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The cognitive-energetic model: an empirical approach to Attention-Deficit Hyperactivity Disorder

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Abstract

Attention Deficit/Hyperactivity Disorder (ADHD) is a childhood psychiatric disorder which when carefully defined, affects around 1% of the childhood population [Swanson JM, Sergeant JA, Taylor E, Sonuga-Barke EJS, Jensen PS, Canwell DP. Attention-deficit hyperactivity disorder and hyperkinetic disorder. *Lancet* 1998;351:429–433]. The primary symptoms: distractibility, impulsivity and overactivity vary in degree and association in such children, which led DSM IV to propose three subgroups. Only one of these subgroups, the combined subtype: deficits in all three areas, meets the ICD-10 criteria. Since the other two subtypes are used extensively in North America (but not in Europe), widely different results between centres are to be expected and have been reported. Central to the ADHD syndrome is the idea of an attention deficit. In order to investigate attention, it is necessary to define what one means by this term and to operationalize it in such a manner that others can test and replicate findings. We have advocated the use of a cognitive-energetic model [Sanders, AF. Towards a model of stress and performance. *Acta Psychologica* 1983;53: 61–97]. The cognitive-energetic model of ADHD approaches the ADHD deficiency at three distinct levels. First, a lower set of cognitive processes: encoding, central processing and response organisation is postulated. Study of these processes has indicated that there are no deficits of processing at encoding or central processing but are present in motor organisation [Sergeant JA, van der Meere JJ. Convergence of approaches in localizing the hyperactivity deficit. In Lahey BB, Kazdin AE, editors. *Advancements in clinical child psychology*, vol. 13. New York: Plenum press, 1990. p. 207–45; Sergeant, JA, van der Meere JJ. Additive factor methodology applied to psychopathology with special reference to hyperactivity. *Acta Psychologica* 1990;74:277–295]. A second level of the cognitive-energetic model consists of the energetic pools: arousal, activation and effort. At this level, the primary deficits of ADHD are associated with the activation pool and (to some extent) effort. The third level of the model contains a management or executive function system. Barkley [Barkley RA, Behavioral inhibition, sustained attention, and executive functions: constructing a unifying theory of ADHD. *Psychological Bulletin* 1997;121:65–94] reviewed the literature and concluded that executive function deficiencies were primarily due to a failure of inhibition. Oosterlaan, Logan and Sergeant [Oosterlaan J, Logan GD, Sergeant JA. Response inhibition in ADHD, CD, comorbid ADHD + CD, anxious and normal children: a meta-analysis of studies with the stop task. *Journal of Child Psychology and Psychiatry* 1998;39:411–426] demonstrated that this explanation was not specific to ADHD but also applied to children with the associated disorders of oppositional defiant and conduct disorder. Other executive functions seem to be intact, while others, are deficient. It is argued here that the cognitive-energetic model is a useful guide for determining not only ADHD deficiencies and associated disorders but also linking human cognitive neuroscience studies with neurobiological models of ADHD using animals [Sadile AG. Multiple evidence of a segmental defect in the anterior forebrain of an animal model of hyperactivity and attention deficits. *Neuroscience and Biobehavioral Reviews*, in press; Sagvolden T, Sergeant JA. Attention-deficit hyperactivity disorder: from brain dysfunctions to behaviour. *Behavioural Brain Research* 1998;94:1–10]. A plea for an integrated attack on this research problem is made and the suggestion that conceptual refinement between levels of analysis is essential for further fundamental work to succeed is offered here. © 2000 Elsevier Science Ltd. All rights reserved.

Keywords: Cognitive-energetic model; Attention deficit hyperactivity disorder; Psychiatric disorder; Executive functioning; Attention; Inhibition

1. Introduction

Children and adolescents with an excess of hyperactive, inattentive and impulsive behaviour are diagnosed currently to have Attention Deficit/Hyperactivity Disorder (ADHD). Currently, the third facet of ADHD, impulsivity, has

become the focus of research effort. It has been strongly advocated that disinhibition, is central to distinguishing this disorder from others [1,2,31,32]. This approach suggests that ADHD is the result of a failure to delay responding associated with inhibitory deficits.

An alternative position has been advocated using a model of information processing which attempts to identify the locus of the ADHD deficit [43] Our approach utilises the

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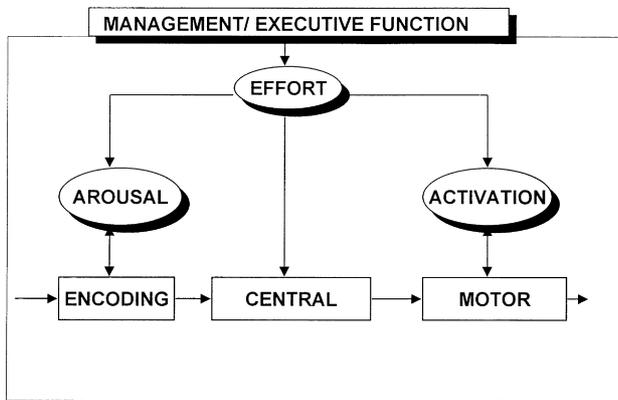


Fig. 1. The Cognitive-energetic model.

cognitive-energetic model described in [36] and elaborated in ADHD in [42–44] and recently [45]. This model suggests that there may be certain aspects of inhibition, which is deficit in ADHD children, but that this is also dependent upon the energetic state of the child.

In order to clarify this position, some of the features of the model guiding our research are presented followed by a brief discussion of related research in ADHD.

2. The cognitive-energetic model

The overall efficiency of information processing in the cognitive-energetic model is said to be determined by both process (computational) and state factors (such as effort, arousal and activation). Computational mechanisms of attention include four general stages: encoding, search, decision and motor organisation [48]. These stages of information processing (see Fig. 1) are associated with experimental task variables [37].

The second level of the model encompasses three energetic pools. These pools include *effort*, which is conceived of as the necessary energy to meet the demands of a task. Factors which effect effort are variables such as cognitive load. Effort is said to be required when the current state of the organism did not meet the state required to perform a task. The second pool, *arousal* is defined as phasic responding which is time locked to stimulus processing [30]. By contrast, tonic changes of physiological activity were thought to represent the operation of the *activation* pool [30]. The activation pool was identified with the basal ganglia and corpus striatum [30].

The cognitive-energetic model includes a third level, management or evaluation mechanism, which is associated with planning, monitoring, detection of errors and their correction. This is currently associated with the concept of Executive Functioning (EF). EF is defined by [51] as the ability to maintain an appropriate problem-solving set for the attainment of a future goal. This includes such functions as: intention to inhibit a response, defer a response to a

future moment, strategic planning, mental representation of a task. We will restrict this review to the inhibition and error detection/correction aspect of EF in ADHD.

2.1. Response inhibition

The terms inhibition/disinhibition have at least 12 different meanings and experimental operationalizations [45]. We consider four of these operationalizations here. These four have been chosen, since there are currently available anatomical and/or functional Magnetic Resonance Imaging (fMRI) and other neural imaging techniques using these paradigms.

Disinhibition is currently best operationalized as a failure to suppress inappropriate responding in Go/No-go tasks or as a failure to suppress inappropriate responding in the stop-signal task and change task. The operationalization of EF which will be considered here is a failure to correct and to adjust responding as a result of the detection of an error.

2.2. Go/no-go task

Human cerebral blood flow studies have shown that the cingulate activation is important in target detection, Go/No-go and Stroop performance [27]. As will be noted later the anterior cingulate gyrus is considered the locus of the EF attention network described in Ref. [29]. Several studies have shown that, when ADHD children are instructed to respond on go signal trials and refrain from responding on no-go trials, they commit more no-go responses or errors of commission [14,47]. Higher proportions of No-Go responses have also been reported in children with “attentional” problems [13]. These researchers found that in a go/no-go task alpha attenuation was related to no-go reactions in ADHD children. This work supported the hypothesis that energetic failures underlie the inhibitory dysfunction of ADHD children, since reduction in energy predicts failures of inhibition. However, there is evidence that aggressive males commit more commission errors (“go” responses to a “no-go” signal) than non-aggressive males [17,21,40]. This being the case, it will be necessary to determine whether what has been claimed to be a specific deficit for ADHD is perhaps reflecting a more general problem of disinhibition in all disruptive disorders.

Autoradiographic studies of rat models of ADHD have shown that such rodent models have a higher density of dopamine (D_{1-5}) at the caudate-putamen and nucleus accumbens [5] reduced calmodulin-dependent protein kinase in the anterior basal forebrain [25]. These findings are intriguing in suggesting that human neuropsychological studies should be directed to determining the differential role of these structures in both executive function tasks. In a go/no-go task in which stimulus-response frequency was manipulated (a manipulation which effects the response stage of the cognitive-energetic model), there has been found decreased striatal activation in ADHD than controls [50]. The same study reported that when MPH was

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