



Early Acquisition of American Sign Language: An Innovative Approach for Treating Attention-Deficit/Hyperactivity Disorder

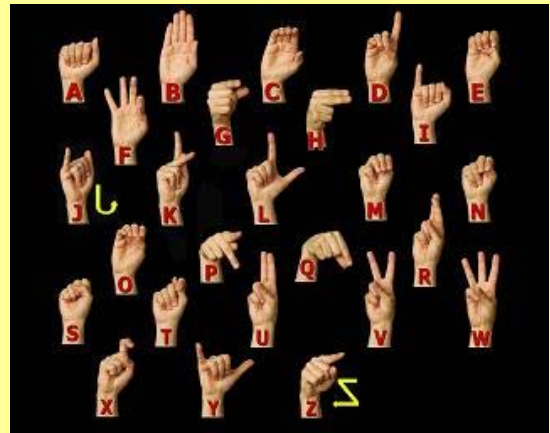


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Overview

Imagine how frustrating it would be to have a child who suffers from Attention-Deficit/Hyperactivity Disorder (ADHD). Now add to that the expenditure of thousands of dollars spent on psychotherapy, behavior modification, biofeedback, and multiple medications, only to discover that nothing has worked. The current treatments for ADHD frequently fail, which is why researchers must find innovative ways to treat the diagnosis. We propose that those answers may be found in the combined literature of brain research and American Sign Language (ASL).

One overlooked approach to the treatment of ADHD is ASL. If ASL is taught to hearing children, prior to puberty, which is the optimal time to learn a second language, it can increase activity in certain parts of the right hemisphere (RH) of the brain. Activity was found in portions of the RH of the brain of native signers, people who learned ASL as a primary language prior to puberty, but that activity was not found in those portions of the brain of people who learned ASL after puberty. This indicates that the brain responds differently to the acquisition of ASL, depending on the age of the child. Approximately 3% of children with

ADHD have a smaller RH of the brain, specifically in the prefrontal cortex (PFC) and basal ganglia, compared to normal children without ADHD. With examples from published research, we explain how ASL may help to increase the size of the RH of the cerebral cortex in children with ADHD.

Using animal models, research has shown that it's possible to increase the size of a rat's cortex by placing it in an enriched learning environment. ASL naturally creates an enriched learning environment for children, therefore, if ASL can cause cortex growth and help the parts of the brain that cause ADHD to function more normally, then early acquisition of ASL may improve attention and decrease impulsivity, inhibition, and hyperactivity. This innovative and safe way to create changes in the brain, may be the answer for those who have tried just about everything but find their child is still suffering from the debilitating symptoms of ADHD.

The information contained in this paper provides the basis for future researchers to design a longitudinal study to test the hypothesis. If there are positive findings from this study and early acquisition of ASL can reduce negative symptoms of ADHD, then ASL will become the newest treatment in the battle against ADHD, or may even inoculate against the development of the disorder altogether, if intervention occurs early enough.

WHAT IS THE CONNECTION BETWEEN ASL AND ADHD?

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| ❖ Approximately 3% of children with ADHD have a smaller RH of the brain, specifically in the PFC and basal ganglia, compared to normal children |
| ❖ ASL stimulates the parts of the brain involved in visual, auditory and kinesthetic learning and creates an enriched learning environment |
| ❖ Learning ASL prior to puberty stimulates and activates certain parts of the right hemisphere of the brain that may be involved in ADHD |
| ❖ We believe ASL may help to increase cortex size, improve brain function, and have a positive effect on reducing ADHD symptoms |

ASL utilizes all learning styles

One obstacle which affects a child's ability to learn is suffering from symptoms caused by ADHD. Three to 5% of children in the population are affected with ADHD which accounts for a disproportionate number of disruptive incidents at schools (Shaffer et al., 1996). The negative effects of one child's struggle with inattention or hyperactivity, ripples through the classroom like a pebble in a pond. Children with ADHD have difficulty attending to, and processing all of the sensory information in the environment. This is a problem because the information they transfer into long-term memory is not an accurate representation of the original communication, resulting in problems with comprehension and academic achievement, which directly affects their self-esteem.

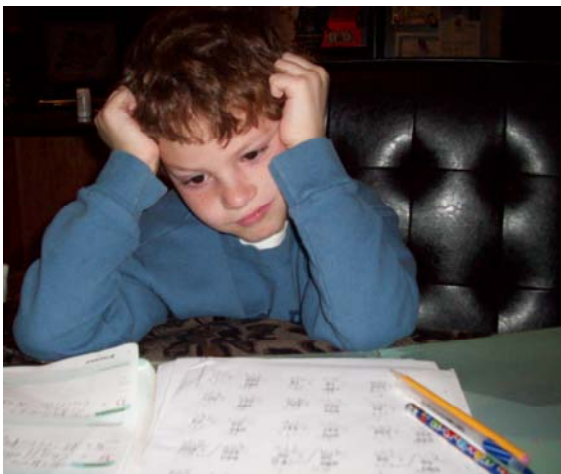
For years educators have been aware of the advantages of introducing children to enriched learning environments that reinforce a child's primary learning style whether they are a visual, auditory, or kinesthetic learner. ASL is a legitimate language that can be taught to hearing children in a way that utilizes all three learning styles and creates an enriched learning environment. Because ASL is experienced as a fun activity, the child becomes an active participant in their learning which helps to encourage motivation. According to Thompson (2004) sign is received visually and spatially by the RH of the brain and is subsequently processed by the left hemisphere (LH).

A visual learner often thinks in pictures and can more easily comprehend a lesson if provided with visual aides. They understand the teacher's intended communications by paying close attention to the teacher's facial expressions, eye contact, hand gestures and body language. For hearing children, the teacher can utilize auditory stimulation when teaching ASL by explaining verbally what is being communicated, as they

are demonstrating the signs or fingerspelling. An auditory learner interprets the underlying meaning of speech through tone, pitch and voice speed. Kinesthetic learning takes place when the child signs and fingerspells which enables them to feel the language. Kinesthetic learners absorb information more easily by physically interacting with their environment through movement, drawing or taking notes (Gardner, 1983; Miller, 2001). The combination of skills used in ASL establishes an enriched learning environment and facilitates a multiple imprint on the child, which enhances their learning and memory skills (Daniels, M., 1996).

Meeting criteria for an ADHD diagnosis

ADHD often co-exists with other problems, including learning disabilities, anxiety, depression, bipolar disorders, or disruptive behavior disorders. Any combination of these emotional challenges can lead to an increased risk for drug abuse and/or suicide (Spencer et al., 1999; Mannuzza et al., 1998; The MTA Cooperative Group, 1999). ADHD is subjectively diagnosed in early childhood through observations, interviews, parent and teacher rating scales, and administration of the Test of Variables and Attention (TOVA) which is a computerized test given to children who are at least four years of age (Parasnis et al., 2001).



According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV), to receive a diagnosis of ADHD, the following criteria must be met:

- Symptoms are manifested prior to age seven and are present in two or more settings (home, school, or work)
- There is a minimum of six symptoms of inattention and/or six or more symptoms of hyperactivity-impulsivity for at least six months
- The severity of the symptoms rises to a degree that is maladaptive and inconsistent with the child's developmental level
- The symptoms are not better accounted for by another severe physical or mental disorder

(American Psychiatric Association, 1994)

How brain function impacts ADHD

To assess how ASL may help children with ADHD, we began by looking at the parts of the brain that are directly involved in the disorder. If ASL stimulates the parts of the brain causing the disorder, then any means to improve brain function in these areas may help to relieve symptoms. Magnetic Resonance Imaging (MRI) brain scans of children with ADHD indicate abnormal brain structures. On average, the RH's total cerebral volume is approximately 3% smaller in children with ADHD, compared with normal children, specifically in the PFC and basal ganglia, which includes the caudate nucleus and globus pallidus (Castellenos et al., 1996).

Functional imaging techniques, such as the Single Photon Emission Computed Tomography, and MRI have revealed that these three areas in the RH of the brain are responsible for **inattention**, **hyperactivity**, and **impulsivity**. Using an automobile analogy, Castellanos et al., (1996) describes the **PFC** as the steering wheel (the brain's command center) which causes **problems with attention** when it is not functioning properly. The **caudate nucleus** acts like the accelerator (gas pedal) and when it isn't working correctly, the child has too much energy and is **hyperactive**. When the **globus pallidus** is dysfunctional, it's as if the brakes aren't working properly and the child acts in an **impulsive** and less inhibited manner. The underlying mechanisms causing ADHD originate from communication problems between these three brain regions (Castellanos et al., 2001).

Children who had severe symptoms of ADHD had a thinner PFC and those who had better outcomes and less severe symptoms, were found to have increased thickness in the right parietal cortex in the PFC (Shaw et al., 2006). The children with fewer symptoms had an enlarged hippocampus as well, which was thought to be a compensatory response enabling the child to cope better with the problematic stimulus-

seeking and impulsive behaviors. Plessen et al., (2006) showed that the amygdala was smaller in children with ADHD and observed there was a poor synapse connection between the amygdala and the PFC which may contribute to a child having problems paying attention, or acting less inhibited.

An important finding by Kassubek et al. (2004) was that increased brain activity was seen in the RH angular gyrus in native speakers, who learned ASL as a primary language prior to puberty. Subjects who knew ASL showed brain activity in the RH occipito-temporal cortex which was not found in English speaking subjects. This finding might have something to do with the visuospatial component of ASL, reflecting the greater kinesthetic movement involved in ASL. Another area of the brain found to have activation due to ASL acquisition was the left lateral superior temporal lobe (Broca's area) which is involved in supporting sign language comprehension. We believe that early acquisition of ASL can duplicate this type of brain activation which would ultimately have a positive effect on an ADHD brain.

According to Newman (2001), the critical period for language acquisition is between the ages of two and 10. Children who do not learn language before puberty, are unable to fully acquire and use the syntactic principles of language. There is evidence that a critical period for second language acquisition also exists. The later in life a second language is acquired, the less proficient one is in that language, particularly with respect to syntax and phonology. There is also a sensitive period in development when certain regions of the brain can be recruited for the processing of ASL. Native signers, those who learned ASL as a primary language, showed activity in the LH and along the RH superior temporal sulcus. However, only native signers who learned ASL prior to puberty showed further activation of a RH inferior parietal region. This suggests there is a limited time period when this region can be recruited into the language system.

ASL may improve right hemisphere functioning

At birth the cerebral cortex, the outer part of the brain, contains 100 billion unconnected neurons. As infants are stimulated with novel and challenging experiences, the highly specialized neurons branch out and create an interwoven tapestry of neural connections. By the child's first birthday, 100 trillion synaptic links have formed.

These connections facilitate the electrical signals that transmit messages throughout the body enabling speech, memory, logical and emotional responses, consciousness, voluntary movement and the ability to interpret sensations. The quantity and strength of connections determines the size and thickness of the cortex which fluctuates (Shankle, et al., 1998). Neuroplasticity describes the brain's dynamic capacity to change and reorganize neural pathways after new experiences and learning takes place.

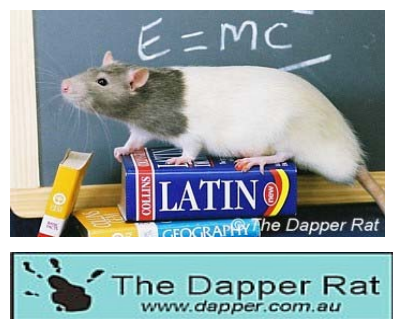
According to Willis (2006) the frequently used neurons and connections remain and flourish, while the rarely used neural pathways undergo a refinement process called synaptic pruning which eliminates the unused neural connections. The three main pruning periods are during the first six months of life, at puberty, and in old age. When there is insufficient neuronal activity, approximately 50% of unused brain tissue will be pruned away by a chemical reaction called apoptosis (programmed cell death). The catalyst to pruning is a reduction in blood flow to the unused connections which leads to an increase in production of calcium ions. In this chemical environment, an enzyme called calpain is secreted which ultimately causes the cell to self destruct. Ultimately, the environment a child grows up in and the stimulation they receive plays a key role in influencing neuroplasticity and pruning.



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Animal models show increased cortex size in enriched learning environments

Using the rat animal model, Bennett, et al. (1964) showed that neuronal connections grew when the rats were exposed to enriched environmental stimulation and opportunities to learn. He compared the impact of three different types of living environments on the dynamic structures of the brain.



IMPACT OF DIFFERENT TYPES OF LEARNING ENVIRONMENTS ON BRAIN STRUCTURE

Enriched Living Environment	Twelve rats lived in a large cage with wheels, ladders, and small mazes	Largest increase in cortex size, primarily in the visual cortex as compared to the rats in the Standard Group
Standard Group/Social Condition	Three rats lived in a small cage without toys	Larger cortex size than rats in the Impoverished Living Group
Impoverished Living Group	One animal lived in a small cage without exploratory objects	Smaller cortex size than rats in the Standard Group

After 30-60 days, the rats were anesthetized and brain samples of the rats were evaluated. The rats living in the enriched environment showed an increase in dendrite branching, increased nerve cell size, and increased length of postsynaptic thickening primarily in the visual cortex, as compared to the brains of the standard group. The rats living in the impoverished group had a decrease in the size of their cortices as compared to the standard group (Bennet et al., 1964; Chenn and Walsh, 2003; Herculario-Houzel et al., 2006). The results showed that the brain structure of the rats was altered as a result of either stimulation or deprivation.

Rat studies performed by Black et al. (1990) and Kleim et al. (1996) determined that learning was required to induce synaptogenesis, the formation of new synapses. A group of adult female rats were trained on a challenging acrobatic course requiring acquisition of a motor skill task. This group was compared to two other groups: one that was allowed to run freely on a treadmill, with very little opportunity for learning; and a second group who were inactive during the experiment. Findings indicated the rats trained on the acrobatic course had a larger number of synapses per neuron, in both motor and cerebellar cortices, compared to the rats in the other two groups. Other researchers have also shown that the thickness of rat cortices can be increased if exposed to enriched learning environments (Bennett et al, 1964; Markham and Greenough, 2004).

Research has shown that an enriched learning environment can stimulate growth of a rat's cortex, therefore, we believe it's possible for a human's cortex to also increase in size when introduced to the correct kinds of stimulation and opportunities to learn. If ASL is introduced prior to adolescents and can affect changes in brain structure, reduce pruning, increase the size of the cortex, and improve functioning in the RH of the brain, then ASL may be an effective treatment tool for improving or preventing ADHD symptomatology.

Current treatment modalities for ADHD



The medications prescribed for ADHD are stimulants in the forms of Methylphenidate (Ritalin SR and Concerta), Clonidine (Catapress, Dixarit), Dextroamphetamine (Dexedrine) and Amphetamine (Adderall XR) (Hunt, 2006; King et al., 2006). Although stimulant medications are generally safe to use and show positive effects for patients with ADHD, medication management is not a parent's first choice, due to possible side effects including decreased appetite, impaired ability to sleep, and problems related to growth. Other medications used to treat ADHD are antidepressants such as Bupropion Hydrochloride (Wellbutrin) but that is not as common as the stimulant prescriptions.

The first non-stimulant medication approved by the FDA to treat ADHD is atomoxetine (Strattera). Although the specific mechanisms explaining the effectiveness of Strattera is still unknown, it is thought to act as a selective norepinephrine reuptake inhibitor which increases the availability of the neurotransmitter norepinephrine at the synaptic receptor sites (Simpson et al., 2004). The end result is an increase in attention, and better control over motor activity and impulsive behaviors. Some of the possible side effects of Strattera are decreased appetite, nausea, vomiting, fatigue, and difficulty sleeping (Wang et al, 2007; Cheng et al., 2007; Faraone et al., 2007). Since many parents worry about side effects and rising out-of-pocket expenses for medication, it is important to provide parents with another non-traditional therapeutic approach to enhance treatment for their children afflicted with ADHD.

As of 2008, the current treatment options for ADHD primarily include psychotherapy, behavior modification and medications. There has been some temporary success using biofeedback methods but there is no scientific evidence that shows biofeedback causes improvements in symptoms of ADHD permanently. According to Goldstein and Ingersoll (2007), "Although this treatment [EEG Biofeedback] has become quite popular and is marketed throughout the country, there continues to be limited, published peer reviewed research to support its use."

Recognizing the importance of diet, stress reduction, exercise and obtaining sufficient amounts of sleep are important subjects when discussing children's health and behavioral issues. Although limiting sugar has not been proven effective in reducing symptoms of ADHD, it is always a good idea to limit junk food, and provide a balanced diet, including fresh fruit, vegetables, whole grains, and proteins.

Since children with ADHD are often restless, and stressors from school and home can cause anxiety, meditation and relaxation exercises which can be easily taught and practiced can be helpful in reducing anxiety, stress and agitation. Physical exercise also helps to reduce anxiety and depression, and receiving an appropriate amount of sleep reduces irritability and behavioral problems.



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Designing a longitudinal study to test the hypothesis

This hypothesis can be tested with a longitudinal experiment studying subjects who have at least one parent diagnosed with ADHD because there is a large genetic component to the disorder. Through identical twin studies and by studying gene association, it is known that ADHD is predominately genetic in origin with a mean heritability of 0.8 (Faraone, 2000) which means 80% of the contribution to ADHD is due to genetic factors. These studies also clearly show that ADHD involves more than one gene in the disease (polygenic), including several genes in the catecholamine (neurotransmitter) system alone. Specific alleles (or forms of these dopaminergic genes) are preferentially transmitted from parents to their children.

Some of these include the 480 bp dopamine transporter 1 (DAT1) allele and the dopamine receptor 4 (DRD4) 7 repeat allele (Swanson et al., 2000). In other words, children with a parent affected by ADHD carry a higher risk of exhibiting symptoms of ADHD.

The longitudinal study would explore the hypothesis that ASL, if introduced before puberty, has the potential to impact the areas of the brain causing ADHD, thereby influencing and reducing ADHD symptoms. Two groups, the experimental and control, would have randomly assigned subjects. Both groups would undergo pre-experimental testing using the Conners' parent and teacher rating scales and the TOVA to diagnose pre-existing ADHD. MRI scans would determine whether there are any abnormalities in the brain. ASL instruction would be provided to the children in the experimental group for at least one year. The control group would not receive any ASL instruction. At the end of the instruction period, post-testing would occur and both pre and post experimental testing results would be compared and analyzed to see if there are any significant differences between the two groups, in terms of hemisphere size on the MRI scans, and degree of severity of symptoms of inattention, hyperactivity and/or impulsivity. The data would be analyzed and a determination would be made whether the findings identify a significant test result. Any significant differences between the two groups could be attributed to the intervention of the ASL instruction.

Conclusion

In order for a child diagnosed with ADHD to reach their highest potential, it is important to find innovative ways to treat the symptoms of ADHD because the treatment modalities currently being used today often fail. We have set forth our hypothesis of why we think ASL is an innovative way to treat ADHD and its potential benefits. We believe that early acquisition of ASL, which is processed by the brain as a legitimate language, (Emmorey et al., 2002 and 2004), may improve attention and reduce hyperactivity and impulsivity if introduced between the ages of two and 10, the critical window of opportunity for learning languages. As shown in the rat experiments, changes in brain structure can occur in an enriched learning environment. Since ASL stimulates visual, auditory and kinesthetic learning modalities, the process of teaching ASL naturally creates an enriched learning environment with the potential for creating changes in the brain structure of humans. As discussed, a smaller right hemisphere, or more specifically, a smaller prefrontal cortex, basal ganglia and amygdala, are correlated with symptoms of ADHD. Therefore, if ASL can increase cortex size in the RH of the brain and impact these brain areas, then using ASL as an early intervention treatment for ADHD has the potential to reduce the severity of ADHD symptoms. In addition, like a vaccination, if ASL is acquired before the symptoms of ADHD appear, it may inoculate against the development of the disorder altogether. ASL is already being taught in high schools like New Community Jewish High School in West Hills, California. In order to take advantage of the potential that ASL holds in creating enriched learning environments, we recommend that ASL curriculum and classes be introduced at the pre-school and elementary school levels as well.

About the Authors



Deborah A. Cutter, Psy.D., MFT is a licensed Clinical Psychologist and licensed Marriage and Family Therapist. She is Clinical Director of Creative Behavioral Consultants (cbc-autism@sbcglobal.net), a nonpublic agency certified by the California Department of Education, which provides behavior interventionists, using Applied Behavior Analysis techniques, to work with children in their classrooms and at home who have been diagnosed with Autism and other related disorders. She is a freelance author/editor, with current articles being published at www.helpguide.org, a website empowering people to understand, prevent and resolve health and life challenges. In private practice she worked with survivors of domestic violence and drug abuse, couples, families, children, adolescents, and adults with Down's syndrome. Dr. Cutter has experience teaching parenting classes, facilitating women's support groups, administering psychiatric evaluations, interpreting psych testing results, and writing medical-legal reports for Workers' Compensation insurance carriers. She lives with her husband, Mark, and three children, Caitlin, Jacob, and Jenna, and in her spare time enjoys creating abstract paintings and chalk pastel drawings, and is an active member at Shomrei Torah Synagogue in West Hills, California.



Susan M. Zneimer, Ph.D., FACMG holds a doctorate degree in genetics, is a board certified Cytogeneticist by the American Board of Medical Genetics, and is a Fellow of the American College of Medical Genetics. Her positions in higher education include, Adjunct Professor at the University of Southern California Keck School of Medicine and Moorpark College, and Clinical Associate at University of California at San Francisco (UCSF). She has over 15 years experience as a clinical genetic laboratory director, and as CEO of MOSYS Consulting (szneimer@socal.rr.com), provides consulting services for genetic laboratories at City of Hope Medical Center, UCSF, US Labs, Signature Genomic Laboratories, and Genzyme Genetics. Dr. Zneimer has numerous publications in the field of genetics and is a seminar speaker at scientific conferences, and in the community at large. As Program Chair of the American Cytogenetic Conference being held in Monterey, California in April, 2008, she will be speaking on Six Sigma methodology to improve laboratory quality and efficiency. Dr. Zneimer is married to Martin Chetlen, Chair of the Computer Science Department at Moorpark Community College, and is the mother of Mira, age 15 and Alana age 12.

References and Related Links

American Psychiatric Association (1994). Diagnostic and statistical manual of mental disorders: DSM IV. (4th ed.). Washington, DC: American Psychiatric Association.

Bennett, E.L., Diamond, M.C., Krech, D., Rosenzweig, M.R. (1964). Chemical and anatomical plasticity of brain. *Science*, 146, 610-619.

Black, J.E., Isaacs, K.R., Anderson, B.J., Alcantra, A.A., Greenough, W.T. (1990). [Learning causes synaptogenesis, whereas motor activity causes angiogenesis, in cerebellar cortex of adult rats.](#) *Proceedings of the National Academy of Sciences of the United States of America*, 87, 5568-5572.

Castellanos, F.X., Giedd, J.N., Berquin, P.C., Walter, J.M., Sharp, W., Thanhlan, T., Vaituzis, A.C., Blumenthal, J.D., Nelson, J., Bastain, T., Zijdenbos, A., Evans, A.C., Rapoport, J.L. (2001). [Quantitative brain magnetic resonance imaging in girls with attention-deficit/hyperactivity disorder.](#) *Archives of General Psychiatry*, 58, 289-295.

Castellanos, F.X., Giedd, J.N., Marsh, W.L., Hamburger, S.D., Vaituzis, A.C., Dickstein, D.P., Sarfatti, S.E., Vauss, Y.C., Snell, J.W., Lange, N., Kaysen, D., Krain, A.L., Ritchie, G.F., Rajapakse, J.C., Rapoport, J.L. (1996). Quantitative brain magnetic resonance imaging in attention-deficit/hyperactivity disorder. *Archives of General Psychiatry*, 53, 607-616.

Cheng, J.Y., Chen, R.Y., Ko, J.S., Ng, E.M. (2007). Efficacy and safety of atomoxetine for attention-deficit/hyperactivity disorder in children and adolescents-meta-analysis and meta-regression analysis. *Psychopharmacology (Berl.)*, June 16, in print.

Chenn, A., Walsh, C. (2003). [Increased neuronal production, enlarged forebrains and cytoarchitectural distortions in B-Catenin overexpressing transgenic mice.](#) *Cerebral Cortex*, 13, 599-606.

Daniels, M. (1996). Seeing language: The effect overtime of sign language on vocabulary development in early childhood education. *Child Study Journal*. 26(3), 193-208.

Emmorey, K., Damasio, H., McCullough, S., Grabowski, T., Ponto, L., Hichwa, R., Belugi, U. (2002). [Neural systems underlying spatial language in American Sign Language.](#) *NeuroImage*, 17, 812-824.

Emmorey, K., Grabowski, T., McCullough, S., Ponto, L.L.B., Hichwa, R.D., Damasio, H. (2004). [The neural correlates of spatial language in English and American Sign Language: a PET study with hearing bilinguals](#). *NeuroImage*, 24, 832-840.

Faraone, S.V., Wigal, S.B., Hodgkins, P. (2007). Forecasting three-month outcomes in a laboratory school comparison of mixed amphetamine salts extended release (Adderall XR) and atomoxetine (Strattera) in school-aged children with ADHD. *Attention Disorders*, 11, 74-82.

Faraone, S.V. (2000). Genetics of childhood disorders: XX. ADHD, Part 4: is ADHD genetically heterogeneous? *Journal American Academy Child Adolescent Psychiatry*, 39, 1455-1457.

Gardner, H. (1983). *Theory of multiple intelligences*. Basic Books Publ., New York, NY.

Goldstein, S., Ingersoll, B. (2007) [Controversial treatments for ADHD](#). Article can be found online at www.naceonline.com.

Herculano-Houzel, S., Mota, B., Lent, R. (2006) [Cellular scaling rules for rodent brains](#). *Proceedings of the National Academy of Sciences of the United States of America*, 103, 12138-12143.

Hunt, R.D. (2006). Functional roles of norepinephrine and dopamine in ADHD. *Medscape Psychiatry and Mental Health*, 11, posted 3/9/2006.

Kassubek, J., Hickok, G., and Erhard, P. (2004). Involvement of classical anterior and posterior language areas in sign language production, as investigated by 4 T functional magnetic resonance imaging. *Neuroscience Letters*, 364, 168 -172.

King, S., Friffin, S., Hodges, Z., Weatherly, H., Asseburg, C., Richardson, G., Golder, S., Taylor, E., Drummand, M., Riemsma, R. (2006) A systematic review and economic model of the effectiveness and cost-effectiveness of methylphenidate, dexamphetamine and atomoxetine for the treatment of attention deficit hyperactivity disorder in children and adolescents. *Health Technology Assessment*, 10 (23), 1-199.

Kleim, J.A., Lussnig, E., Schwartz, E.R., Comery, T.A., Greenough, W.T. (1996). Synaptogenesis and Fos expression in the motor cortex of the adult rat after motor skill learning. *Journal of Neuroscience*, 16, 4529-4535.

Mannuzza, S., Klein, R.G., Bessler, A., Malloy P., LaPadula M (1998). Adult psychiatric status of hyperactive boys grown up. *American Journal of Psychiatry*, 155, 493-8.

Markham, J., and Greenough, W. (2004). Experience-driven brain plasticity: beyond the synapse. *Neuron Glia Biology*, 1 (4), 351-363.

Miller, P. (2001). Learning styles: The multimedia of the mind. Research report. Retrieved on 6/21/07 from the World Wide Web <http://www.eric.ed.gov> ERIC#: ED451140

Newman, A.J., Bavelier, D., and Corina, D., Jezzard, P., Neville, H.J. (2001). [A critical period for right hemisphere recruitment in American Sign Language processing](#). *Nature Neuroscience*, 5, 76-80.

Parasnis, I., Samar, V.J., Berent, G.P., (2001). [Evaluating attention-deficit/hyperactivity disorder in the deaf population: challenges to validity](#) NITD Research Bulletin, 6, 1-5.

Plessen, K.J., Bansai, R., Zhu, H., Whiteman, R., Amat, J., Quackenbush, G.A., Martin, L., Durkin, K., Blair, C., Royal, J., Hugdahl, K., Peterson, B.S. (2006). [Hippocampus and amygdala morphology in attention-deficit/hyperactivity disorder](#). Archives of General Psychiatry, 63, 795-807.

Purvis, K.L., and Tannock, R. (1997). Language abilities in children with attention deficit/hyperactivity disorder, reading disabilities and normal controls. Journal of Abnormal Child Psychology, 25 (2),133-44.

Shaffer, D., Fisher, P., Dulcan, M.K., Davies, M., Piacentini, J., Schwab-Stone, M.E., Lahey, B.B., Bourdon, K., Jensen, P.S., Bird, H.R., Canino, G., Regier, D.A. (1996). The NIMH diagnostic interview schedule for children version 2.3 (DISC-2.3): description, acceptability, prevalence rates, and performance in the MECA study. Methods for the Epidemiology of Child and Adolescent Mental Disorders Study. Journal of the American Academy of Child and Adolescent Psychiatry, 35, 865-877.

Shankle, W., Romney, K., Landing, B., Hara, J. (1998). Developmental patterns in the cytoarchitecture of the human cerebral cortex from birth to 6 years examined by correspondence analysis. Proceedings of the National Academy of Sciences of the United States of America, 95, 4023-4028.

Shaw, P., Lerch, J., Greenstein, D., Sharp, P., Clasen, L., Evans, A., Giedd, J., Castellanos, F.X., Rapoport, J. (2006).[Longitudinal mapping of cortical thickness and clinical outcome in children and adolescents with attention-deficit/hyperactivity disorder](#). Archives of General Psychiatry, 63, 540-549.

Simpson, D., Plosker, G., (2004). Atomoxetine: a review of its use in adults with ADHD. Drugs, 64 (2), 205-222.

Spencer, T., Biederman, J., Wilens, T. (1999). Attention-deficit/hyperactivity disorder and comorbidity. Pediatric Clinics of North America, 46, 915-927, vii.

Swanson, J.M., Flodman, P., Kennedy, J., Spence, M.A., Moyzis, R., Schuck, S., Murias, M., Moriarity, J., Barr, C., Smith, M., Possner, M. (2000). Dopamine genes and ADHD. Neuroscience Biobehav Rev (2000), 24(1), 21-25.

The MTA Cooperative Group (1999). A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder. Multimodal treatment study of children with ADHD. Archives of General Psychiatry, 56, 1073-1086.

Thompson, J.C., Abbott, D.F., Wheaton, K.J., Syngeniotis, A., Puce, A. (2004). [Digit representation is more than just hand waving](#). Cognitive Brain Research, 21, 412-417.

Wang, Y., Zheng, Y., Du, Y., Song, D.H., Shin, Y.J., Cho, S.C., Kim, B.N., Ahn, D.H., Marquez-Caraveo, M.E., Gao, H., Williams, D.W., Livine, L.R. (2007). Atomoxetine versus methylphenidate in pediatric outpatients with attention deficit hyperactivity disorder: a randomized, double-blinded comparison trial. Aust N Z J Psychiatry, 41, 222-230.

Willis, J. (2006). Research-based strategies to ignite student learning: insights from a neurologist and classroom teacher. Association for supervision and curriculum development, Publ., Virginia.