In real life, however, there are too few such resourceful parents to go around; and their availability to children suffering from the problems of ADHD is even more restricted. This is because about one in five of these parents themselves have ADHD, some with added complications of depression, personality disorder, learning disability, or substance abuse. Parents with such problems of their own will have even greater difficulty coping with their child's special needs (Lesesne et al. 2003). A child with ADHD growing up in these circumstances is at high risk for additional emotional and behaviour problems, with their likelihood further increased by low social class, parental psychopathology, and family conflict (Biederman et al. 2002b; Minde et al. 2003).

To elucidate the risk mechanisms involved, the authors juxtapose predictions from their theory with those of the coercion theory of antisocial behaviour disorder by Patterson (1982). According to Patterson, child non-compliance develops through a circular process of negative reinforcement between child and parent. Sagvolden et al. argue that such coercive child behaviour, once established, is especially hard to extinguish in children with ADHD (and in their often ADHD parents).

Because it is a highly familial disorder, ADHD also means that the same parents provide the genes and the environment. Parental ADHD, as a result of its core symptoms and/or comorbidities, is associated with disruptive family environment and suboptimal parenting practices that often are resistant to modification (Chronis et al. 2004; Sonuga-Barke et al. 2002). ADHD in fathers, for example, predicts higher levels of family disruption as a consequence of parental desertion and custodial sentences for impulsive behaviour (Minde et al. 2003). The already demanding tasks of childrearing place a parent with ADHD at considerable disadvantage: Maintaining patience and emotional responsiveness towards the child, providing attentive supervision, and organising domestic duties and childcare frequently present the parent with an unmanageable challenge. Also, extrapolating from the proposed theory, a parent with ADHD will find it hard to emotionally disengage amidst a child's temper tantrum, but will easily end up contributing to its escalation, instead.

These parenting styles bear resemblance to those observed in studies of depressed mothers. For example, a recent longitudinal study involving detailed observations of the interaction between postnatally depressed mothers and their infants revealed a striking pattern of "coercive caretaking" – a phenomenon hardly ever seen in mothers who were not depressed (Murray et al. 1996). This pattern of early interaction had long-lasting connections, predicting disruptive behaviour at least to age 8 (Morrell & Murray 2003). Thus, there is a particular reason to pay attention to ADHD in girls in whom the problems are often overlooked until teenage years, or entirely missed. Compared with boys with similar levels of ADHD, girls are at a higher risk for anxiety, depression, and poor psychosocial functioning (Rucklidge & Tannock 2001). If ignored, these problems are likely to continue into adulthood and will determine the future style of parenting – of children probably sharing the mother's ADHD genes.

It seems fit to conclude by agreeing with Sagvolden et al. in that "ADHD . . . is a case where functions of the central nervous system occasionally exceed the limits of normal variation and adaptation" (sect. 3, para. 3) – and add environmental accommodation.

The dynamic developmental theory of ADHD: Reflections from a cognitive energetic model standpoint

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Abstract: "A dynamic developmental theory of attention-deficit/hyperactivity disorder (ADHD) predominantly hyperactive/impulsive and combined subtypes" is a major contribution linking comparative psychology with clinical developmental neuropsychopathology. In this commentary, I place some critical remarks concerning the theory's explanation of sleep problems, inhibition, error monitoring, and motor control.

The target article by Sagvolden et al. is a veritable blockbuster linking comparative psychology with clinical developmental neuropsychopathology. The neuroscience of attention-deficit/hyperactivity disorder (ADHD) has only recently begun to emerge as a major contributor to our understanding of the aetiology and development of this disorder. The target article is both timely and informative and sets a research agenda for neuroscientists in the field of ADHD. The variety of issues that have been treated and to varying degrees integrated in the dynamic developmental theory (DDT) is exceptional.

Sagvolden and colleagues argue that in DDT there are two main behavioural processes causing ADHD: altered reinforcement of novel behaviour and deficient extinction of previously reinforced behaviour. Further, the authors argue that the time available for associating behaviour with its consequences is shorter in ADHD than in normal children, on account of the delay gradient being steeper and shorter in children with ADHD than in normal children.

First, I briefly address the relationship between state factors such as sleep and diurnal rhythm and the independence or interaction of both reinforcement and inhibition. Second, I argue that the DDT does not recognize in its current form how both state and inhibition contribute to explaining ADHD. Third, I draw attention to the fact that a comprehensive model of ADHD must account not only for correct responding but also the effect of detecting an error upon the following trial. Fourth, I refer to an omission in the DDT, namely, the role of motor factors in accounting for ADHD behaviour.

Convergence. Clinical neuropsychologists have been for some time interested in the relation between performance and reinforcement in ADHD children (Douglas & Parry 1994). Few areas of neuropsychopathology have been blessed with a richly researched animal model of the disorder of interest, and it is, therefore, timely that prior to the awaited DSM-V (*Diagnostic and Statistical Manual, 5th edition*), neuroscientists inform the clinical community of basic findings relevant to the definition of the disorder. The DDT provides an account of ADHD that requires careful evaluation. Hence, from a clinical neuropsychological point of view, one wishes to determine where is the convergence and where is the divergence between the DDT and, say, a cognitive-energetic model (CEM) explanation of ADHD.

Thankfully, there is convergence of evidence from the animal research reviewed in the DDT with the CEM. Both models note the variability of responding in ADHD, and both agree that reinforcement is a key factor in determining current and future behaviours in ADHD. They both appeal to a dopamine deficiency as the biochemical substrate of the disorder. The DDT and CEM instruct researchers to examine the clear association of the interval used to demonstrate deficiency: short intervals producing little or none; long intervals producing clear manifestations of deficiency compared with control children or animals. The DDT and CEM implicate widely distributed neural circuits being involved in ADHD, namely, the frontostriatal-limbic and cerebellar networks. The DDT is stronger than the CEM in its genetic predictions. For the purposes of this commentary, I briefly address four

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points of divergence between the DDT and CEM: the role of state factors on ADHD, the independent role of reinforcement and inhibition, cognitive readjustment to protect future emissions of response, and motor behaviour.

State factors. The DDT contrasts with the CEM in that the former is a behavioural explanation of ADHD, and the CEM, as its name suggests, emphasizes both cognitive and energetic aspects of human behaviour. The CEM notes that behavioural overactivity of ADHD children occurs not only in the awake state but can also occur in sleep (Porrino et al. 1983). It is hard to know how an altered reinforcement mechanism could explain this finding, without having to appeal to additional biochemical mechanisms not specified in the DDT. Similarly, the DDT is unclear how diurnal effects which are related to behavioural activity occur when they do following midday (Porrino et al. 1983). What is the specific alteration in reinforcement that is linked to this diurnal effect? Furthermore, changes in brain state have been shown to predict the occurrence of succeeding errors (Brandeis et al. 2002).

Reinforcement and inhibition. Sagvolden et al. write "the response unit that is supposed to be inhibited is hard to define empirically (Catania 1998)" (sect. 1.2.3, para. 6). Inhibition, although a loose construct and operationalised in a variety of manners, can be measured by stop-signal reaction time (SSRT; Logan & Cowan 1984). It has been demonstrated to have high reliability (Band et al. 2003), associated specifically with the inferior frontal gyrus (Aaron et al. 2003), to be correlated with familial manifestations of ADHD and, in two meta-analyses, to distinguish ADHD from controls at a specific latency (Lijffijt et al., in press; Oosterlaan et al. 1998). Inhibition has been shown to be independent of reinforcement in predicting ADHD group membership (Solanto et al. 2001a). Several studies have shown that inhibition deficits in ADHD are independent of reinforcement (Oosterlaan & Sergeant 1998a; Scheres et al. 2003). One study showed an interaction between inhibition and reinforcement (Slusarek et al. 2001). These studies suggest, at the very least, that both inhibition (operationalised by the SSRT) and reinforcement are needed to explain ADHD.

Cognitive adjustment. When a human commits an error, cognitive resources are allocated to ensure that on the following trial, an error is not committed by slowing down the speed of responding (Rabbitt & Rodgers 1977). Normal children do this, but ADHD children fail to make this cognitive adjustment (Sergeant & van der Meere 1988). This effect is independent of SSRT (Schachar et al. 2004) and can be improved by methylphenidate (Krusch et al. 1996). The DDT in its present form cannot account for this phenomenon, because it requires error detection, correction, and resource allocation – concepts not in the DDT.

Motor factors in ADHD. There has long been a clinical interest in motor functioning in ADHD (cf. Clements & Peters 1962) and even recently in differentiating ADHD children from children with a neurological disorder (Konrad et al. 2000). ADHD children can be differentiated from controls on repetitive movements (Carte et al. 1996), fine motor difficulty (Pitcher et al. 2003), movement control (Eliasson et al. 2004), poor balancing (Raberger & Wimmer 2003), and excessive overflow movements (Mostofsky et al. 2003).

Abnormal rhythmic motor response in ADHD has been demonstrated using a tapping task (Ben-Pazi et al. 2003). ADHD children had difficulty modulating their responses with changing rhythms. Motor deficits need to be incorporated in the DDT.

Conclusion. The DDT model is an interesting contribution to the neuroscience of ADHD but requires expansion to accommodate the four areas noted here to be relevant for ADHD.

A common core dysfunction in attention-deficit/hyperactivity disorder: A scientific red herring?

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Abstract: The reinforcement/extinction disorder hypothesis (Sagvolden et al.) is an important counterweight to the executive dysfunction model of attention-deficit/hyperactivity disorder (ADHD). However, like that model, it conceptualises ADHD as pathophysiologically homogeneous, resulting from a common core dysfunction. Recent studies reporting neuropsychological heterogeneity suggest that this common core dysfunction may be the scientific equivalent of a red herring.

The classical disease model of mental disorder rests on a number of assumptions (ideas taken for granted rather than tested empirically): Disorders are discrete entities, qualitatively different from normality and resulting from dysfunction (neurobiological, neuropsychological) at some level *within* the patient (Sonuga-Barke 1998). These assumptions have played a defining role in the contemporary neuroscience of mental disorder. They provide a metatheoretical framework allowing shared points of reference that link science and clinical practice through common language, assumptions, and goals. They also constrain the types of questions that are deemed legitimate and the methods employed to answer them. In the neuroscience of ADHD, this has meant that one question above all has provided the ultimate challenge for researchers: Where, within the brain or mind of the ADHD child, is the site of the common core dysfunction that *causes* ADHD (Sonuga-Barke 1994)?

It is typical of "normal" science that one particular model garners the support of a large, cohesive, and influential group of supporters. This model then takes on the mantle of scientific orthodoxy. In the neuroscience of ADHD, this mantle has fallen on the executive dysfunction model (Arnsten 2001). This model proposes that ADHD is the result of a common core dysfunction in executive control associated with deficient inhibition-dependent processes such as working memory, planning, and interference control. These are underpinned by the prefrontal cortex and related neural circuits and neurotransmitter branches (especially mesocortical dopamine and norepinephrine pathways; cf. Roth & Saykin 2004). This "classical" executive dysfunction model, although initially based on an analogy between ADHD and the hyperactive and distractible behaviour of patients with prefrontal lesions, now receives support from (1) psychopharmacological studies highlighting the role played by catecholamines in the pathophysiology of ADHD (Bedard et al. 2004), and (2) neuroimaging studies demonstrating abnormalities within the frontal-striatal networks of children with ADHD (Castellanos 1997). Although few studies have tested its causal status, these data have been taken as compelling evidence for the executive dysfunction model of ADHD.

Challenges to this model take a number of different forms. First, there are those alternatives that call for its reinterpretation rather than its overthrow: The "field" has been looking in the right place (prefrontal cortex-executive function) for the right thing (a common core dysfunction) but needs to adjust the current model to take account of new data or ways of thinking. For example, the state dysregulation account proposed by Sergeant and colleagues elaborates the executive dysfunction model to account for the effects of factors such as reward, stimulus presentation speed, and stimulant drugs by incorporating the concept of cognitive energetic dysregulation (Sergeant et al. 1999). Second, some accounts propose a more radical departure from the dominant model. They argue that, while looking for the right sort of thing (a common core dysfunction), the field is looking in the wrong place. The model proposed by Terje Sagvolden and colleagues in the target article

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