

The biopsychosocial context of ADHD

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Abstract: Attention-deficit/hyperactivity disorder (ADHD) represents adaptation to defective neurotransmission – an adaptation seldom with benefit. The resulting behavioural style not only increases vulnerability to adverse experiences, but also creates a context in which encountering adversity is more likely. Furthermore, the fact that ADHD is a highly heritable condition increases the probability of a child with a compromised neurobiological disposition being raised by caregivers with suboptimal resources.

The target article is, to my knowledge, the first serious attempt to present a unified theory expanding from the biology of brain neurochemistry to continuously evolving interaction between a child with attention-deficit/hyperactivity disorder (ADHD) and his or her social environment. It is also noteworthy that the first author, Sagvolden, is a renowned scientist in murine research.

At the basis of the dynamic developmental theory, put forward by Sagvolden et al., is a model of dysfunctional dopamine systems in the brain. Three hypofunctioning dopamine system branches and their behavioural consequences, representing the core symptoms of ADHD, are outlined. These compromised properties result from a combination of intrinsic (genetic) and extrinsic (e.g., drugs and toxins) influences on the developing brain. The altered neurobiological disposition gives rise to two main behavioural processes causing ADHD: altered reinforcement of novel behaviour and deficient extinction of previously reinforced behaviour. According to the theory, ADHD symptoms are a product of a dynamic process of the individual's adaptation to defective neurotransmission.

The authors have construed a coherent account spanning from biochemistry, via behaviour, to a reciprocal interplay between the affected child and his/her biosocial environment. The theory predicts that ADHD behaviour results from, and is continuously modified by, the dynamic context of individual predispositions and interpersonal surroundings well into adulthood. And in the case of many adults, the individual predispositions come to form the interpersonal surroundings of another individual – their child.

The individual predispositions are primarily guided by genes. However, the interplay also starts early – going back (at least) to the intrauterine life (Grossman et al. 2003; Schneider et al. 1998). By the time the child's behaviour reaches the level of abnormality qualifying for ADHD, years of active interaction have taken place. And yet, as Sagvolden et al. note, not all children presenting with the core symptoms of ADHD get identified as maladjusted. This is because the environment has been unusually insightful and supportive in guiding the child's excessive and disorganised activity into constructive creativity. The individual ADHD symptoms at different times in a person's life vary and are influenced by factors exerting either a positive or negative effect. In other words, the environment can either protect from maladjustment, or predispose to it.

Crucial here is the caregiver's ability to adjust the environment to the child's needs for optimal development of adaptive skills. The resulting behavioural style, in turn, determines the long-term consequences of the early interactions. The theory predicts that a child with ADHD finds it hard learning how to match their behaviour to the demands of a given situation. Consequently, there will be few chances for the child to be rewarded for compliant behaviour. Instead, the resulting chaotic behavioural style will only magnify the negative interactions with carers. For optimal upbringing, the caregivers have to adapt to the child's special needs by taking into account the implications of the underlying deficits and adjust their expectations and demands accordingly. As the authors spell out, "a child with ADHD requires exceptional parenting skills" (sect. 4.2, para. 3).

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.