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A New Understanding of
ADHD
in Children and Adults
Executive Function Impairments

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HOW TREATMENTS FOR ADHD AFFECT THE BRAIN AND IMPROVE EXECUTIVE FUNCTIONS

Medical and psychological treatment usually has two goals: 1) to minimize suffering and alleviate impairment; and 2) to improve functioning. Treatment also has two primary constraints: 1) availability of knowledgeable persons and resources necessary for adequate treatment; and, 2) willingness of the patient to engage in the necessary procedures.

Education about ADHD: A Critical Component of Treatment

For ADHD, adequate education of the patient and family about the nature and treatment of ADHD is an essential aspect of minimizing suffering and damage; it is also a critical factor in eliciting willingness of the patient to engage adequately in the treatment process. When not adequately treated, ADHD can produce considerable psychological damage. It can cause a person to feel stupid and lazy when they are neither. Children and adults significantly impaired by ADHD often suffer years of frustration, demoralization and humiliation from their inability to do what most of their peers are able to do and what they themselves and those around them expect them to do. Repeatedly they are confronted with reminders of their failure to fulfill hopes and expectations held by themselves and those around them.

Often these difficulties are exacerbated by the persisting inconsistency that is typical of those with ADHD. When parents, teachers, co-workers and friends observe repeatedly that an individual with ADHD is able to focus very well and exercise excellent executive functioning for playing a sport or video game or some other favored activity while not demonstrating even adequate EF for many other tasks that may be more important, they understandably respond with "If you can do it for this, why don't you do it for that?" They tend to assume that the problem is a simple lack of willpower, an unwillingness to do here and now what this person has clearly been able to do in another task or setting.

HOW TREATMENTS FOR ADHD AFFECT THE BRAIN

Most patients seeking an initial consultation for ADHD and many of those significant in their life are likely to be operating with an implicit, if not explicit assumption that the person with ADHD is stubbornly refusing to do what they clearly seem able to do when they are motivated to do so. And it is assumed that the needed motivation can be generated by the individual if only they were willing to try harder more often or were more frequently and forcefully reminded to do so.

The educational aspect of treatment provides opportunity to explicitly recognize and challenge such erroneous assumptions by providing the patient and family a clear and understandable factual description of the nature of ADHD, the wide scope of the syndrome, its developmental aspects and its situational specificity. Education of patient and family about ADHD should also include honest description of the nature, uses and limitations of treatment. It is important that such education be provided not only for the patient, but also for others with whom the patient is currently involved, parents and teachers for children, spouses and/or others for adults—anyone whose support is needed and who may be likely to exacerbate the patient's demoralization or to undermine their motivation for sustaining appropriate treatment. Effective education about ADHD can help to reduce the patient's suffering from demoralization; it may also help to increase their willingness to engage in the process of treatment.

ADHD is essentially a problem in the chemical dynamics of the brain. Changes in the environment of the affected individual sometimes can make a positive difference: increased opportunities to earn positive recognition for appropriate performance, reduced exposure to unnecessary frustration, introduction of more effective strategies for work and learning, more adequate support, removal from toxic social environments, etc. However, though environmental changes may be quite helpful, in themselves, they are rarely sufficient to alleviate the syndrome of executive function impairments characteristic of our new model of ADHD.

Mechanism of Action in Medication Treatment for ADHD

The most effective treatment for ADHD impairments is usually a regimen of medication to facilitate more effective functioning of the brain's self-management system. A regimen of appropriate medication, well-tailored to the specific individual, cannot cure ADHD, but for about 80% of those affected it may provide significant reduction of impairment and substantial improvement of functioning. Such benefits from medication treatment are achieved by transient modifications of the dynamics of the chemistry of the brain.

Often people speak of ADHD as caused by "a chemical imbalance in the brain." This makes it sound as though the problem were a lack or excess of sufficient salt in the soup of the cerebral spinal fluid which continually flows around the brain. That is simply wrong. The impairments of ADHD are not due to a global excess or lack of a specific chemical within or around the brain. The problem is at the level of synapses, the infinitesimal junctions between certain networks of the 100 billion neurons that manage activities within the brain.

When an electrical impulse (action potential) travels along neural networks, it encounters numerous junctions between neurons over which the electrical impulse must travel with an action somewhat like a spark plug. To continue traveling down the network, this action potential must jump across each synapse from one neuron to another. This jump requires the release of micro-dots of the neurotransmitter chemical made in each neuron and released when the electrical impulse arrives at the edge of the synapse for each neuron.

Volkow and colleagues (2002a, 2002b) and Swanson, et al. (2011) have described the mechanisms by which stimulant medications improve this communication between neurons to alleviate impairments of ADHD. Using PET scans they have shown that stimulant medications such as methylphenidate operate in multiple synapses of the brain where they bind to dopamine transporter proteins (DAT) which "suck up" dopamine released from the presynaptic neuron, thus terminating its connective action. This binding onto the transporters, when sufficient, (usually 50 to 70% occupancy is required) slows reuptake of the dopamine, leaving more of it in position to make and briefly sustain a better connection; this results in more effective communication between that presynaptic neuron and the post-synaptic neuron with which it is attempting to communicate. A similar mechanism is involved in the actions of medications for ADHD that target neuronal communication between neurons utilizing norepinephrine, the other primary neurotransmitter involved in ADHD impairments.

This mechanism of slowing reuptake of dopamine is the same for both methylphenidate and amphetamine medications for ADHD; however amphetamines have a secondary mechanism. They also stimulate some additional release of dopamine as well as slowing its reuptake. For many with ADHD, methylphenidate and amphetamine medications work equally well, though one type may cause adverse effects while the other may not. However, if neurons of an individual are chronically releasing an insufficient quantity of dopamine into the synapse, slowing reuptake is not likely to produce significant improvement in synaptic communication because the insufficient quantity of

dopamine constitutes a rate-limiting effect which may not allow adequate correction of the inter-neuron communication problem. In such cases, amphetamine may be more effective than methylphenidate.

Arnsten and Li (2005) have reviewed the influence of both dopamine and norepinephrine on executive functions. Citing relevant studies of both animals and humans, they highlight the sensitivity of the PFC to its neurochemical environment within the brain. They noted that small changes in the catecholamine modulation of PFC cells can have profound effects on the capacity of the PFC to guide behavior utilizing working memory. They argue that low doses of D1 agonists improve working memory and attention regulation while excessively high levels of dopamine release impair PFC function. Similarly, low to moderate levels of norepinephrine have important beneficial effects on the functioning of the PFC while excessively high levels, such as may be released in stress, contribute to impaired PFC function. Another review of the separate and interactive roles of dopamine and noradrenaline in the pathophysiology and treatment of ADHD has been provided by Del Campo, et al. (2011).

Most of the research done on medications approved by the U.S. Food and Drug Administration (FDA) thus far for treatment of ADHD has been focused on stimulants (methylphenidate and amphetamines) which act to increase levels of both dopamine and norepinephrine in neuronal networks. One non-stimulant medication, atomoxetine, which functions as a selective norepinephrine reuptake inhibitor acting specifically upon the norepinephrine and not on the dopamine, has also been the focus of considerable research. Long-acting formulations of two alpha-2 agonists, guanfacine, and clonidine, have more recently been approved for treatment of ADHD, either as monotherapy or as adjunctive treatment with stimulant medications. These more recent additions to the approved medications for treatment of ADHD have not yet been systematically tested on the broader range of ADHD-related executive functions which is the primary focus of this book.

