

Company News

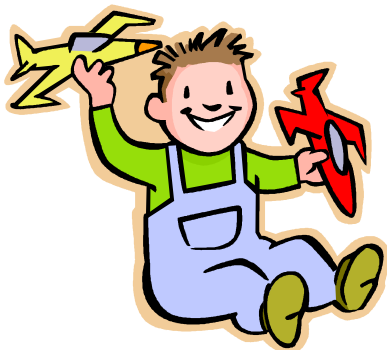
-Please check your checks to make sure enough is taken out for taxes. If not, please contact Farrah at farrah@sonnenbergconsultants.com

-If a person you recommend to the company is hired and stays for a min. of 6 months, you will receive a \$50 bonus!

-Monday June 14th, will be the start of the summer schedule! If you haven't already, let your senior know your availability asap!

-If you are interested in being a floater contact Char asap at cgeissman@hotmail.com

A floater will be able to work at many different houses, so a great position to have as it will give everyone that participates so much more experience!



Always be creative when you are playing! If you are bored, chances are the child is, too!



A COMFORTABLE ENVIRONMENT

Reel Movies for Real Needs creates a welcoming and comfortable environment - lower sound, lights up - where families with children who need accommodations will be able to share the experience of seeing family friendly films at a theatre.

AN OPPORTUNITY TO SHARE THE MOVIE EXPERIENCE

Reel Movies for Real Needs is ideal for families who may not feel comfortable attending regularly scheduled shows.

A select first run movie is featured one Saturday each month at a convenient 10:30AM showtime. South Shore Cinema in Oak Creek, WI is participating! Information on Marcus.com

May 22nd will be Shrek- The Final Chapter

June 16th will be Toy Story 3





Imaging Study Discovers Brain Development Differences in Kids With Fragile X Syndrome

ScienceDaily (May 4, 2010) — **Fragile X syndrome is the most common known cause of inherited intellectual disability and autism.**

Now, researchers using advanced, noninvasive imaging techniques have shown how the brains of very young boys with fragile X syndrome differ from those of young boys without it, providing critical information for the development of treatments for the condition.

Triggered by a mutation in a gene located on the X chromosome, fragile X syndrome affects about one in every 4,000 people, with more significant symptoms occurring in males than females. This condition's genetics and neurobiology are relatively well understood, accelerating the pace with which potential drug therapies have been moving through the pharmaceutical pipeline, said the study's senior author, Allan Reiss, MD, the Howard C. Robbins Professor of Psychiatry and Behavioral Sciences and professor of radiology.

Fragile X syndrome alone accounts for about 2-3 percent of all cases of autism, making it the most common known, specific genetic risk factor for that disorder, although not all people with fragile X syndrome develop autism. Autism is increasingly viewed as not a single disease but a spectrum of them. A large number of diverse genes have been identified as contributing to autism, but with each responsible for only a sliver of cases. Fragile X syndrome patients often manifest discomfort with eye contact, hypersensitivity to sound or touch, abnormalities of language and movement, and varying levels of developmental delay.

In the study, the Stanford and UNC investigators used high-resolution MRI to obtain detailed images of 1- to 3-year-old boys' brains, and followed up two years later with a second imaging session. The MRI results were analyzed at Stanford, primarily by Reiss and the study's lead authors: Fumiko Hoeft, MD, PhD, an

imaging expert and instructor at the CIBSR, and medical student John Carter. Brain images from 41 fragile X syndrome boys were compared with those from age- and developmentally-matched control subjects: 21 boys who were developing typically, and seven others who were experiencing non-fragile-X-related developmental delay.

While many aspects of brain anatomy were similar from one group to the next, the fragile X brains evidenced at an early age (that is, during their first imaging session at 1-3 years of age) an overabundance of gray matter in such regions as the caudate and thalamus, and a diminished presence in a part of the cerebellum called the vermis. This suggests that the fragile X syndrome mutation had already begun to cause identifiable, consistent alterations in brain development, perhaps even before birth. However, the basal forebrain as well as a different part of the thalamus and many regions of the cerebral cortex of fragile X patients, while indistinguishable from those of control subjects during the first imaging session, diverged from their counterparts two years later. These results suggest that certain downstream effects of the mutation become evident only later in brain development. Knowing the locations of fragile X syndrome brain-structure abnormalities and the developmental time course over which they occur -- and being able to noninvasively detect those changes in young patients -- will make it possible to monitor new therapies' effectiveness in (it is hoped) restoring patients' brain structure and function to normality.

The imaging study was funded by a grant from the National Institute for Mental Health. CIBSR research associate Amy Lightbody, PhD, is the other Stanford co-author.



Enjoy getting ready for summer! If you are going on any vacations, let your senior know in advance as soon as possible!