On October 19, 2006, the Brain Research Foundation held their annual meeting at The Chicago Club. The business meeting was followed by lunch and a presentation by Dr. Vernon Leo Towle.

Dr. Vernon L. Towle, Professor in the Department of Neurology and Director of the Evoked Potentials Laboratory and Operating Room Monitoring Service, gave an interesting talk on improved brain mapping for epilepsy surgery. Dr. Towle discussed how he is developing a new method for mapping cortical language areas in patients who are being invasively worked-up for epilepsy surgery.

Towle explained that a risk of epilepsy surgery is the prevalence of postoperative deficits in expressive or receptive language, or memory retrieval. Currently, these deficits are minimized using direct electrical stimulation of cortex during language tasks. However, although it works well for expressive speech, this classic technique does not reliably assess memory or receptive speech areas.

Dr. Towle is developing a passive language mapping technique; similar to the approach used in functional magnetic resonance image (fMRI) studies, but instead looking for changes in subdural electrocorticogram (ECoG) recordings. This technique, based on changes in local field potentials, is more closely related to neural processing than blood dynamics. It may also allow more accurate mapping of areas that are involved in receptive speech.
The Brain Research Foundation began its fight against brain disorders more than five decades ago. From our name, it is evident that we fund brain research. However, sometimes we may forget another important part of the foundation's mission: “to focus public attention on the possibilities and problems of the human brain.”

During the past few months, we have been increasing awareness about brain disorders and the foundation. As well as our standard vehicles of communication, like newsletters, website and year end letters, we have been utilizing new avenues to expand our public outreach.

Over the past year, we have been extremely busy working with John Drury and his family to increase understanding and funding for ALS. We were thrilled to team up with the Chicago White Sox to hold John Drury Night for ALS at U.S. Cellular Field. On September 18, we informed over 30,000 baseball fans about ALS and the Brain Research Foundation.

ABC7 was there to support the Drury family and BRF by televising a portion of John Drury Night on the weekday news. They also produced a public service announcement on ALS and John’s condition that was played on the scoreboard during the game and broadcast on ABC7. I am sure many people heard the words _amyotrophic lateral sclerosis_ for the first time that evening, and hopefully some of them will be moved to find out more.

The foundation also continues to sponsor a variety of conferences and presentations. We organized an extremely interesting and comprehensive seminar on Parkinson’s disease. The talks began with a historical background of Parkinson’s, led to a review of latest research and finished with a tour of a laboratory. It was a terrific opportunity to see the progression of science.

In addition to advancing science through research funding, the foundation is always excited when it can educate people about the brain and illustrate how important brain research is to us all. We hope to see some new faces at future presentations.

Sincerely,

Terre A. Sharma, Ph.D.
Executive Director
John Drury, a distinguished newscaster and Chicago legend, started his career in 1962. His straightforward approach and sincerity caused viewers to tune in every evening for 40 years until his retirement in 2002. John Drury’s significant contributions to broadcast journalism have been recognized by his peers. He has been named to the Chicago Journalism Hall of Fame, honored by the Museum of Broadcast Communications and awarded the prestigious Silver Circle Award.

Shortly after his retirement, John Drury was diagnosed with a neuromuscular disease, amyotrophic lateral sclerosis (ALS). ALS, also known as Lou Gehrig’s disease, is a progressive neurodegenerative disease that attacks motor neurons, resulting in decreased muscle movement control and eventual paralysis.

Since his diagnosis, John Drury and his family have been determined to raise awareness and funds for this devastating illness. John’s wife and family attended John Drury Night for ALS at U.S. Cellular Field. John’s son, Jim Drury, threw out the ceremonial first pitch, while a video about ALS and his father’s battle with the disease played on the scoreboard. During the evening, Brain Research Foundation volunteers increased awareness by distributing information about ALS and how White Sox fans can help put an end to the disease.
2006 Seed Grant Recipients

Glyn Dawson, Ph.D.
Department of Pediatrics
Phospholipids, Endosomes and Brain Function in Battens Disease

Brian K. Gehlbach, M.D.
Department of Medicine-Pulmonary and Critical Care
Improving the Sleep and Circadian Rhythms of Mechanically Ventilated Patients

Fernando D. Goldenberg, M.D.
Department of Neurology
Tackling Cerebral Salt Wasting: Searching for the Cause and Cure

Royce J. Lee, M.D.
Department of Psychiatry
The Effects of Intranasal Vasopressin and Oxytocin on Arousal and Aggression

Maciej Lesniak, M.D.
Department of Surgery
Role of Regulatory T Cells in Malignant Gliomas

Chunyu Liu, Ph.D.
Department of Psychiatry
Identification of Cis-regulatory Elements for Gene Expressed in Human Brain

Christopher J. Lowe, Ph.D.
Department of Organismal Biology and Anatomy
Development and Evolution of Brain Signaling Centers: Hemichordates as a Simple System for Characterizing Neurodevelopment

Dario Maestripieri, Ph.D.
Department of Comparative Human Development
Effects of Early Experience on the Development of Brain Monoamine Systems and Behavior

Martha K. McClintock, Ph.D.
Department of Psychology
Neural Mechanisms of Androstadienone

Deborah J. Nelson, Ph.D.
Department of Neurobiology, Pharmacology and Physiology
ClC3 Chloride Channels as Regulators of Synaptic Efficacy

Abraham A. Palmer, Ph.D.
Department of Human Genetics
Genetic Analysis of Motor Circuits

Ye-Ping Tang, M.D., Ph.D.
Department of Psychiatry
The Neuronal Mechanisms for the Involvement of BDNF in Depression

Gopal Thinakaran, Ph.D.
Department of Neurobiology, Pharmacology and Physiology
Regulation of Alzheimer’s Disease Aβ Production by Lipid Modification of γ-Secretase

Paul R. Vezina, Ph.D.
Department of Psychiatry
Behavioral and Dopaminergic Sensitization by Nicotine

Yimin Zou, Ph.D.
Department of Neurobiology, Pharmacology & Physiology
Molecular and Cellular Mechanisms of Cortical Development

Women’s Council Seed Grant

Qian Chen, Ph.D.
Department of Psychiatry
Modeling of Parkinson’s Disease in the Mouse

Symptoms:
- Progressive weakness or difficulty in coordination
- Changes in speech
- Weight loss or loss of muscle mass

Types:
- Sporadic: the most common form of ALS (90 to 95% of all cases). It occurs randomly. It may affect anyone.
- Familial: a family history or genetic inheritance of ALS (5 to 10% of all cases).
- Guamanian: an extremely high incidence of ALS was observed in Guam in the 1950s. A neurotoxin from a tropical plant found in Guam was suspected to be the cause.

Facts:
- 3,000,000 Americans afflicted
- Mean age of onset in the fifties
- 5 to 10% of cases are hereditary
- Average survival is 3 to 5 years

About ALS
Amyotrophic lateral sclerosis (ALS), also called Lou Gehrig’s disease, is a progressive neurodegenerative disease that weakens and eventually destroys the motor neurons. Motor neurons are components of the nervous system that connect the brain with the skeletal muscles. As these nerves die, muscle movement control decreases. A person with ALS usually presents problems with dexterity or speech, resulting from muscle weakness. At a later stage of the disease, patients become paralyzed while their minds remain unaffected.

ALS is one of the most common neuromuscular diseases worldwide, and people of all races and ethnic backgrounds are affected. Approximately 30,000 people in the United States currently have ALS. ALS most commonly strikes people between the ages of 40 to 60 years of age. Studies suggest that men are somewhat more likely to develop ALS than are women (1.2 men to every woman).

So Much So Fast
Chicago Premiere at the
Gene Siskel Film Center
at the Art Institute

Saturday, November 18
at 7:30 pm (panel after)
Sunday, November 19
at 3:00 pm
Monday, November 20
at 7:45 pm

From the Academy Award nominated directors of Troublesome Creek, Steven Ascher and Jeannie Jordan, comes a new documentary film, So Much So Fast. A black-humored cliffhanger of romance, guerrilla science and the redefinition of time. So Much So Fast unfolds like a nonfiction novel. Stephen Heywood finds out he has ALS. His brother Jamie becomes obsessed with finding a cure. And the woman who’s falling in love with Stephen has a decision to make.

Info and trailer at www.somuchsofast.com

“Elegantly presents both a critique and a celebration of American optimism.” Grade A-
Entertainment Weekly
SMA is the leading genetic cause of death in infants and toddlers, yet it is still a relatively unknown and under-funded disease. It is a group of inherited and often fatal diseases that destroys the nerves controlling voluntary muscle movement, which affects crawling, walking, head and neck control, and even swallowing. One in 35-40 Americans are carriers for SMA, or approximately 7 million people. Over 25,000 American children are estimated to suffer from SMA, comparable to in prevalence to a better known disease—amyotrophic lateral sclerosis (ALS or Lou Gehrig’s Disease). SMA affects the general population equally, there are no race or gender preferences.

SMA is caused by mutations or deletions in the human survival motor neuron 1 (SMN1) gene that produces a protein in the body called survival motor neuron (SMN) protein. This protein deficiency has its most severe affect on motor neurons. Motor neurons are nerve cells in the spinal cord which send out nerve fibers to muscles throughout the body. Since SMN protein is critical to the survival and health of motor neurons, without this protein nerve cells may atrophy, shrink and eventually die, resulting in muscle weakness and atrophy. Destruction of motor neurons affects voluntary muscle movement. And as muscles weaken, even the respiratory system and breathing become compromised.

The main objective of Dr. Sharma’s project is test the hypothesis that the SMN protein function is required for the rapid transport of nutrients and other trophic factors in motor nerves. Motor nerves consist of long-processes called axons that originate from motor neurons located in the spinal cord. These axons are responsible for carrying neural signals from the spinal cord to the muscle. The length of motor axons depends on the location of the muscle target. For example, axons of those motor neurons that control the function of feet and hands are longer than those that control shoulder or thigh muscles. The maintenance of the fine structure of the motor axon and its ability to control muscles depends on fast-transport of proteins and nutrients from the motor neuron to the tip of the axon located in the muscle. This transport is accomplished by proteins that move material through the axon along slender filaments called microtubules.

Previously, Dr. Sharma discovered that in the neural stem cells SMN function is needed for the fast-transport machinery to work efficiently. His lab found that reduced concentration of SMN in the neural stem cells prevents the forward-directed fast-transport machinery. They also discovered that this deficiency can be corrected by simultaneously introducing a protein involved in transport called p50 dynamitin into the neural stem cells. With a known deficiency caused by reduced SMN protein and a potential remedy mediated by the p50 dynamitin in the neural stem cells, the obvious next step is to test this paradigm in motor neurons.

Dr. Sharma has started to evaluate fast-transport in the axons of spinal motor neurons in chickens. His research will help elucidate the function of SMN protein, and uncover promising strategies for possible therapeutic reagents for the treatment of SMA.

On September 21, 2006, Brain Research Foundation representatives were guests at Peregrine Charities fundraiser—Oktoberfest. At the event, Peregrine Charities presented the Brain Research Foundation with a $50,000 check to support spinal muscular atrophy research.
Women's Council Seed Grants

For over forty years, the Women's Council of the Brain Research Foundation has supported the advancement of brain research. Members have tirelessly raised awareness, perception, and research dollars for the neurosciences. Most recently, they have been awarding an annual Women's Council Seed Grant in the amount of $25,000 to deserving female researchers.

These seed grants are extremely important to scientists. The grants provide start-up monies for projects that have the potential of becoming competitive for government funding or other outside sources. Outside granting agencies require demonstration of feasibility, and this required preliminary data can only be obtained by taking an idea and testing it. To prove their hypothesis, a limited amount of money is needed—a "seed."

A few years ago, it was decided that contributions and revenue from events would be targeted toward the Women's Council Seed Grant Fund with the idea of increasing the fund to sustain fully an annual Women's Council Seed Grant. By endowing this fund, they can ensure a legacy of discovery for the Women's Council.

The Women's Council has formed a Seed Grant Committee, chaired by Mary Beattie, to fundraise for this vital program. The goal is to increase the fund by $160,000, bringing the total to $500,000. Creating an endowment will be an investment in scientific discovery. All medical breakthroughs start with untested ideas.

If you would like more information or would like to donate to the Women's Council Seed Grant Fund, please call the Brain Research Foundation at 773-834-6750.