aberrant learning and insufficient extinction of antisocial behaviours, as a result of the fundamental reinforcement/extinction deficit combined with inadequate parental and societal structuring of ADHD children's behaviour. However, reinforcement/extinction per se may not be a sufficient explanation of why some children respond to verbal requests and/or reassurance and why others refuse to comply.

Current ADHD theories have postulated a deficit of inhibition (Barkley 1997a; Quay 1988). Sagvolden et al. challenge the more current theories of ADHD and characterise inhibition as a fundamentally vague and circular concept, which is more usefully replaced by the concept of synaptic gating (Grace 1995; Levy 2004). While the dynamic developmental theory does not directly address the issue of comorbidity in ADHD, it provides a theoretical basis for understanding the pathophysiology of the core ADHD symptoms described above, which may also help to understand comorbidity. The authors draw on the work of Grace (1995; 2000a; 2000b), who showed that accumbens neurons exist in a bistable state, with their membrane potential alternating between a hyperpolarized non-firing state and a depolarised plateau lasting several hundred milliseconds, during which spike activity is generated. This bistable accumbens state allows the operation of a synaptic gating mechanism between cortical and limbic (emotional) influences on behaviour. The nucleus accumbens receives input from a number of limbic-related cortical structures, including the prefrontal cortex, hippocampus, and amygdala (Grace 2001). In particular, the hippocampus and amygdala strongly influence the ability of the prefrontal cortex to activate accumbens cell firing, allowing an emotional override to the executive system.

Goto and O'Donnell (2002) have reported timing-dependant limbic-motor synaptic integration in the nucleus accumbens (NAcc). They found that synaptic inputs from the prefrontal cortex and limbic structures interacted differently depending on their timing. Coincident inputs were likely to enhance information transmission by reducing excitatory postsynaptic potential (EPSP) amplitude variability, whereas asynchronous inputs depend on the order of arrival. Prefrontal inputs tended to dampen limbic responses, whereas limbic inputs allowed subsequent prefrontal responses by exhibiting a linear decrease in EPSP amplitude at more depolarised membrane potentials. PFC inputs were most effective in the NAcc at depolarised membrane potentials (Up state), whereas limbic membrane inputs were effective primarily during a resting membrane potential (Down state). The authors concluded that these two simultaneous mechanisms by which input (and response) selection can take place in the NAcc, depending on the state of the neurons and timing of inputs provide a mechanism for attention and emotional or motivation factors that affect responses to stimuli, with an important role in cognitive function.

A further implication of reciprocal amygdala/hippocampal/prefrontal relationships may be found in the neuroanatomical work of Heimer (2003). Heimer has described "a new anatomical framework for neuropsychiatric disorders and drug abuse." Improved electron microscopic methods have allowed the demonstration of a ventral cortical-striatal-pallidal system. This circuit (which includes accumbens/ventral striatum) is one of three reentrant circuits "anterior cingulate, lateral orbital frontal, and medial orbital frontal, related to the ventral emotional-motivational striatal domain." According to Heimer, the ventral striatal-pallidal pallidal system and extended amygdala are major components of the new anatomy of the basal forebrain. "Since the entire cerebral cortex, including the hippocampus, the olfactory cortex and major parts of the amygdala project to the basal ganglia, all major telencephalic disorders are to some extent at least disorders of the basal ganglia" (p. 1737).

For present purposes, the demonstration of ventral striatal-hippocampal-prefrontal re-entrant circuits (including accumbens) allows for the possibility of iteration of emotional reactions from amygdala through hippocampus and prefrontal cortex, allowing executive monitoring of emotional behavior. Thus, impaired synaptic gating at integrative locations such as the accumbens will interfere with the development of controlled behavior. Sagvolden et al. state that behavior is gradually brought under discriminative control, including the establishment of verbally governed behavior as a result of training. They describe verbal stimuli as contingency specifying stimuli. However, rather like Chomsky's (1959) criticism of Skinner's explanation of language, the operant explanation of verbally governed behavior does not explain the sometimes immediate and dramatic changes in oppositional behavior of ADHD children on stimulant medication. These changes require a biological explanation, which may relate to integration at the above re-entrant circuits and between these circuits. This does not diminish the important role of language in human development. In ADHD, language deficits may well limit the scope of reentrant circuits in the elaboration of behaviour.

What is the purpose of a new behaviorally based dynamic developmental theory of ADHD? The perspective of the educational psychologist

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Abstract: In Sagvolden et al.'s conceptualization of how a poor behavioral, social, and academic repertoire arises from an impaired interaction with the environment of an individual with a neurological disorder, we see a convergence between the medical diagnosis and the functional assessment on which the behavioral educational approach is based. If children with such a disorder do show delay-of-reinforcement steepened gradients, it is possible to predict their behavior under given circumstances. This could bring us to more precise diagnostic criteria and better intervention techniques.

In the advancement of science, literature reviews accomplish a fundamental role: Occasionally they try to sum up the state of the art, that is, what is known on a certain subject at a certain point in time, and try to point up new insights and suggestions to understand given phenomena. The target article by Sagvolden et al. thus prompts the fundamental question: Are we in need of a new point of view on the attention-deficit/hyperactivity disorder (ADHD) syndrome?

For many years, neuroscientists, psychiatrists, and psychologists have been trying to shed light on the deficits supposedly underlying attention-deficit/hyperactivity disorder (ADHD), with recent studies mostly supporting the idea that ADHD is a result of deficits in executive control and regulation that influence emotional and cognitive processes (Barkley 1998). This approach has been so influential that, starting from the end of the 1990s, ADHD has been commonly regarded as resulting more from neurological and genetic factors than from environmental events.

However, a medical diagnosis of ADHD does not necessarily imply that all the children with it show the same degree of disability, and a functional assessment is needed to fully address their behavioral and academic repertoires, if a rehabilitative intervention is to be implemented. Educational approaches based on functional analysis of the behavioral and academic repertoire, though pursuing a parallel but not strictly related path to the neurosciences, have been reported to work well. In literature a wide array of interventions planned to modify children with ADHD behavior ranging from behavioral procedures such as token economies (e.g., Williams et al. 1989), daily report cards (e.g., Burkwist et al. 1987), self-monitoring (e.g., Edwards et al. 1995), verbal praise (e.g., Williams et al. 1991) and contingency contracting (e.g., Newstrom et al. 1999), to cite just a few, have been published. These and other procedures have been found to be effective in enhancing school performance and social behaviors of children with ADHD.

The two levels of analysis (neurosciences and behavior) and interventions (pharmacological and educational) have evolved strictly separated, though aiming at the same target: understanding the disorder and providing ways to deal with it. Sagvolden et al.'s article gives new insight and useful suggestions for dealing with ADHD, being able to correlate either the behavioral repertoire or the neurological impairment, both at the level of the brain pathways and at the level the neurotransmitters.

If it is true that a neurological deficit exists, then nevertheless it translates into an impaired interaction of the individual with the surrounding environment. Sagvolden et al.'s conceptualization of how a poor behavioral, social, and academic repertoire arises from an impaired interaction with the environment of an individual with a neurological disorder is consistent with a behavior analytic vision of development (see e.g., Bijou 1966). However, it is, to our best knowledge, the very first time that a thorough and coherent picture is given at both the levels of structure and of function.

The approach that is suggested can be easily translated into better prediction and control. If these children show delay-of-reinforcement steepened gradients, it is possible to predict their behavior under given circumstances. This could bring us to more precise diagnostic criteria, as suggested also in Catania's precommentary. In the point made by Sagvolden et al., we do see a convergence between the medical diagnosis and the functional assessment on which the behavioral educational approach is based. At the beginning of the 1980s these two ways of looking at developmentally retarded individuals who showed a common pattern of behavior were definitely separated. In most cases they were two antagonistic ways of conceptualizing behavior disorders. Although in some fields of the psychiatric domain, for example, anxiety or depression, functional analysis and medical diagnosis continue to be separate, this is not the case when looking at genetically based developmental disorders like fragile X or Asperger syndrome. The structural description of the behavioral phenotype, given at the medical level, is complemented by the functional description of behavior. Though not specifically linked to a precise genetic change, the same, we think, could apply to ADHD.

Up to now there has been no concrete medical test to diagnose ADHD, which often makes the diagnosis of ADHD subjective. Vague criteria in diagnosis lead to confusion in epidemiology, so that the numbers of those diagnosed range up to 17%, as reported by 19 community-based studies in the past two decades (Scahill & Schwab-Stone 2000). The differences in epidemiological surveys are a consequence of the choice of informant, methods of sampling and data collection, and, above all, the diagnostic definition. Such a big number is not confirmed by our daily experience. One should observe three to four subjects per class of 20 to 25 pupils – which is not the case. This weakness in diagnostic precision exposes scientific procedure to easy criticism, which states that ADHD does not exist up to the point where it is necessary to publish consensus statements (Barkley et al. 2002). The loose descriptive category of the DSM-IV (American Psychiatric Association 1994) or the AAP criteria (Herrerias et al. 2001) can be better restricted on the behavioral level by registering the ADHD child behavioral pattern with operant procedures, distinguishing true "pathology" from false positive fidgety children or from a child with other behavioral disturbances not directly related to ADHD. Objective behavioral based procedures that analyze delay-of-reinforcement gradients might become a better substitute for subjective judgment of behavioral patterns.

A parallel analysis may also be made as far as functional analysis is concerned. A functional analysis that will not take into account the decay steepness of the curve might overlook the fundamental unit of individual–environment interaction. The percentage of failure, as shown by various studies (e.g., MTA Cooperative Group 1999), might be a result of the delivery of reinforcing stimuli outside the boundaries of the "curve" allowed by each single subject. This point might be empirically addressed and if proven, it might show ways to improve educational techniques with children showing ADHD.

The analysis of the conjoint efficacy of stimulant drugs and behavioral procedure might also benefit from conceptualizing ADHD as an anomaly of delay-of-reinforcement gradients. The aforementioned Multimodal Treatment Study for Attention-Deficit Hyperactivity Disorder (MTA) study demonstrates that using behavioral techniques in children under psychostimulant medication is the best strategy if compared to either drugs or behavioral techniques alone. A child's behavior might be tested to analyze the steepness of the gradient curve under two different conditions, before and after drug administration. It has been demonstrated that drug administration increases sensitivity to reinforcement in ADHD individuals (Murray & Kollins 2000; Northup et al. 1997), but no research has pointed to the core shown by Sagvolden et al. The difference in the curve between response-to-reinforcement schedule as an effect of training and as an effect of the drug might be related to an index of high or low probability of success in the intervention as a consequence of adding the drug to behavioral intervention. This consideration directly prompts another one. Based on objective and clearly demonstrated data, collaboration between medical personnel and educators, specifically, psychologists and teachers, can be strengthened.

Individuals with learning disabilities carry an increased risk of physical, behavioral, and psychiatric problems that can severely affect the quality of life and increase burden of care. Sagvolden et al.'s analysis aims also to this specific point. Early intervention on children has been looking traditionally at ways to increase their attention span progressively shaping attentive behavior with easily edible (small) frequent reinforcers. We now have a way to measure, refine, and better control the basis of behavioral intervention.

In the late 1970s the concept of prosthetic environment came up in the field of behavior analysis and modification. There are many ways to organize a prosthetic environment, but the core business is always the same: programming and establishing antecedent and consequent conditions that are really effective for individuals with special needs. The concept of prosthetic environment has been very successful in helping people with physical impairments and disabilities, and in reducing their handicaps with regard to typically developing subjects. Less successful has been the concept of helping people with "mental" atypical development. There are many reasons for that, including the idea of mind, the idea of freedom, and the difficulty in arranging a tailored prosthetic environment for cognitive disabilities. However, this is, in our opinion, the challenge of the future, and, fortunately, a computer-based world can afford it.

Every single behavior of our life is videotaped, computer recorded, analyzed, controlled, and so on. Does all this technology allow us to implement interventions to enhance the academic and social repertoire, making the environment conducive to the delivery of specific reinforcement?

To conclude: in our opinion, Sagvolden et al. provide us with an explanation at a more parsimonious level than that of the deficits in executive control and regulation, eventually taking into account higher levels such as attention delays and failure to outline goal-oriented instructions and rules of behavior, while maintaining a strict correlation with basic research, medical practice, and education or rehabilitation of individuals with ADHD.

The question that still remains on the ground is how to move this hypothesis from the animal lab to humans and experimentally test individuals with ADHD in comparison to those without. If Sagvolden et al.'s vision is empirically confirmed, we need to definitely refocus the theoretical approach to ADHD cited in the introductory part of this commentary. So, on the basis of all the points we have considered here, including the one in the preceding paragraph, we heartily answer our question without any doubt: Yes, we do need the new point of view.

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