. However, no deficit was found in PKU children for the maintenance function of WM, neither in orienting nor in stimulus-inherent interference suppression [6]. The contrast between PKU and ADHD is a useful one in informing us of the differences between an established and putative disorder of dopamine in childhood.

7. Discussion

In this paper the main thrust has been to consider in detail the implications of the top-down and the bottom-up processes which may be implicated in ADHD and other childhood psychopathological disorders. Memorial span task requiring no other concurrent function were distinguished from span tasks requiring concurrent processing. In addition, it was noted that concurrent processes such as monitoring or selective attention are intimately associated with WM [51]. Hence, as a working hypothesis, it is assumed here that WM = short term memory (or activated long-term memory) + selective attention [10,64].

In previous research with the Sternberg memory scanning paradigm, differences between ADHD and control children could not be accounted for by the increase in memory load [80–82]. Interestingly, this effect has been replicated in ADHD comparisons with normal controls using a non-verbal visual WM task, the Self-ordered Pointing task [4]. However, other WM tasks such as the N-back task have differentiated ADHD children from controls [83]. Shallice et al concluded that the EF differences found in their study between ADHD children and controls might be associated with a higher-level effort mechanism and did not simply reflect only WM [2].

It has been noted that one may consider WM as the combination of short-term memory plus selective attention [64]. This definition stems, in part, from research originating in the controlled processing literature on selective attention [69,84]. Selective attention was defined in that literature as a limitation in the rate of controlled processing located in short term memory. This definition led to a series of studies using memory and visual search paradigms in
ADHD children [25]. The tasks used in that research programme were memory maintenance tasks (with varying attentional load) requiring a simple concurrent process: target detection. From that research it was evident that the maintenance function of WM was intact in ADHD children both as a controlled process [80,81] and as an automatic process [85]. The corollary of this is that, when attentional demands are manipulated parametrically, ADHD children do not have a deficit in selective attention. However, when monitoring of errors and adjustment in performance was required, ADHD children had a deficit of response adjustment compared with controls [86]. This is where ADHD children may differ from for example PKU children, since error monitoring and adjustment appear not to be problematic in children with PKU [6].

Engle et al [10] have addressed the relation between EF and WM. Engle et al. suggested that, before one can draw conclusions on the executive element of EF, one needs to account for such factors as general and fluid intelligence and short term maintenance memory. When these factors have been partialed out, the remaining variance may be considered as EF or WM-manipulation and monitoring. To reach this goal, studies will need to employ structural equation modelling in order to partial out the variance shared between these various processes.

Specificity of dysfunction is generally achieved in neuropsychology by a double-dissociation. Double-dissociation requires that two clinical groups can be shown to differ from one another such that one group shows deficits in one task (A) but not the other (B), whereas the second group shows deficits on test B but not A. An attempt to obtain a double dissociation between HFA and ADHD has recently been conducted by our group [4]. The comparison of HFA with ADHD indicated that the EF deficit was more general and severe in children with HFA. HFA children had deficits in six cognitive domains: inhibiting a prepotent response, interference control, visual WM, cognitive flexibility, verbal fluency, and planning. In contrast, the ADHD group was deficient in three areas of EF: inhibiting a prepotent response, cognitive flexibility, and verbal fluency. Despite the variety of differences between controls and HFA and ADHD respectively, a striking finding from that study was that the ADHD and HFA groups could only be differentiated from one another on visual WM, whereby the HFA group had greater visual WM deficits than ADHD children.

The results reported by Geurts et al [14] contrast to those of Ozonoff and Jenssen [87] and of Nyden et al. [88]. Ozonoff and Jenssen concluded that children with autism have deficits in planning and flexibility, but not in inhibition. ADHD children in the Ozonoff and Jenssen study exhibited deficits which were exactly the opposite. In the Nyden study, children with ADHD had deficits in flexibility and response inhibition. The autistic children had deficits in response inhibition only. It is unclear whether the differences between the studies may be explained in terms of comorbid disorders, such as ODD or CD [9,12].

The importance of also assessing ODD and CD in double-dissociation studies has been demonstrated by Séguin, Boulerice, Harden, Tremblay, and Pihl [89]. Séguin et al used the SoP [Petrides, 199]. Children with physically aggressive behaviour had poorer visual WM than children who were aggressive but not physical. Séguin et al showed in their analysis that ADHD did not account for the EF deficits in children with a history of physical aggression. Further research is needed to establish the neuropsychological commonalities and differences between ADHD, ODD/CD and the comorbid groups [40,90–92].

This selective review of the literature suggests that the discovery of a neuropsychological profile for each of the childhood disorders will be dependent upon using sophisticated processing batteries and analysis techniques. The batteries need to control for on the one hand general effects of short term memory, flexible intelligence and, on the other hand, both modality effects as well as the distinction between maintenance and manipulation. The existing data suggest that childhood disorders can be differentiated only quantitatively and not qualitatively in contrast to predictions from the double dissociation paradigm. The target of interest would seem to be WM for the differentiation of HFA from ADHD as well as ODD/CD. Contrasting levels of encoding: graphic, orthographic, semantic, appears a fruitful approach for differentiation of dyslectic children from children with pathological conditions. Comorbidity of psychopathology with dyslexia may be expected to lead to complex process interactions, which may not lend themselves easily for group differentiation.

Throughout this paper, there has been noted that the reward-punishment relationship is essential to comprehending ADHD [36]. In addition, it has been argued here that there is reason to believe that the differentiation of ADHD and CD is to be found in this relationship [8]. Recently, it has been found that children rated to be ADHD, when administered a punishment trial following a period of reward trials, responded like controls to avoid punishment [93]. This finding would seem a useful starting point in attempting to discriminate ADHD children from CD children. This finding contrasts with the position of Newman and Wallace [94] that inhibitory deficits would be more apparent in ADHD, when signals of punishment are introduced. Two possible caveats here should be noted: the period between signal and administration of reward/punishment may be critical [32], since this is related to dopamine functioning; secondly, the magnitude of subjective expectation of reward/punishment may determine performance [21].

We have suggested that comparison between the five current models of ADHD is a fruitful exercise. It has become evident that two models: inhibition and delay aversion independently explain variance in ADHD symptoms. This finding indicates in itself that neither model is sufficient to inform us what the ADHD deficit is. Therefore, a model is needed which incorporates both models. We have
argued that the cognitive energetic model is currently the most comprehensive account of ADHD. Nevertheless, there remains much to be done to achieve the specificity required to account for childhood psychopathological disorders. A comprehensive account of ADHD in relation to the associated disorders has yet to be assembled. In order to get to the bottom of the disorder, not only top-down but bottom-up processes need to be accounted for in ADHD.

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