Brain Research Foundation
2008-2009 Annual Report

The research we fund will help unravel the neurological mysteries that impact all ages and touch all families.

Whether it's a child coping with autism, a parent struggling with dementia or a friend battling depression—every one of us has experienced the effects of brain-related disorders.

The Brain Research Foundation supports research that will lead to life-changing discoveries in neuroscience and have a profound influence on the future of us all.

"It's a personal passion—as well as a legacy to my late husband—to help people understand the devastating impact of neurological disorders on those affected and their families. The Brain Research Foundation can play a pivotal role in building this understanding."

"The research supported by the Brain Research Foundation will ultimately help people of all ages live the fullest, most dignified lives they can. I want to increase awareness of the organization and help raise funds that will enable it to continue to strengthen its support of this critical work."

—Suzanne Kopp-Moskow
Board of Trustees
Brain Research Foundation

Since its inception, the Brain Research Foundation has contributed over $34 million to advance neuroscience.

"I've always had a strong interest in the problems children suffer as a result of brain disease and believe that they warrant more attention. I want to make these problems more well known and support the search for solutions."

"Brain Research Foundation will continue to focus on the problems of the brain, diagnose behavioral predictors that indicate disease and work to find answers."

"My role is to help focus attention to the challenges we face, foster support and financing and, with success, eventually put ourselves out of business."

—Norman R. Bobins
Vice President
Brain Research Foundation

To Our Brain Research Foundation Friends

The work of the Brain Research Foundation is increasingly important. As the population grows and our life expectancy increases, the incidence of debilitating neurological disorders rises. The research that we fund today fuels the innovations that will become the much needed treatments and cures of tomorrow. While our government and corporate research organizations continue to search for answers, there is still much to be done and funding gaps that need to be filled. Our unique mission crosses traditional funding boundaries and bridges these gaps by considering all diseases, disorders and age groups.

One very important way we help accelerate new discoveries is through our Seed Grant Program. In 2009, we have successfully expanded our Seed Grant Program which funds neuroscience research in the greater Chicago area. We are pleased to have been able to increase the individual seed grants by $15,000 each, enabling researchers to focus more energy on new projects which will hopefully allow them to obtain results faster and achieve additional grants even sooner.

In conjunction with the remodeled Seed Grant Program, we established the Brain Research Foundation Scientific Review Committee to review our annual grant applications. This committee is made up of eight researchers from several institutions throughout greater Chicago and nationwide. Their scientific expertise was invaluable when reviewing the 2009 Brain Research Foundation Seed Grant proposals.

This past year we were presented with an opportunity that would strengthen our position and increase our resources. We merged with another Chicago-based neuroscience foundation, the Children's Brain Research Foundation. The CBRF brings significant experience and assets to the BRF. We are excited by the ideas that are being brought forward; utilizing many will help the BRF improve and grow. The combined organization is even more enthusiastic about accelerating new discoveries in brain research.

As you read this annual report, you will see the difference the Foundation is making through the important research we have funded over the past two years. We hope these stories will provide you with an understanding of the amazing answers these scientists are trying to uncover. And with this understanding, we hope you commit to make a difference and get involved in our work.

We need your help. We want more people to know about the work of the Brain Research Foundation. The more you know about us, the more you can help spread the word. And the result will be generous donors of time and resources to support our mission and our work.

All of us have family and friends whose lives are touched by neurological disease every day. Through your support of the Brain Research Foundation, you are able to change those lives. We hope you will help us continue our search for answers.

Sincerely,

Terre A. Sharma, Ph.D.
Executive Director

Nathan Hansen
President

Supporting Novel Research with Seed Grants

The Brain Research Foundation Fay/Frank Seed Grant Program was established consistent with our fundamental purpose—Innovate, Explore, Discover. Supporting groundbreaking research in neuroscience has been the role of the Seed Grant Program for almost three decades and represents some of the most important work the Foundation does: funding pilot stage innovative ideas and promising investigations that drive advances in our knowledge of how the human brain functions.

Philanthropy is often the only source of support for scientists working on a pilot project. In large part this is because the federal government does not fund cutting-edge investigative leads without some assurance that the ideas are feasible and worthwhile. It is a problematic situation. Preliminary data is needed to prove feasibility and apply for grants, but without funding, the researchers cannot create the data.

Our Seed Grant Program enables scientists to create the data necessary to obtain larger grants and publish new results. These larger grants and papers, in turn, generate even more results and answer more questions about the brain. Our goal is to fund research that leads to new treatments and eventual cures for brain-related disorders.

In 2008-2009, the Foundation allocated $1 million to 31 neuroscientists. The following pages feature seven recipients. Much has been accomplished in a short amount of time. Two have received additional funding for the project that we supported; two have already published articles in important scientific journals; all are making progress on innovative projects that will help children and adults with neurological diseases.

2008-2009 Seed Grant Recipients

Rajeshwar Awatramani, Ph.D.
Department of Neurology, Northwestern University
The Developmental Basis of Dopaminergic Neuron Diversity

Thomas C. Bozza, Ph.D.
Department of Neurobiology and Physiology, Northwestern University
Optogenetic Analysis of Mammalian Olfactory Circuits

Jianhua Cang, Ph.D.
Department of Neurobiology and Physiology, Northwestern University
Organization and Development of Motor Maps in the Mouse Superior Colliculus

Dane M. Chetkovich, M.D., Ph.D.
Department of Neurology, Northwestern University
Gene Therapy for Treatment of Epilepsy

Anis Contractor, Ph.D.
Department of Physiology, Northwestern University
In Vivo Analysis of the Role of FMRP in Dendrite Maturation and Plasticity in the Somatosensory Cortex

David J. Freedman, Ph.D.
Department of Neurobiology, The University of Chicago
Neuronal Mechanisms of Visual Category Learning and Recognition

David Gallo, Ph.D.
Department of Psychology, The University of Chicago
An fMRI Study of Complex Scene Memory in Younger and Older Adults

Jaime García-Añoveros, Ph.D.
Department of Anesthesiology, Northwestern University
Insm1 in Terminal Neurogenic Proliferation

William Green, Ph.D.
Department of Neurobiology, The University of Chicago
Role of RING Domain of Rapsyn in the Formation of the Neuromuscular Junction and Disease

Christian R. W. Hansel, Ph.D.
Department of Neurobiology, The University of Chicago
The Role of AMPA Receptor (GluR2) Trafficking in Cerebellar Plasticity in Adult Mice: An Imaging Study

Dean M. Hartley, Ph.D.
Department of Neurological Sciences, Rush University Medical Center
The Role of Seizure-like Activity Driving the Progression of Alzheimer's Disease

Nicholas Hatsopoulos, Ph.D.
Department of Organismal Biology & Anatomy, The University of Chicago
Encoding of Cortical Information in the Coordination of Reach to Grasp and Feeding

Naoum Issa, M.D., Ph.D.
Department of Neurobiology, The University of Chicago
Mechanisms for Coding Complex Images in the Early Visual System

Adil Javed, M.D., Ph.D.
Department of Neurology, The University of Chicago
Immunological and Molecular Mechanisms Involved in the Pathogenesis of Devic's Disease

Benjamin B. Lahey, Ph.D.
Department of Health Studies, The University of Chicago
Functional Neurobiology of Harsh Maternal Parenting

Chunyu Liu, Ph.D.
Department of Psychiatry, The University of Chicago
Genetic Mapping of DNA Methylation Regulators in Human Cerebellum

Jason MacLean, Ph.D.
Department of Neurobiology, The University of Chicago
Imaging Locomotor Network Activity–Toward a Wiring Diagram of the Locomotor Central Pattern Generator

James A. Mastrianni, M.D., Ph.D.
Department of Neurology, The University of Chicago
The Role of the AGAAAAGA Palindrome in Prion Disease

Jill A. Morris, Ph.D.
Department of Pediatrics, Northwestern University/ Children's Memorial Research Center
Deciphering the Roles of DISC1 Isoforms in Embryonic Brain Development
Women's Council Seed Grant

Puneet Opal, M.D., Ph.D.
Department of Neurology, Northwestern University
Epigenetics in Spinocerebellar Ataxia

P. Hande Ozdinler, Ph.D.
Department of Neurology, Northwestern University
Investigation of Molecular and Genetic Controls over Cell-Type Specific Vulnerability of CSMN in ALS

Brian J. Prendergast, Ph.D.
Department of Psychology, The University of Chicago
Cytokine and Adrenocortical Mediation of Cancer-Induced Depression

Raymond P. Roos, M.D.
Department of Neurology, The University of Chicago
A Study of Provocation and Treatment of HNPP

Jeremy Amiel Rosenkranz, Ph.D. *Department of Cellular and Molecular Pharmacology, Rosalind Franklin University of Medicine and Science*
Chronic Stress Causes Pathology of K^+ Channels in Amygdala Neurons

Steven Roth, M.D.
Department of Anesthesia and Critical Care, The University of Chicago
Rodent Model of Perioperative Ischemic Optic Neuropathy

Ilya Ruvinsky, Ph.D.
Department of Ecology and Evolution, The University of Chicago
Sex-Specific Regulation of GABAergic Neurons

Nancy B. Schwartz, Ph.D.
Department of Pediatrics, The University of Chicago
Gliogenesis in the Hindbrain: A Slice Culture Approach

Kamal Sharma, Ph.D.
Department of Neurobiology, The University of Chicago
Neural Control of Motor Functions

Gordon M.G. Shepherd, M.D., Ph.D.
Department of Physiology, Northwestern University
Synaptic Circuit Organization of the Rubro-Olivary System

Sangram S. Sisodia, Ph.D.
Department of Neurobiology, The University of Chicago
Biochemical and Crystallographic Characterization of Abeta in Complex With Transthyretin–A Proposed Abeta Scavenger

Marc W. Slutzky, M.D., Ph.D.
Department of Neurology, Northwestern University
A Brain-Machine Interface Based on Movement-Related Potentials from Epidural vs. Subdural Signals

Our Seed Grants Are The First Step

Results from his 2008 BRF Seed Grant study enabled **David J. Freedman, Ph.D., Neurobiology at the University of Chicago,** to submit a proposal to the National Science Foundation.

In 2010, Dr. Freedman was awarded the National Science Foundation CAREER award for junior faculty. This award is a 5 year grant in the amount of $950,000.

Dr. Freedman's research will help us understand the mechanisms that underlie learning, memory and recognition.

Humans and other advanced animals are not born with a built in library of meaningful categories, such as "tables" and "chairs," which we are preprogrammed to recognize. Instead we learn to recognize the meaning of such stimuli through experience. This ability, which is disrupted by a number of brain diseases and conditions such as Alzheimer's disease, schizophrenia and stroke, is critical because it allows us to respond appropriately to the continuous stream of stimuli and events that we encounter in our interactions with the environment.

While much is known about the encoding of basic visual features (such as contrast, orientation, and motion direction) in early stages of the visual system, much less is known about how the brain learns, stores, recognizes and recalls the meaning of our sensory experiences. With his 2008 Brain Research Foundation Seed Grant, Dr. David J. Freedman conducted research to determine a more detailed understanding of the brain mechanisms of visual learning, memory and recognition.

A greater understanding of visual learning and categorization is important for addressing a number of brain disorders and conditions that leave patients impaired in everyday tasks that require an appropriate response to sensory information. These studies also have particular relevance for understanding and addressing learning disabilities, such as attention deficit disorder and dyslexia, which affect a substantial number of school age children and young adults. The long-term goal of Dr. Freedman's research is to help guide the next generation of treatments for these brain-based diseases and disorders by helping to develop a detailed understanding of the brain mechanisms that underlie learning, memory and recognition.

Identifying "silent seizures" in Alzheimer's patients and seeking novel ways to use anti-seizure medications.

Dean M. Hartley, Ph.D., Neurological Sciences at Rush University Medical Center

Dean M. Hartley, Ph.D.,Neurological Sciences at Rush University Medical Center, is testing his hypothesis that "silent" or very mild seizures are involved in the progression of Alzheimer's disease. If this is correct, current anti-epileptic drugs may be useful in treating the disease.

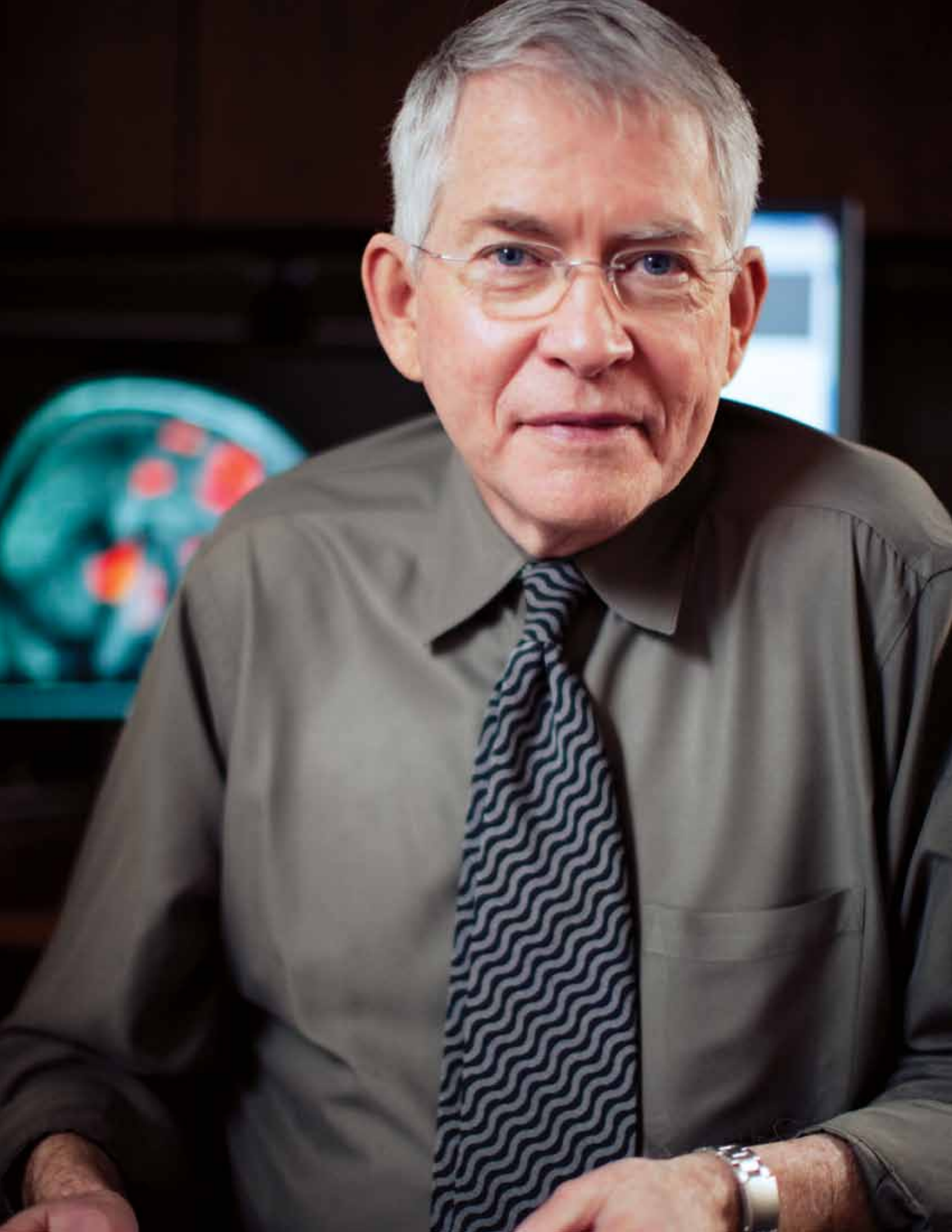
Alzheimer's disease (AD) is an irreversible, progressive brain disorder. AD destroys neurons, causing memory loss, confusion and impaired judgment. It is characterized by the formation of two pathological features, plaques and tangles. Plaques are made from a fragment of the protein called beta-amyloid and buildup between the neurons. Tangles form inside neurons and consist of twisted bundles of fibers of a protein called tau.

Research has shown that over time, these two abnormal pathologies spread through the brain in a very characteristic pattern and Dr. Dean M. Hartley is particularly interested in the progression of the plaques. In the earliest stages, plaques are only present in the upper areas of the brain. However, as the disease progresses the plaques move to other specific areas lower in the brain, as though they are following specific pathways. These cues suggest that the disease is moving by electrical activity through specific neuronal pathways, connecting one area to the next, driving new pathology. Understanding how this progression occurs is key in understanding how to stop this disease.

Dr. Hartley is using his 2009 BRF Seed Grant to examine the possible role of seizure-like activity in the progression of AD. Dr. Hartley's working model is that brain cells become "hyperactive," similar to the activity measured during very mild seizures; this abnormal activity then causes the characteristic AD pathology to develop in this area. Moreover, this hyperactivity travels to other regions by specific connections causing a cascade of hyperexcitability and subsequent AD pathology; this specific hyperactivity drives the progressive pathology in the brain.

Studies have reported seizure-like activity in AD patients and animal models of AD. Recent studies in animal models of AD have shown a type of "silent seizure," suggesting there is an undetected hyperactivity in the AD brain. Dr. Hartley is testing his hypothesis by placing sensitive monitoring devices in a mouse model of AD to determine if the sequence of the developing pathology is preceded by hyperactivity.

To further understand this problem, drugs that block this hyperactivity, including anti-seizure medications, will be administered at different time periods. This will help in understanding if hyperactivity is involved, and also determine if interrupting hyperactivity at a specific time may block "downstream" areas from developing AD pathology. A better understanding of this relationship is warranted and may be extremely valuable in identifying mechanisms responsible for this devastating disease. Most importantly, this understanding may suggest that drugs blocking or reducing hyperactivity in the brain may be able to stop the initiation or progression of the disease; currently we are only able to treat the symptoms. The potential of this research is that antiepileptic drugs, which block neuronal hyperexcitability, may be useful in treating AD. Because these drugs are currently used to treat epilepsy, they could be rapidly transitioned to the treatment of AD.



In 1984, **Benjamin B. Lahey, Ph.D., Health Studies at the University of Chicago,** published a paper in which he hypothesized that mothers who abuse their children may have a lower threshold for child misbehavior and may react more punitively to it.

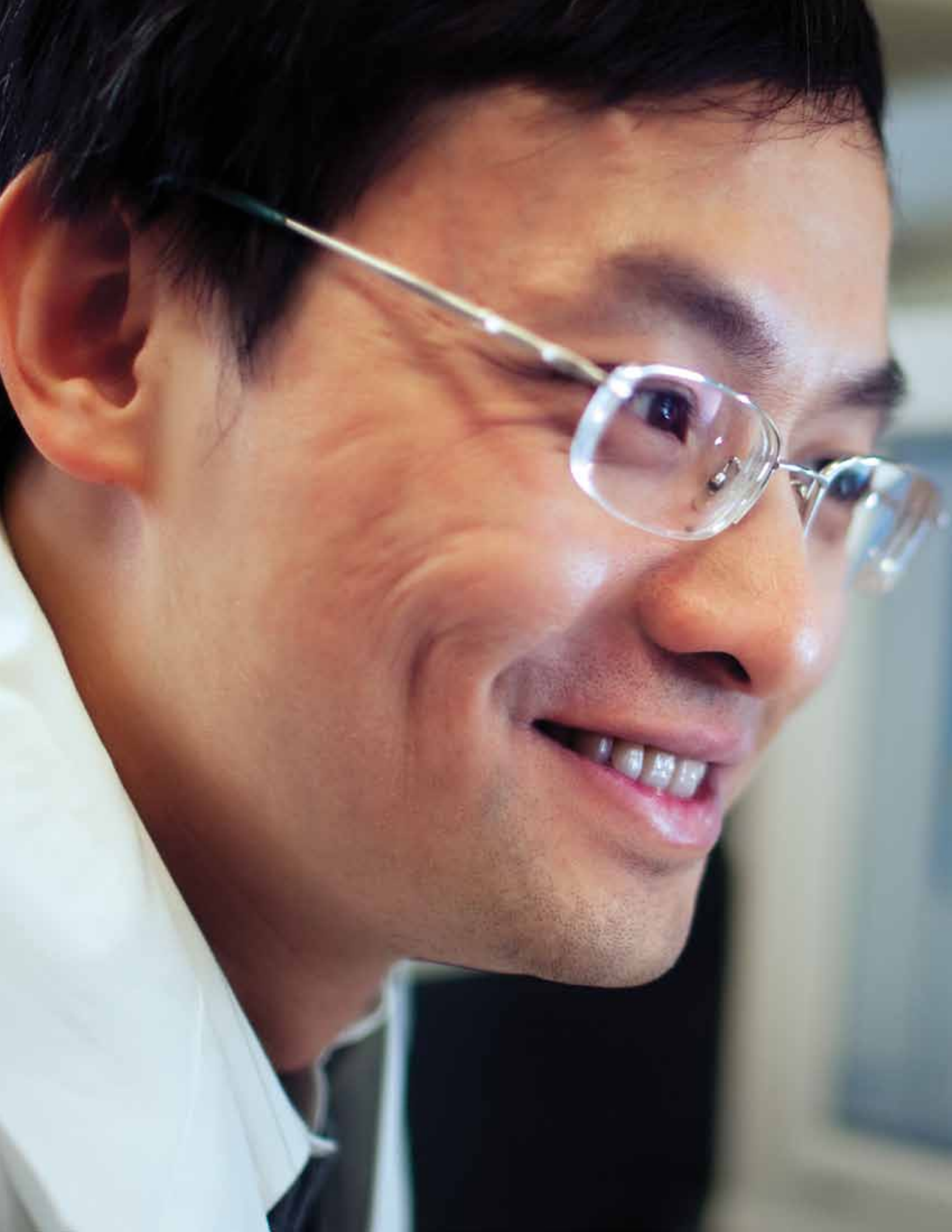
Twenty-five years later, advancements in technology and a 2009 Brain Research Foundation Seed Grant have allowed him to test this important hypothesis.

Harsh, abusive maternal parenting in childhood is a robust risk factor for many mental disorders and health problems including cardiovascular disease, diabetes, and obesity. Studies of non-human mammals reveal that atypical early mothering causes lasting changes in the expression of genes that are involved in reactions to stress. Dr. Benjamin B. Lahey would like to prevent these outcomes by helping mothers parent less harshly. Ideally, intervention begins with mothers when they are pregnant or having problems with their children, at which time they are taught to respond in non-harsh ways. Unfortunately, it is difficult to help mothers to reduce their harsh parenting. Even when abusive mothers are motivated to change, it is difficult for them to do so.

Dr. Lahey believes that there is a neurobiological reason why the maternal behavior is different in these mothers. When they experience their child misbehaving or their child defies them, they have an intense negative emotional reaction that they are unable to control. Dr. Lahey will use functional magnetic resonance imaging (fMRI) to test the hypothesis that harsh mothers will exhibit both greater activation of brain systems involved in negative emotion, such as the amygdala, and less coordinated activation of cortical control systems when viewing images of child misbehavior. This would indicate that these mothers have problems in voluntarily dampening their emotional reaction. Because these situations happen very quickly, the harsh mothers are likely to hurt their children in a disciplinary response rather than sitting down and talking about the misbehavior.

Dr. Lahey also predicts that genes known to influence animal maternal behavior are associated with maternal neural responses to child stimuli. He will study dopaminergic reward circuits which, when activated normally, result in infant stimuli becoming reinforcing and play a key role in mammalian mothering. Lahey will focus on the dopamine transporter gene (DAT1), predicting variations between normal and harsh mothers.

Understanding harsh parenting at neurobiological levels will lead to breakthroughs in treating harsh parenting. Programs that intervene and reduce early harsh parenting could have great public health benefits, just as do programs to reduce smoking related lung cancer.



According to the World Health Organization, as many as 450 million people worldwide suffer from a mental or behavioral disorder.

Chunyu Liu, Ph.D., Psychiatry and Behavioral Neuroscience at the University of Chicago, is trying to make a difference in the lives of so many by determining the link between genetic factors, environmental factors and neuropsychiatric diseases.

Dr. Liu was awarded a BRF Seed Grant in 2008. To date, the data from this grant has generated nine research articles in journals such as *American Journal of Human Genetics*, *Behavior Genetics* and *Molecular Psychiatry.*

We were taught that changes in genes take place over many generations and through years of natural selection. Would you be surprised to learn that our environment and the choices we make in life today can influence our genetic code and have a profound effect on our health and the health of our children in the near future?

The field of epigenetics is illustrating how quickly functions of one's genetic code can be altered through environmental changes. Epigenetics refers to changes in gene expression or phenotype (appearance) that do not change the underlying deoxyribonucleic acid (DNA; the chemical substance of genes) sequence. In recent years, researchers have identified various chemical modifications to DNA and to histones (proteins that associate tightly with DNA) that can determine when or even if a given gene is expressed.

Dr. Chunyu Liu studies one of those chemical modifications—DNA methylation. DNA methylation involves the addition of a methyl group to DNA and is essential for normal development and cellular differentiation in higher organisms. DNA methylation can be altered by environment. More specifically, it can be altered by factors like diet, radiation and drugs. Abnormal DNA methylation has been associated with diseases such as cancer and diabetes as well as multiple sclerosis, schizophrenia and other neuropsychiatric disorders. Studies have suggested that DNA methylation could be a heritable quantitative trait. However, the regulation mechanism of DNA methylation of specific genes, especially in the brain, is still largely a mystery. Like all other quantitative traits, genetic regulation factors that affect traits can be mapped by genetic methods. Dr. Liu uses genetic methods to map regulatory elements of DNA methylation in the human brain.

Liu's lab compared brain samples from people afflicted with schizophrenia, bipolar disorder, major depression, and healthy controls. With the funding from a BRF Seed Grant, Liu's lab became the first group to identify that both regional and distant genomic variations could affect the methylation level of a specific DNA sequence in the brain. The study showed a convincing evidence of genetic factors of epigenetic traits. Environmental factors and genetic factors underlying human phenotypes and diseases found their shared spaces. Now, Liu is working on a study that will investigate DNA methylation and gene expression differences among the same groups—individuals with schizophrenia, bipolar disorder, major depression and healthy controls. The results of these studies will help scientists better understand the mechanisms of DNA methylation and regulation of gene expression as well as the biological basis of disease, particularly neuropsychiatric diseases.

Schizophrenia is an inherited disorder, in that, if a family member is affected by schizophrenia, the other members have an increased risk of developing the disease as well.

Jill A. Morris, Ph.D., Pediatrics at Northwestern University/Children's Memorial Research Center, studies a schizophrenia susceptibility gene, DISC1, which was initially identified in a large Scottish family with members who suffered from schizophrenia, bipolar disorder and recurrent major depression.

Schizophrenia is a debilitating developmental illness characterized by multiple symptoms including hallucinations, delusions and social withdrawal. Although drugs are available to treat some of the symptoms of schizophrenia, their profound side effects make non-compliance a major issue and often result in relapse and hospitalization. Schizophrenia is believed to have a neurodevelopmental origin in that patients demonstrate early cognitive impairments, behavioral dysfunction in childhood and adolescence, abnormalities in central nervous system (CNS) development, and no demonstrative neurodegeneration. One of the contributing factors to abnormal neurodevelopment in schizophrenia is thought to be a genetic insult. Therefore, understanding the genetic and developmental basis of schizophrenia is critical for discovering treatments.

Multiple schizophrenia susceptibility genes have been identified including DISC1 (Disrupted in Schizophrenia 1). DISC1 was initially identified in a large Scottish family with members who suffered from schizophrenia, bipolar disorder and recurrent major depression. DISC1's association with schizophrenia has been confirmed in other population and family studies. Genetic studies have also indicated a DISC1 association with bipolar affective disorder, autism and Asperger syndrome. There are multiple forms of DISC1 expressed in the developing brain. Dr. Jill A. Morris is using her 2009 BRF Seed Grant to examine the function of these multiple DISC1 variants during development.

Dr. Morris' lab will determine the developmental defects in the developing mouse brain due to the loss of different DISC1 isoforms. Studies in the developing mouse have demonstrated prominent expression of mouse DISC1 in the hippocampus as well as expression in the developing cerebral cortex, hypothalamus, cerebellum and olfactory bulbs. These are brain regions that have been implicated in schizophrenia pathogenesis. It may be that disrupting the function of a specific DISC1 variant determines if an individual has increased susceptibility to schizophrenia, bipolar affective disorder or autism. In order to develop new therapeutics, it is critical that we understand the genetic basis of the disease. This very novel research will greatly aid in the understanding of DISC1's role in major psychiatric disorders, and perhaps in identifying a new treatment target.

Chronic stress causes a dysregulation of emotional behaviors, and is a potent triggering factor in depression and other psychiatric illnesses.

Jeremy Amiel Rosenkranz, Ph.D., Cellular and Molecular Pharmacology at Rosalind Franklin University of Medicine and Science

The 2009 Brain Research Foundation grant awarded to **Jeremy Amiel Rosenkranz, Ph.D., Cellular and Molecular Pharmacology at Rosalind Franklin University,** was very important to his work. Not only did it allow him to pursue a novel research idea, it was also his first grant as a principal investigator of his own lab.

He's using his grant to understand how stress impacts depression. Ultimately, this work could lead to the identification of a new pharmacological target for the prevention and treatment of emotional disorders.

Our bodies can handle most common stressors by adapting. However, chronic stress can wear them down and cause us to become ill, both physically and mentally. If one is not able to cope with chronic stress well, stressful life situations can increase the risk of serious health problems, including psychiatric disorders such as depression. Despite the prevalence of stress and the severity of its effects, surprisingly little is known about how this risk factor influences depression.

Dr. Jeremy Amiel Rosenkranz is using his 2009 BRF Seed Grant to understand how stress impacts depression. If one can reduce the impact of stress, one can reduce the incidence of depression and other psychiatric disorders. There are many effective antidepressants, but waiting until someone already has depression is not a preventative strategy.

To determine a novel therapeutic target to reverse stress-induced impairments, Dr. Rosenkranz must first determine how stress modifies emotion. Stress activates "emotion circuits" in the brain; repeated stress sensitizes these circuits. The amygdala, which is located deep within the medial temporal lobes of the brain, is involved in the processing and memory of emotional reactions. Because of its key role in emotion, the amygdala is a possible target of the effects of stress on emotion. Under normal stress, the amygdala is activated and an appropriate behavioral response is elicited. But under chronic stress the amygdala becomes hyperactivated which leads to an abnormal response or emotional disturbance.

While there is growing evidence for the enduring effects of chronic stress on morphological, physiological and biochemical features of neurons in several brain regions, little is known about the effects of chronic stress on amygdala neuronal electrophysiology–the electrical activity of neurons. Because neurons share similar characteristics across mammalian species, Dr. Rosenkranz is using rodents to study the effects of stress on the amygdala. The rodents are exposed to stressors and then electrophysiology recording which measures the activity of neurons is performed. Dr. Rosenkranz will test if chronic stress increases excitability of amygdala neurons via a specific ion channel (K_{Ca} channel) that regulates neuronal activity. These channels are likely candidates because they are modified by the steroid corticosterone, which is a major stress hormone in rodents. If it is shown that K_{Ca} channel activity is altered during chronic stress, then this channel is a new potential pharmacological target to prevent and treat the effects of stress on disorders of emotion such as depression.

Dane M. Chetkovich, M.D., Ph.D., Neurology at Northwestern University, is working on a gene therapy treatment for epilepsy that may one day help patients who are otherwise resistant to medical and surgical therapy.

With his 2009 BRF Seed Grant, Dr. Chetkovich generated enough data early on that allowed him to submit a grant proposal to NIH. In 2010, he was awarded an R21 (an exploratory/developmental research grant provided by NIH) in the amount of $275,000.

Epilepsy is a brain disorder of abnormally increased brain excitability that leads to seizures. A person is considered to have epilepsy when he or she has two or more unprovoked seizures. Advances in therapeutics have improved the lives of patients with epilepsy, yet seizures refractory to medical intervention remain a significant cause of disability. Additionally, many of the patients who do achieve acceptable control of seizures with anti-epilepsy drugs suffer side-effects from multi-drug combinations or high dosages, and may still develop drug resistance. Surgical removal of seizure-producing areas of the brain can control seizures in some patients, but is less effective in others. Thus, although substantial strides have been made in treating epilepsy, new therapies are warranted to help the many patients who suffer intractable seizures or complications from medical or surgical treatment efforts. Dr. Dane M. Chetkovich is using his 2009 BRF Seed Grant to attempt to develop new epilepsy treatments to better the lives of these patients.

The abnormal brain excitability that causes seizures often results from genetic or acquired deficiencies in ion channels that control neuronal excitability. Ion channels are proteins that form a pore across the plasma membrane of cells. In neurons, these channels help regulate the electrical activity by controlling the flow of ions across the membrane. When the regulation is disrupted, leading to a seizure, neurons may fire, or send signals on to other neurons in patterns that are very different from normal.

Dr. Chetkovich is focusing on a likely candidate for explaining abnormalities of excitability in both hereditary and acquired epilepsy—the hyperpolarization-activated cyclic nucleotide-gated (HCN) channel (h-channel). The h-channel family of ion channels consists of four different genes, HCN1-4, and has been implicated in epilepsy in animals and human patients. Dr. Chetkovich's project will examine whether using engineered viruses to produce HCN2 in abnormal areas of epileptic brain can stop seizures. These experiments aim to develop and test techniques for viral gene therapy in an animal model of epilepsy with the ultimate goal of translating these techniques to patients with intractable epilepsy.

Letter from the Treasurer

The Brain Research Foundation, like many other nonprofits, weathered some difficult times due to the historic financial crisis in 2008. Even though our investment performance was down, we minimized our losses and outperformed the Lipper Balance Fund Index (an average of publicly available mutual funds that invest in both stocks and bonds). Because we continue to follow our investment policies, the Foundation was still able to maintain our budgeted level of grant making in both fiscal years 2008 and 2009 (fiscal year from July 1st to June 30th). We are pleased to report that in fiscal year 2009, our investment growth was reassuring and once again outperformed the Lipper Balance Fund Index.

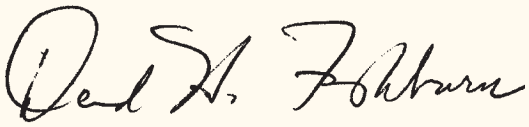
In fiscal year 2009, the Brain Research Foundation expanded its Fay/Frank Seed Grant Program to the greater Chicago area. This important program provides start-up funds to researchers for innovative investigations that have the potential to lead to new understanding and treatments of neurological disorders. We were pleased to be able to increase the total program funding by $200,000 to a total of $600,000, enabling us to better help researchers initiate new projects.

The following is a summary of our income and major expenses for fiscal years 2008 and 2009. In addition, we have provided a condensed balance sheet for those fiscal years. During both fiscal years, the Brain Research Foundation supported over $1.2 million in neuroscience research. In fiscal year 2009, we acquired $2,055,786 through our merger with the Children's Brain Research Foundation. Our total assets at the end of fiscal year 2009 were in excess of $9 million.

We are proud to report that finances are encouraging in fiscal year 2010. Through more favorable market performance and substantially higher donor contributions, our total assets on December 31, 2009 had increased to $10.3 million. We encourage you to review our audited financial statements on our website or contact the Brain Research Foundation office.

With the support of its Board of Directors, staff and donors, the Brain Research Foundation continues to be a financially strong organization that provides critical funding for exciting new research projects and valuable educational programs. We will work hard to sustain your support and fulfill our mission.

Sincerely,



David H. Fishburn
Treasurer

Financial Statements

Highlights of Income Statement
Years Ended June 30, 2008 and June 30, 2009

	2008	2009
Beginning net assets	$ 8,950,062	$ 7,952,704
Assets acquired through merger with CBRF	–	2,055,786
Contributions	822,047	689,128
Interest and dividends	318,525	287,199
Net realized loss on investments	(123,741)	(166,728)
Net unrealized loss on investments	(448,229)	(837,510)
Total	$ 9,518,664	$ 9,980,579
Expenses		
Program services	$ 1,232,426	$ 1,224,397
Supporting services	333,534	364,194
Total	$ 1,565,960	$ 1,588,591
Total net assets	**$ 7,952,704**	**$ 8,391,988**

Financial Statements (continued)

Statement of Financial Position
As of June 30, 2008 and 2009

Assets		**2008**		**2009**
Cash	$	34,958	$	26,851
Current prepaid expenses and deposits		13,250		19,095
Investments		8,678,419		9,185,207
Contributions receivable		150,000		155,500
Property and equipment		8,262		23,351
Other assets		155,651		5,651
Total	**$**	**9,040,540**	**$**	**9,415,655**

Liabilities and Net Assets		**2008**		**2009**
Liabilities				
Current liabilities	$	552,919	$	238,750
Long-term liabilities		534,917		784,917
Total liabilities	$	1,087,836	$	1,023,667
Net Assets				
Unrestricted	$	3,970,457	$	5,236,860
Unrestricted-Board-designated		2,821,592		2,637,363
Temporarily restricted		1,160,655		517,765
Total net assets	$	7,952,704	$	8,391,988
Total	**$**	**9,040,540**	**$**	**9,415,655**

Our Heartfelt Thanks

We rely on the generosity of our donors to continue the important work we do. With support from our Board of Trustees, corporations, foundations, longtime friends and new donors, we made tremendous progress in research and education during the last two years.

Our comittment to advancing neuroscience research depends on the continued support of all of you who share in our mission. With your help, we will continue to fund discoveries that shape the way we conquer disorders of the brain.

Thank you.

Donors to the Brain Research Foundation

$250,000+

Dr. Ralph and Marian Falk Medical Research Trust

The Pioneer Fund

$50,000–$249,999

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