Your Employee Assistance Program is a support service that can help you take the first step toward change.

Changes in The Brain with ADHD

Researchers have studied both brain structures and levels of brain activity in individuals with ADHD. The studies suggest that people with ADHD have brains that are about 4% smaller than normal. Most of the size reduction occurs in the prefrontal cortex and the anterior temporal areas. Size reductions in the prefrontal cortex could impair an individual's self-awareness and his or her ability to manage emotions and behavior, as well as increase impulsive, angry, and hyperactive behavior. The anterior temporal areas influence hearing functions and language skills, including the ability to comprehend and/or communicate verbally, as well as the perception of non-language sounds. Decreases in this area of the brain could also influence memory and learning.

An overall smaller brain volume can also impair executive functioning, the term used by psychologists to refer to higher level skills such as organizing information, sustaining attention, and determining a reasonable course of action based on the information available. Whether or not decreased brain size is a cause or a consequence of ADHD remains to be seen, as more research on the topic is conducted.

Other researchers have found that children with lesser amounts of gray matter (the component of nerves composed of cell bodies that helps to send sensory or motor messages throughout the central nervous system) in the brain seem to have a harder time paying attention than do children with more gray matter. Research also suggests that there may be a subset of children with ADHD who have larger frontal lobes than normal. These children seem to have predominately hyperactive behavior. More research is necessary to determine whether there are truly brain-based differences between the three ADHD subtypes.

One line of brain-based research suggests that individuals with impulsive and hyperactive forms of ADHD are more sensitive to immediate rewards (rather than delayed rewards). So, these people are more likely to engage in a high amount of behavior (over activity), rather than being able to wait patiently for something rewarding to occur later on (e.g., obtaining a good grade for a well-written research paper in school). Children with ADHD also seem to respond poorly to punishment.

Neurotransmitter Changes with ADHD

Scientists are also investigating the causes of ADHD at the neurotransmitter level of brain functioning. Neurotransmitters are chemical messengers that occur in the brain and central nervous system. These neurotransmitters carry messages between neurons, the cells in the brain and central nervous system. Previously, scientists believed that low levels of dopamine (a neurotransmitter that impacts movement, mood, motivation, and attention) caused ADHD.

More recent evidence suggests that the relationship between dopamine and ADHD is bit more complicated. Researchers have found that in addition to reduced overall levels of dopamine, individuals with ADHD have a higher concentration of dopamine transporters (the specific chemical element that moves dopamine from the synapse into the neuron, after which the dopamine action is terminated and reabsorbed by neurons; a process known as reuptake) in the striatum (the part of the brain that is responsible for planning ahead). This may sound confusing, but in individuals with ADHD, the small amount of dopamine present doesn't have enough time to exert its effects before it is reabsorbed by neurons (nerve cells in the brain and central nervous system).

Additional evidence of the link between dopamine and ADHD comes from studies of the effects of medication on symptoms. Ritalin (a commonly used and very effective stimulant medication) blocks

dopamine transporters and prevents the reuptake of dopamine after it is released. Other neurotransmitter studies suggest that ADHD symptoms may be also be caused by lowered levels of norepinephrine and serotonin (two other neurotransmitters that influence brain function) in the cerebellum.

Medication Studies

As suggested above, additional evidence that ADHD is a biological disease comes from observing what happens to the brains and brain chemistry of individuals who take medications that successfully treat symptoms.

In 1937, amphetamine (a central nervous system stimulant) was used successfully to treat a group of children with ADHD-like behaviors, including limited self-control, aggressiveness, defiance, resistance to discipline and extreme emotionality. Later studies suggested that stimulant medications also seemed to reduce disruptive behavior and improve academic performance. During the 1950's, further evidence suggested that amphetamines were extremely helpful in the treatment of hyperactive children. The FDA approved dextroamphetamines (e.g., Dexedrine) for treating childhood disorders in 1958. In the 1970's, stimulant medication was the most popular treatment for ADHD. The use of Dexedrine decreased from 1962 to the mid 80’s, as Ritalin became the medication of choice.

Experts initially thought that ADHD was caused by excessive activity in the brain. Since stimulants increase overall activity in the brain, their use in the treatment of ADHD seemed counterintuitive. In other words, why would you give someone whose brain is already overactive a drug that made his or her brain even more active? The generally accepted view at the time was that stimulant medication had a unique paradoxical, or opposite, effect on children with ADHD.

Since then, however, research has shown that ADHD is actually a result of lower levels of brain functioning, decreased dopamine levels, and frontal lobe functioning deficits. Children with ADHD have cognitive "sluggishness", or a slower thinking process than their peers. Stimulants raise the brain activity levels of children with ADHD to within the normal range. In addition, stimulants also increase an individual's ability to screen out irrelevant stimuli, more successfully focus on the designated task, and manage impulsive behavior.