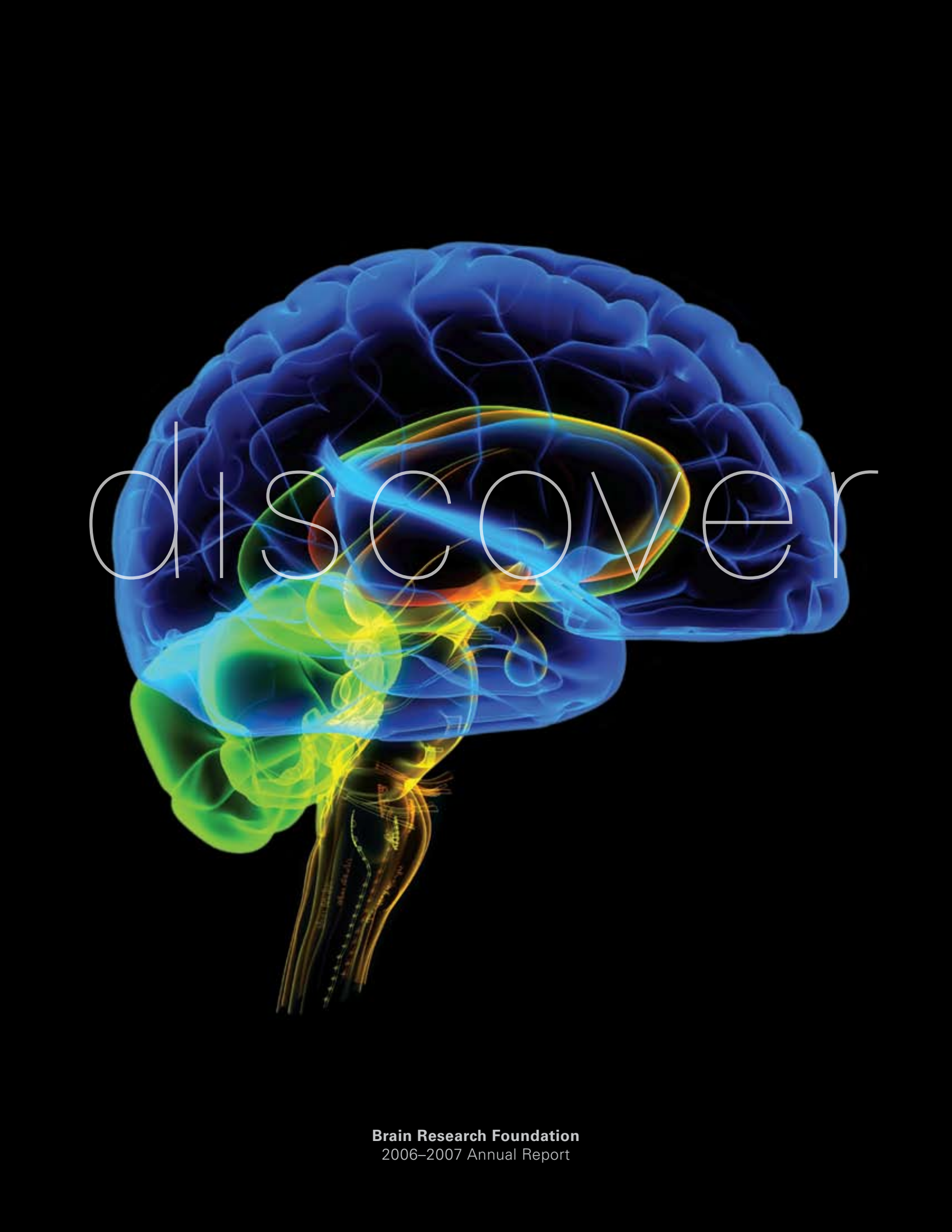
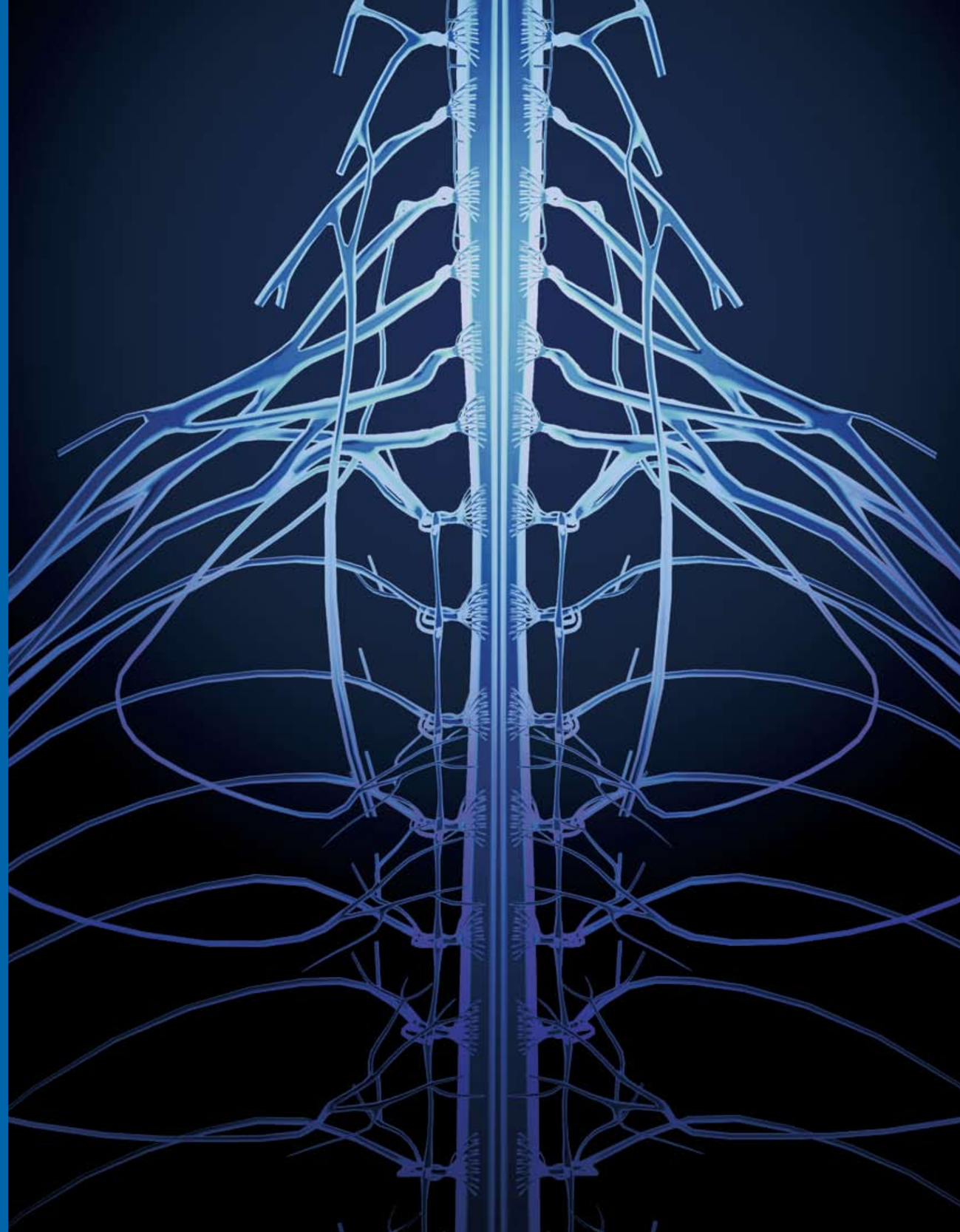


Brain Research Foundation
2006–2007 Annual Report

The Brain Research Foundation is committed to supporting neuroscience research that leads to advances in understanding of brain function and treatment of brain-related disorders.

This Foundation is distinctive in its comprehension that advancing the understanding of the brain will help all neurological disorders.

We deliver this commitment through our Seed Grant Program which provides start-up money for innovative neuroscience research projects.



Letter from the Brain Research Foundation

After more than five decades of supporting brain research, the Brain Research Foundation and its mission continue to adapt and grow to fund the most effective research within the ever advancing fields of science. When the Foundation began in 1953, we were supporting "brain research", but since then our areas of support have expanded, our fundraising has expanded and our vision has expanded.

We continue to support cutting-edge research on the brain, but we have also grown in our understanding of what areas of research should be supported that will someday develop ways to prevent and treat the neurological disorders that affect millions of people. We realize that understanding the function of the nervous system, both central and peripheral, is an absolute necessity. That is why a considerable portion of the scientific community has dedicated their lives to a field of research that is devoted to the study of the nervous system known as *neuroscience*.

Neuroscience is the study of the brain, the spinal cord, and networks of sensory nerves throughout the body. Over the years, just like the Brain Research Foundation, the scope of neuroscience has broadened to include any research of the central nervous system (brain and spinal cord) and peripheral nervous system (all other nerves outside of the central nervous system). Because neuroscience is such a rapidly progressing science, today it encompasses a wide variety of subfields, including neuroanatomy, neurophysiology, neurobiology, neurochemistry, neuropharmacology, neuropathology, psychiatry, neurology, neurosurgery, neuroimaging, clinical psychology, and several others. The Brain Research Foundation has funded these areas and others to impact scientific discovery. It is this array of research that will lead to important information on how the human brain functions; and how the nervous system develops and matures through life. Once we understand how the nervous system works, only then can we begin to repair it.

The Brain Research Foundation and the study of the nervous system both grew in the second half of the 20th century. Neuroscience research intensified primarily because of the advancements in scientific techniques such as molecular biology, genetics and brain imaging. The Brain Research Foundation flourished because of supporters who became involved through the years. Some were acquaintances of trustees and others were friends of a friend, but most were people with personal stories of loved ones touched with brain illnesses. They wanted to make a difference by supporting research.

The Foundation has always provided unwavering support for new directions in research, especially our contributions to scientists through our successful Seed Grant Program. As you glance at the list of our latest recipients, you will see the wide variety of science that we fund to enhance the quality and amount of information about the nervous system, creating the necessary knowledge from which scientists can build.

You will read in this report that funding the pilot stage of an innovative scientific idea can have enormous rewards. Although we only share with you five scientists' stories, we are intensely proud of all these investments, and will continue to fund top-quality science that will produce tangible results in the years to come.

Neuroscience holds great promise for understanding devastating illnesses like Alzheimer's disease, epilepsy, schizophrenia and multiple sclerosis. The Brain Research Foundation looks forward to funding the future—who knows what wonders we will touch.

Sincerely,

Thomas A. Reynolds III
President

Terre A. Sharma, Ph.D.
Executive Director

Expanding Discovery Through Seed Grants

Supporting groundbreaking research in neuroscience is the sole function of the Brain Research Foundation's Fay/Frank Seed Grant Program. The Brain Research Foundation realizes that cultivating early-stage research and bold ideas is the best way to impact science. Established in 1981 by William E. Fay, Jr. and Clinton E. Frank, the Seed Grant Program represents the most important work the Foundation does: funding the pilot stage of the innovative ideas and promising investigations that drive advances in our knowledge of how the human brain functions.

The Fay/Frank Seed Grant Program Research Seed Grant Program is a competitive, peer-reviewed program which provides initial support for new, long-term programs of collaborative interdisciplinary research that will have strong potential to attract external funding. Scientists in the area of basic, translational or clinical research are able to explore interesting new research avenues without undue delay, generating the necessary preliminary data that can be used to develop a competitive grant proposal to the National Institutes of Health (NIH) or other external funding sources. Our goal is to have this initial effort succeed in opening future opportunities for research, collaboration and scientific advancement.

Seed grants are an investment in promise, allowing new insights and approaches to be explored, tested, and proven, and this promise has been powerfully repaid. Since the Program's inception, the Brain Research Foundation has distributed over $6.9 million to neuroscientists. Over the years, we have increased both the number of recipients and the seed grant amount.

In 2006 and 2007, the Foundation provided a total of $800,000 in seed grants to 32 dedicated researchers. For our increased commitment, we have seen an increase in "payback." The figures for 2006 show that for every $1 the Brain Research Foundation has invested in these new ideas, researchers have attracted over $25 in future funding.

The five seed grant recipients who we've profiled in this report illustrate the Brain Research Foundation's investment in discovery. Some are very basic research, while some will hopefully have great clinical impact in the near future. In one story, the research is just a few experiments away from making a significant difference in a patient's life. All of them are steps of discovery.

2006-2007 Seed Grant Program Recipients

Brain Research Institute at The University of Chicago

Glyn Dawson, Ph.D.
Department of Pediatrics
Phospholipids, endosomes and brain function in Batten disease

Stephanie Dulawa, Ph.D.
Department of Psychiatry
Identifying novel genes for aggressive behavior

Brian Gehlbach, M.D.
Department of Medicine
Improving the sleep and circadian rhythms of mechanically ventilated patients

Fernando Goldenberg, M.D.
Department of Neurology
Tackling cerebral salt wasting: searching for the cause and cure

Robert Ho, Ph.D.
Department of Organismal Biology and Anatomy
The autism susceptibility gene, MET, and its role during neural development

Naoum Issa, M.D., Ph.D.
Department of Neurobiology
The effect of scene-statistics on cortical maps

Kristen Jacobson, Ph.D.
Department of Psychiatry
A pilot study of genetic and environmental influences on amygdala, orbital medial prefrontal cortex and dorsal anterior cingulate cortex activation: a twin study of fMRI

Adil Javed, M.D., Ph.D.
Department of Neurology
Immunological and molecular mechanisms involved in the pathogenesis of Devic's disease

Royce 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 glioma

Chunyu Liu, Ph.D.
Department of Psychiatry
Identification of cis-regulatory elements for gene expressed in human brain

Daniel Llano, M.D., Ph.D.
Department of Neurology
Investigation of attentional modulation via fronto-thalamic networks

Christopher Lowe, Ph.D.
Department of Organismal Biology and Anatomy
Development and evolution of brain signaling centers: hemichordates as a simple system for characterizing neurodevelopment

Jason MacLean, Ph.D.
Department of Neurobiology
The role of ongoing cortical activity in sensory information processing: a comparative evaluation across modalitities

Dario Maestripieri, Ph.D.
Department of Comparative Human Development
Effects of early experience on the development of brain monoamine systems and behavior

Martha McClintock, Ph.D.
Department of Psychology
Neural mechanisms of androstadienone

Kathleen Millen, Ph.D.
Department of Human Genetics
A cause of epilepsy and cerebellar ataxia

Deborah Nelson, Ph.D.
Department of Neurobiology, Pharmacology and Physiology
ClC3 chloride channels as regulators of synaptic efficacy

M. Kelly Nicholas, M.D., Ph.D.
Department of Neurology and Medicine
A novel fusion protein, FABP7R4, in an experimental brain tumor model

Abraham Palmer, Ph.D.
Department of Human Genetics
Development of siRNA microinjections to evaluate candidate genes for behavioral phenotypes

Clifton Ragsdale, Ph.D.
Department of Neurobiology
Molecular mechanisms of neocortical cell type specification

Callum Ross, Ph.D.
Department of Organismal Biology and Anatomy
Kinematics, electromyography, and cortical activity during reaching, grasping and feeding in macaque primates

Ilya Ruvinsky, Ph.D.
Department of Ecology and Evolution
Experimental and computational studies of pan-neuronal gene regulation

Kamal Sharma, Ph.D.
Department of Neurobiology
2006—Genetic analysis of motor circuits
2007—Neural control of respiration

Sangram Sisodia, Ph.D.
Department of Neurobiology
Enrichment-induced neurogenesis in adult mouse hippocampus: modulation by FAD-linked PS1 variants

Ya-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
Alschuler Scholar
These grants were made possible by the Leonore and Ernest Alschuler Fund.
2006—Regulation of Alzheimer's disease Aβ production by lipid modification of γ-secretase
2007—Role of presenilin in dendritic spine formation

Paul Vezina, Ph.D.
Department of Psychiatry
Behavioral and dopaminergic sensitization by nicotine

Xiaoxi Zhuang, Ph.D.
Department of Neurobiology
Phasic dopamine in reward-based learning and addiction

Yimin Zou, Ph.D.
Department of Neurobiology
Molecular and cellular mechanisms of cortical development

Women's Council Seed Grants

Qian Chen, Ph.D.
Department of Psychiatry
Modeling of Parkinson's disease in the mouse

Elena Rozhkova, Ph.D.
Department of Neurology
Advanced bio-inorganic materials for targeted thermal and photodynamic glioblastoma multiforme therapy

Comprehending the Organization and Function of the Nervous System

Ilya Ruvinsky, Ph.D.

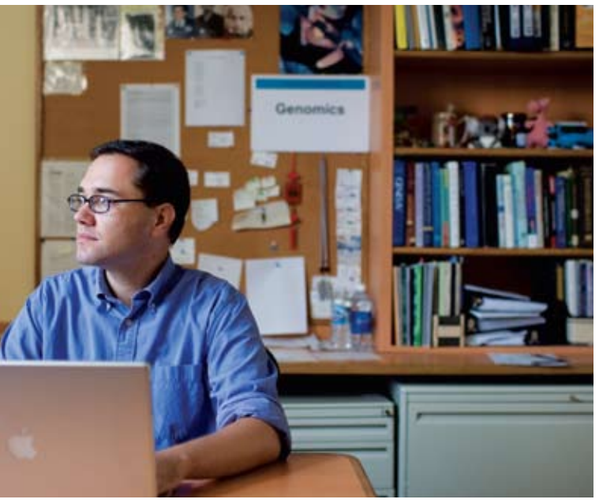
The title of this article could be the mission statement of the Brain Research Foundation—to support research that will uncover the organization and function of the nervous system. Once you understand how something works, you can then begin to repair it.

The brain is made up of neurons. Despite remarkable variation in the structure and function, all neurons possess a shared set of attributes that are not seen in cells elsewhere in the body. Do these shared attributes make neurons more susceptible to disease causing agents and prevent regeneration following injury to the brain? Not surprisingly, all neurons also express certain genes, called pan-neuronal genes that are not activated by cells elsewhere in the body. Some of the pan-neuronal genes are known, but others need to be identified. The expression of pan-neuronal genes is in turn regulated by factors that recognize specific promoter sequences, shared by these genes. Pan-neuronal genes, conserved DNA sequences in their promoters and factors that bind to these conserved sequences are hidden in the genomes of all organisms that have neurons.

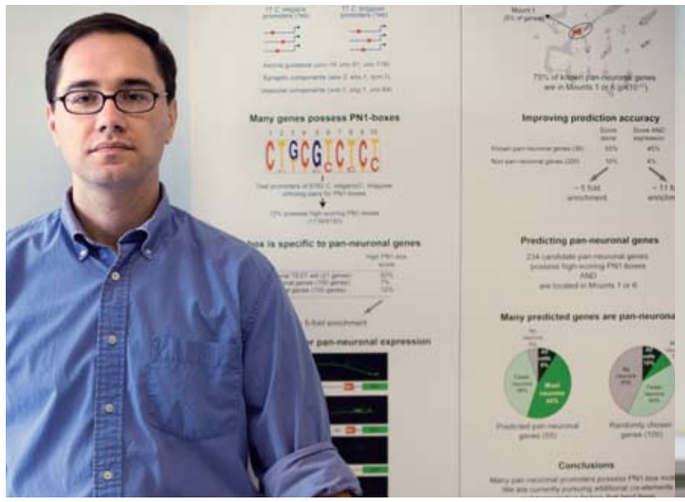
Dr. Ilya Ruvinsky, Assistant Professor of Ecology and Evolution at The University of Chicago, is using a cutting-edge genomics approach to generate a complete list of pan-neuronal genes and to understand their regulation at the transcriptional level. His research would identify parts of the genome that regulate the expression of pan-neuronal genes. He has chosen to embark upon this task by using a simple, well-established model organism, a small roundworm, *C. elegans*. The worm is about 1 mm in length, its nervous system is comprised of 302 neurons whose pattern of connectivity has been completely mapped. The genome of this worm consists of approximately 20,000 genes, and has been sequenced. Many of the genes associated with human disease are included among these. What makes this worm an attractive experimental model for Dr. Ruvinsky's research is the availability of detailed genome sequence data and their ability to knock out gene expression using RNA interference (RNAi). By silencing the function of a gene, Dr. Ruvinsky's research team would determine whether or not that gene regulates the expression of pan-neuronal genes. Gene by gene, they will test whether neurons are able to maintain the expression of a pan-neuronal gene. In parallel studies Dr. Ruvinsky will use computational genomics methods to search the whole genome for DNA sequences that are responsible for ensuring that pan-neuronal genes are expressed in neurons.

These studies highlight an important fact. The more we learn about neurological disorders, the more we become aware of the remarkable complexity of the brain and its constituent neurons. One might wonder why the nervous system is susceptible to mutations in some genes when the rest of the body is not. Although every cell has a full complement of the whole genome, only those genes that the cell chooses to express give it a set of properties. Neurons, for reasons that we do not yet understand, express certain genes that are excluded from the rest of the body. Supporting research into the basic functioning of genes and neurons will lead to a better understanding of organization and function of the nervous system, and thus to potential diagnosis and treatment of human disorders.

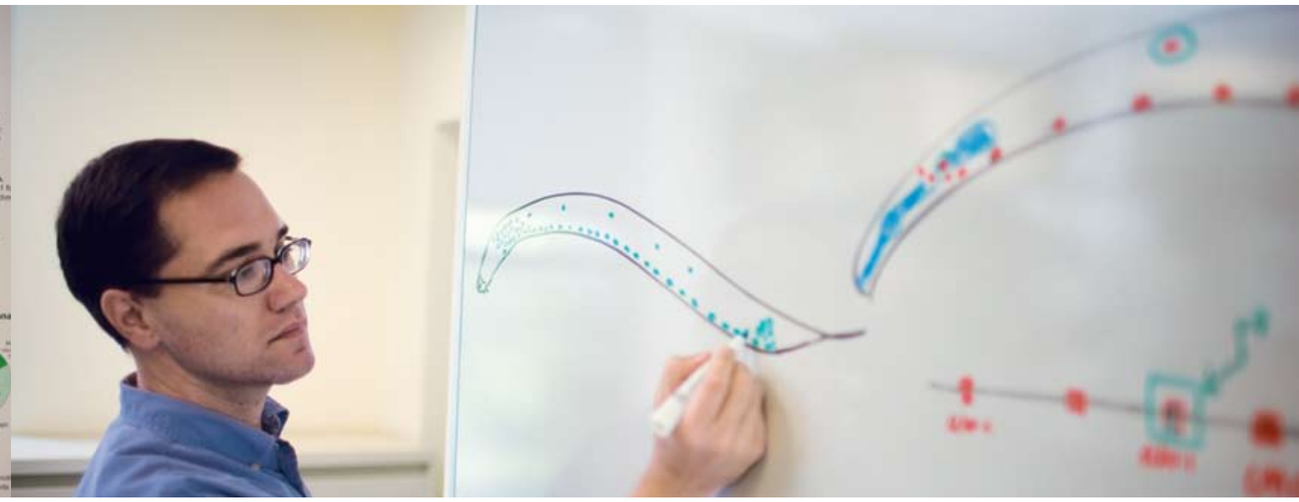
Dr. Ruvinsky utilizes computational and molecular techniques in an effort to understand the regulation of broad neuronal expression.



He will use RNAi (interference) to identify transcriptional regulators of broadly expressed neuronal genes in a small roundworm, C. elegans.



The C. elegans is about 1 mm in length. The genome of this worm consists of approximately 20,000 genes, and has been sequenced.



Understanding Nicotine Addiction

Paul Vezina, Ph.D.

Tobacco use in humans is the single most preventable cause of death in the United States. According to a statistical review by the National Cancer Institute, cigarette smoking accounts for 30 percent of all cancer deaths annually in America. Despite these facts, almost a quarter of the American population continues to smoke—in part because of their dependence on nicotine.

There are more than 4,000 chemicals found in the smoke of tobacco products, but nicotine is the primary reinforcing component that acts on the brain and causes addiction. Addiction is characterized by compulsive drug seeking and use, even though these behaviors increase the probability of dire health consequences for the individual. Most smokers realize that tobacco use is harmful and express a desire to stop using it. Unfortunately, less than 10 percent of the people who try to quit are successful.

Although considerable progress has been made in understanding the neural effects of nicotine, the knowledge falls short of what it takes to successfully stop tobacco use. The aim of Dr. Paul Vezina, Associate Professor of Psychiatry at The University of Chicago, is to ultimately understand how nicotine acts to initiate self-administration behaviors and how repeated exposure to nicotine produces changes that promote their continued expression.

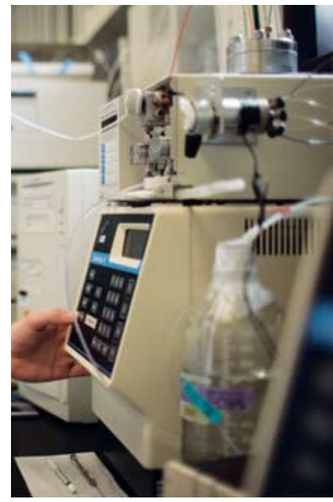
With his Brain Research Foundation Seed Grant, Dr. Vezina initiated experiments that helped identify the brain regions containing nAChRs (nicotinic acetylcholine receptors) that are necessary for the induction of locomotor and dopaminergic sensitization by nicotine. This form of plasticity has been linked to the development of addictive states. By using the antagonist mecamylamine, he assessed the effect of blocking nAChRs in different brain regions on the induction of long lasting sensitization by nicotine. So far, Dr. Vezina has found that nAChRs in the midbrain ventral tegmental area are critical for the development of this form of plasticity. Interestingly, blocking nAChRs in forebrain regions like the nucleus accumbens was without effect. These two nuclei are interconnected in a circuit well known to participate in the generation of appetitive behaviors. When activity in this pathway becomes sensitized, these behaviors become exaggerated and can lead to addiction.

Identification of those nAChR fields necessary for the induction of sensitization will help delineate the circuitry and neurotransmitters involved in the generation of maladaptive behaviors like those associated with addiction. This information could lead to the development of compounds and therapeutic approaches capable of preventing these kinds of neuroadaptations that inevitably result from exposure to drugs like nicotine.

These experiments supported by the Brain Research Foundation Seed Grant Program helped Dr. Vezina compete successfully with two of his colleagues at The University of Chicago for a program project grant from the NIH. This grant will fund the continued study of the effects of exposure to nicotine on nAChRs, neuronal excitability and behavior. Already Dr. Vezina and his colleagues are characterizing the effects of nicotine exposure on the regulation of nAChRs in those brain areas identified in the above studies. The overall aim of this program project grant is to use a multidisciplinary approach to examine how the molecular, cellular and behavioral consequences of nicotine exposure impacts the ability of this drug to support self-administration over prolonged periods of time.

Dr. Vezina helped identify the brain regions containing nAChRs that are necessary for induction of locomotor and dopaminergic sensitization by nicotine.

These experiments helped Dr. Vezina and colleagues obtain a program project grant from NIH that will characterize the long-term molecular, cellular and behavioral consequences of exposure to nicotine.



Genetic Influences on Behavior

Kristen C. Jacobson, Ph.D.

Dr. Kristen Jacobson, Assistant Professor of Psychiatry at The University of Chicago, has always been interested in environmental experiences and their effect on behavior, but thought there was something more to why people behave how they do. It is easy to blame poor parenting, or the wrong peer group to explain why a child makes bad decisions. But Dr. Jacobson wanted to show that sometimes we make choices—good and bad—because we are who we are. And we are who we are, in part, because of our genetic make-up.

A twin design is a powerful way to examine the contribution of genes and environment on behavior. Dr. Jacobson's project, which was awarded a Brain Research Foundation Seed Grant, proposes to collect functional imaging data related to impulsivity and socioemotional information processing from adult twin pairs. Twins will be subjected to impulsivity and emotional tasks in order to better understand the genetic factors behind neurophysiological response patterns that are related to the development of aggression and antisocial behavior.

This study will focus on three brain areas that underlie aggression and antisocial behavior: the amygdala (involved in fight-or-flight response), the orbital medial prefrontal cortex (OMPC) and the dorsal anterior cingulated cortex (dACC). Sixteen sets of twins, eight identical and eight fraternal, will perform various tasks while whole-brain functional magnetic resonance imaging is carried out. These performance tasks are well-established activities used to assess a person's impulsivity and aggression.

In the first task, subjects are shown photographs of faces that illustrate varying degrees of emotion while brain images of the amygdala and OMPC are captured. They are asked to rate the photograph's emotion as positive, negative or neutral. In addition to faces, they are also shown emotional pictures that display pleasant, neutral or unpleasant scenes (puppies, a snake, car accident, etc.). People that exhibit aggressive behavior interpret these pictures differently. For example, aggressive people are more likely to view neutral faces as hostile, compared to non-aggressive people.

Another task is called "Go/No-go" which looks at motor and cognitive impulsivity. The mechanism behind impulsivity behavior is thought to be the dACC. In this performance task, the subject views letters that are flashed on a computer screen as they are in the magnetic resonance scanner. For example, the subject is told to press a button when they see a "red D," but is not supposed to push the button for any other letter, including other colored "D's." This task is extremely hard for children with attention deficit hyperactivity disorder. In fact, extremely impulsive kids can't stop themselves from hitting the button repeatedly. In the scanner, Dr. Jacobson is able to see what the brain does when the correct answer flashes on the screen, and she can also see what happens in the brain before the button is pushed for the wrong letter.

The data obtained from these tasks will be combined with lab-based behavioral assessments and self-reports of aggression and antisocial behavior to determine whether the genetic factors that underlie individual differences in brain activation levels overlap with genetic influences on aggression and antisocial behavior. If in fact the way brains are wired and the way this information gets transmitted are heritable, identical twins' brains should be more similar than fraternal twins' brains, illustrating a genetic influence on brain pathways.

Manipulate the Immune System to Destroy Brain Tumors

Maciej S. Lesniak, M.D.

High grade gliomas represent the most common primary malignant tumor of the adult central nervous system. Unfortunately, the median survival after surgical intervention alone is only six months and the addition of radiotherapy can extend this time to only nine months. More aggressive therapeutic regimens are imperative to improve the survival rates of these patients.

Dr. Maciej Lesniak, Associate Professor of Surgery at The University of Chicago, is working on a novel treatment strategy that will specifically target tumor cells and spare normal cells by manipulating the immune system—immunotherapy. Immunotherapy means a treatment based upon the concept of modulating the immune system to fight disease, in this case destroy tumors.

Brain tumors have been previously considered to exist in an area that restricted immune response. However, recent studies have indicated that the immune system is finely regulated within the brain and that immunotherapeutic approaches could be utilized in the treatment of malignant brain tumors. Dr. Lesniak used his Brain Research Foundation Seed Grant to determine if regulatory T cells (Treg), a component of the immune system, found in malignant gliomas selectively inhibit the host immune response within the CNS and contribute to the rapid progression of brain cancer.

The immune system is a complex group of defense responses that protect against infection by identifying and killing pathogens and tumor cells. An important component of the immune response is specialized white blood cells called T cells. There are a variety of subsets of T cells. Dr. Lesniak's project studied T cells that expressed the cell-surface glycoproteins CD4 and CD25 and the transcription factor FoxP3. CD4+CD25+FoxP3+ regulatory T cells (Treg) suppress immune responses of other cells. Treg are involved in shutting down immune responses after they have successfully tackled invading organisms and also in keeping in check immune responses that may potentially attack one's own tissues.

Dr. Lesniak utilized a mouse glioma model to examine the presence of Treg in CNS tumors. He manipulated the immune response by depleting the CD4+CD25+FoxP3+ regulatory T cells (Treg) to determine what effect the absence of Treg would have on intracranial tumor growth and progression. His results showed that Treg are present and in fact elevated in intracranial versus peripheral tumors. It seems this high number of Treg permits the aggressive growth rate seen in malignant brain tumors. When the suppressing effect of Treg is interfered with using an anti-CD25 monoclonal antibody, the immune system is activated and can destroy tumor cells, increasing the survival rate of mice with intracranial tumors.

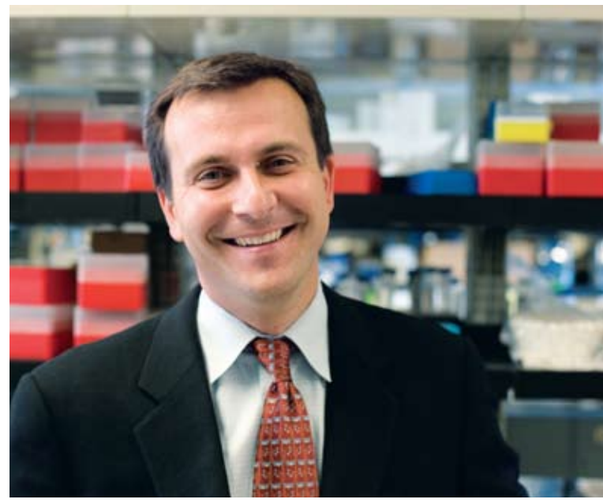
In addition, Dr. Lesniak demonstrated that Treg progressively infiltrate gliomas with increasing tumor grade (tumor aggressiveness). Heme oxygenase-1 (HO-1), an enzyme that catalyzes the degradation of heme and is induced in response to stress such as oxidative stress, hypoxia and cytokines, has been shown to accumulate during glioma progression and to play a critical role in FoxP3 mediated immune suppression. The expression of FoxP3 and HO-1 was analyzed in patients with different grades of gliomas. It was observed that (1) the highest level of FoxP3 was expressed in patients with the most aggressive gliomas, grade IV; and (2) the expression of HO-1 directly correlates with the expression of FoxP3.

Based on these studies, it is clear that Treg play a role in the rapid progression of brain cancer, and may be an optimal target for new therapies. By interfering with Treg to remove the suppression of the immune system, the immune system will now be able to destroy cancer cells. The ultimate goal would be to design appropriate immunotherapeutic interventions that could be used in clinical applications to increase the survival rate of patients with gliomas.

Dr. Lesniak focuses his attention on brain tumor immunotherapy as a new treatment strategy that would specifically target tumor cells and spare normal cells.

A high grade glioma is a type of primary brain tumor that is fast growing and carries a dismal prognosis. The average survival after surgery is only six months.

Dr. Lesniak's lab has been able to manipulate immune responses in mice, activating the immune system to destroy tumors cells, and increasing their survival rate.



Sleep Disruption May Impair Recovery from Clinical Illness

Brian K. Gehlbach, M.D.

Sleep is considered restorative and important for illness recovery. In converse, sleep disruption has been shown to adversely affect recovery from illness by reducing cognition, immune function, respiratory drive and cardiovascular function. Yet, the sleep of critically ill patients has received little attention, despite the clinical importance and possibility of reducing mortality. Dr. Brian Gehlbach, Assistant Professor of Medicine at The University of Chicago, recognized this need for investigation of sleep disruption in critically ill patients undergoing mechanical ventilation, and will utilize the Brain Research Foundation Seed Grant to elucidate the correlation between sleep quality and clinical outcomes.

The experience of critical care encompasses a variety of stimuli and interventions that may interfere with normal sleep. The physical environment of a hospital room has been shown to be poorly suited for normal sleep. Constant background noise levels that frequently range between 60 and 80 dB and ambient light that frequently is not in phase with the patient's normal light/dark cycle can interfere with a patient's "internal body clock" or circadian rhythms. In addition, procedures and nursing care are generally performed without consideration of the time of day which can also disrupt sleep. Finally, the critical illness itself may disturb sleep through the experiences of pain, fear or central nervous system disturbance.

While a handful of studies have demonstrated circadian rhythm disturbances in patients on ventilators, the infrequent sampling of hormone levels to determine rhythmicity and the absence of concurrent polysomnography (sleep recording) have left a lot of unanswered questions. Dr. Gehlbach plans to definitively characterize the circadian rhythms and quality of sleep of acutely ill patients undergoing mechanical ventilation for respiratory failure.

The first aspect of the project will characterize the sleep quality and circadian rhythms of critically ill patients on a ventilator. All patients in the study will undergo continuous polysomnography to determine sleep quality. It is expected that most patients will exhibit reduced and fragmented REM sleep patterns. In addition, the levels of 6-sulfatoxymelatonin (the major metabolite of the pineal hormone melatonin) will be measured at hourly intervals in order to assess circadian rhythmicity, which is expected to be disrupted.

In addition to analyzing the patient's sleep quality and circadian rhythm, Dr. Gehlbach and his colleagues will manipulate the hospital room environment to promote better sleep by reducing noise, enforcing a normal light-dark cycle, and if possible performing nursing care according to time of day. This management of the environment should strengthen circadian rhythms and improve sleep quality.

The ultimate outcome of this study is to speed up recovery and lessen the duration of mechanical ventilation. It has been shown that the presence of delirium has been associated with adverse clinical outcomes, including longer hospital stay and increased six-month mortality for patients with respiratory failure. However, the role of sleep disruption in mediating delirium has not been investigated. If a relationship between disrupted sleep and delirium in critical illness is determined, doctors could utilize practical strategies that can be employed at the bedside to greatly impact a patient's recuperation.

Dr. Gehlbach is confident that this previously neglected but extremely important area of research will generate results that are immediately applicable to the care of our sickest patients, and that something as simple as turning off the light at night may help save a life.

Independent Auditor's Report

Board of Directors
Brain Research Foundation
Chicago, Illinois

We have audited the accompanying statements of financial position of the Brain Research Foundation (the Foundation) as of June 30, 2007 and 2006, and the related statements of activities and cash flows for the years then ended. These financial statements are the responsibility of the Foundation's management. Our responsibility is to express an opinion on these financial statements based on our audits.

We conducted our audits in accordance with auditing standards generally accepted in the United States of America. Those standards require that we plan and perform the audit to obtain reasonable assurance about whether the financial statements are free of material misstatement. An audit includes examining, on a test basis, evidence supporting the amounts and disclosures in the financial statements. An audit also includes assessing the accounting principles used and significant estimates made by management, as well as evaluating the overall financial statement presentation. We believe that our audits provide a reasonable basis for our opinion.

In our opinion, the financial statements referred to above present fairly, in all material respects, the financial position of Brain Research Foundation as of June 30, 2007 and 2006, and the results of its operations and its cash flows for the years then ended in conformity with accounting principles generally accepted in the United States of America.

Blackman Kallick Bartelstein, LLP

Blackman Kallick Bartlestein, LLP

August 22, 2007

Financial Statements

Statements of Financial Position

June 30, 2007 with Comparative Totals as of June 30, 2006

Assets	**2007**			**2006**
	Unrestricted	Temporarily Restricted	Total	Total
Current Assets				
Cash and cash equivalents	$ 191,216	$ 171,572	$ 362,788	$ 364,813
Current prepaid expenses and deposits	24,159	—	24,159	11,389
Investments	5,617,445	1,479,555	7,097,000	6,739,982
Investments — Board-designated	2,238,402	—	2,238,402	2,435,452
Total Current Assets	**8,071,222**	**1,651,127**	**9,722,349**	**9,551,636**
Property and Equipment				
Leasehold improvements	—	—	—	128,935
Furniture and equipment	65,722	—	65,722	62,478
Software	23,759	—	23,759	23,759
Less accumulated depreciation	(80,862)	—	(80,862)	(145,203)
Net Property and Equipment	**8,619**	**—**	**8,619**	**69,969**
Noncurrent Assets — Security deposits	5,200	—	5,200	—
Total Assets	**$ 8,085,041**	**$ 1,651,127**	**$ 9,736,168**	**$ 9,621,605**

The accompanying notes are an integral part of the financial statements

Liabilities and Net Assets (Exhibit A)	**2007**			**2006**
	Unrestricted	Temporarily Restricted	Total	Total
Current Liabilities				
Accounts payable and accrued expenses	$ 1,189	$ —	$ 1,189	$ 3,620
Neuroscience Professorship payable — Current portion	400,000	—	400,000	400,000
Total Current Liabilities	**401,189**	**—**	**401,189**	**403,620**
Neuroscience Professorship Payable (Net of portion included in current liabilities)	384,917	—	384,917	755,318
Total Liabilities	**786,106**	**—**	**786,106**	**1,158,938**
Net Assets				
Unrestricted	5,060,533	—	5,060,533	4,504,623
Unrestricted — Board-designated	2,238,402		2,238,402	2,435,452
Temporarily restricted	—	1,651,127	1,651,127	1,522,592
Total Net Assets (Exhibit B)	**7,298,935**	**1,651,127**	**8,950,062**	**8,462,667**
Total Liabilities and Net Assets	**$ 8,085,041**	**$ 1,651,127**	**$ 9,736,168**	**$ 9,621,605**

Financial Statements (continued)

Statements of Activities (Exhibit B)

Year Ended June 30, 2007 with Comparative Totals for the Year Ended June 30, 2006

	2007			2006
	Unrestricted	Temporarily Restricted	Total	Total
Revenues				
Support				
Contributions	$ 306,317	$ 748,286	$ 1,054,603	$ 1,422,444
Fundraising event revenue, net of expenses	—	—	—	4,718
Total Support Revenue	**306,317**	**748,286**	**1,054,603**	**1,427,162**
Income (loss) from investing activities				
Interest and dividends	227,857	140,314	368,171	350,451
Net realized gain (loss) on sale of investments	355	(3,035)	(2,680)	(193,059)
Net unrealized gain on investments	572,884	240,152	813,036	228,911
Total Income from Investing Activities	**801,096**	**377,431**	**1,178,527**	**386,303**
Net assets released from restriction	997,182	(997,182)	—	—
Total Revenues	**2,104,595**	**128,535**	**2,233,130**	**1,813,465**
Expenses				
Program services				
Fay/Frank Seed Grant Fund	459,728	—	459,728	431,202
Discovery Campaign and Neuroscience Professorship	86,467	—	86,467	117,521
Special Fund	656,251	—	656,251	900,857
Public Information, Health and Education	282,208	—	282,208	339,209
Total Program Services	**1,484,654**	**—**	**1,484,654**	**1,788,789**
Supporting services				
General administration	146,530	—	146,530	193,531
Fundraising expenses	114,551	—	114,551	119,977
Total Supporting Services	**261,081**	**—**	**261,081**	**313,508**
Total Expenses	**1,745,735**	**—**	**1,745,735**	**2,102,297**
Change in Net Assets Before Loss on Uncollectible Pledge	**358,860**	**128,535**	**487,395**	**(288,832)**
Loss on Uncollectible Pledge	**—**	**—**	**—**	**(42,105)**
Change in Net Assets	**358,860**	**128,535**	**487,395**	**(330,937)**
Net Assets, Beginning of Year	**6,940,075**	**1,522,592**	**8,462,667**	**8,793,604**
Net Assets, End of Year (Exhibit A)	**$ 7,298,935**	**$ 1,651,127**	**$ 8,950,062**	**$ 8,462,667**

The accompanying notes are an integral part of the financial statements

Statements of Cash Flows (Exhibit C)

Years Ended June 30, 2007 and 2006

	2007	2006
Cash Flows from Operating Activities		
Change in net assets	$ 487,395	$ (330,937)
Adjustments to reconcile change in net assets to net cash used in operating activities		
Depreciation	64,594	67,953
Loss on uncollectible pledge	—	42,105
Net realized loss on sale of investments	2,680	193,059
Net unrealized gain on investments	(813,036)	(228,911)
Donated stock	(100,882)	(409,567)
Donated furniture and equipment	(3,244)	—
(Increase) decrease in		
Contributions receivable	—	165,000
Prepaid expenses and other	(17,970)	(11,314)
Increase (decrease) in		
Accounts payable and accrued expenses	(2,431)	2,923
Discovery Campaign payable	—	(324,893)
Neuroscience Professorship payable	(370,401)	(356,434)
Net Cash Used in Operating Activities	**(753,295)**	**(1,191,016)**
Cash Flows from Investing Activities		
Capital expenditures	—	(2,833)
Sale of investment securities	3,703,213	4,460,075
Purchase of investment securities	(2,951,943)	(3,417,298)
Net Cash Provided by Investing Activities	**751,270**	**1,039,944**
Net Decrease in Cash and Cash Equivalents	**(2,025)**	**(151,072)**
Cash and Cash Equivalents, Beginning of Year	**364,813**	**515,885**
Cash and Cash Equivalents, End of Year	**$ 362,788**	**$ 364,813**

The accompanying notes are an integral part of the financial statements

Notes to Financial Statements

Years Ended June 30, 2007 and 2006

Note 1— Summary of Significant Accounting Policies

Organization
The Brain Research Foundation (the Foundation) is a corporation organized under the Illinois Not-for-Profit Corporation Act. The Foundation is committed to promoting basic research and knowledge concerning the human brain.

Significant accounting policies consistently followed by the Foundation are summarized below:

Basis of Presentation
These financial statements have been prepared on the accrual basis of accounting and report amounts separately by class of net assets, which are defined as follows:

Unrestricted
Amounts that are currently available for use in the Foundation's operations and for the acquisition of equipment.

Unrestricted — Board-designated
Amounts that are currently designated by the Board for the use further described in Note 5.

Temporarily Restricted
Amounts that are stipulated by donors for specific operating purposes, restricted by time or purpose.

Support and Expenses
Contributions received and unconditional promises to give are measured at their fair values and are reported as an increase in net assets. The Foundation reports gifts of cash and other assets as restricted support if they are received with donor stipulations that limit the use of the donated assets, or if they are designated as support for future periods. When a donor restriction expires, that is, when a stipulated time restriction ends or purpose restriction is accomplished, temporarily restricted net assets are reclassified to unrestricted net assets and reported in the statement of activities as net assets released from restriction. For the years ended June 30, 2007 and 2006, all donor-restricted contributions are reported as temporarily restricted support and all restrictions that were met during the period are shown as releases from restriction.

Expenses are recorded when incurred in accordance with the accrual basis of accounting.

Cash Equivalents
For purposes of the statements of cash flows, the Foundation considers investments in money market accounts to be cash equivalents. The carrying value of cash equivalents approximates fair value as of June 30, 2007 and 2006.

Pledge Commitments
Unconditional promises to give that are expected to be collected within one year are recorded at net realizable value. Unconditional promises to give that are expected to be collected in future years are recorded at the present value of their estimated future cash flows. The discounts on those amounts are computed using interest rates based on the long-term federal rate applicable to the years in which the promises are received. Amortization of the discounts is included in contribution revenue. Conditional promises to give are not included as support until the conditions are substantially met.

Investments
Investments are recorded at quoted market prices. Contributions of marketable securities are recorded at fair market value as of the date of the gift. It is the Foundation's policy to sell such gifts of securities as soon as it is practical to allow for an orderly disposition. The realized gains and losses on investments sold are computed using the specific recorded cost of each security.

The Foundation's investments are exposed to various risks, such as interest rate, credit and overall market volatility. Due to these risk factors, it is reasonably possible that changes in the value of investments will occur in the near term and could materially affect the amounts reported in the statements of financial position. The Foundation places its cash, cash equivalents and marketable securities with high-quality institutions and, accordingly, limits its credit exposure.

Depreciation
Property, plant and equipment are valued at cost or fair market value for donated items. The Foundation's policy is to capitalize items with a cost exceeding $500. Depreciation is provided on the straight-line method over the estimated useful lives of the assets.

	Years
Furniture and equipment	3–7
Leasehold improvements	5
Software	3

Seed Grants
The Fay/Frank Seed Grant has been temporarily restricted by donors for the purpose of funding Seed Grants for researchers at the University of Chicago and is not available for general operating expenses or other uses. The Foundation segregated the Seed Grant funds in a separate investment account to provide specific annual grant support. In accordance with the spending policy, the Foundation uses 6% of the assets for the annual Seed Grant awards. The annual Seed Grant awards typically exceed the Foundation's spending policy and are released from restriction when the grants are awarded. See Note 5 for the portion of the Seed Grant investment account that has been released from restriction, but is designated by the Board for future Seed Grant Awards. Also see Note 7 for additional disclosures on the temporarily restricted net asset balance.

Committed to Discovery Campaign
The Committed to Discovery Campaign Fund (Discovery Campaign) had been temporarily restricted by donors for the purpose of funding the Committed to Discovery Campaign, a joint capital campaign with the University of Chicago to raise $25,000,000 for the Brain Research Institute. The campaign was to run from July 1, 1998 through June 30, 2001, but was extended until June 30, 2002 to reach the goal. As of June 30, 2006, the Foundation and the University of Chicago had received pledges of approximately $26 million and successfully met their joint goal. The campaign account was closed as of June 30, 2006 and the balance was transferred to the main investment account.

Special Gifts
The Special Fund has been set up to collect various donations that have temporary donor restrictions, but not a special program such as Seed Grants or the Discovery Campaign.

Women's Council
The Women's Council conducts Public Information and Health and Education programs for the Foundation. The Women's Council merged with the Foundation on July 1, 2005. The Council's assets were recorded at fair value as temporarily restricted assets by the Foundation. A temporarily restricted contribution of $318,631 was recorded as this was the total fair value of assets received as of the date of the merger.

Functional Allocation of Expenses
The costs of providing the various programs, fund-raising and other activities have been summarized on a functional basis in the schedule of functional expenses. Accordingly, certain costs have been allocated among the programs and fund-raising activities benefited based on time studies.

Management Estimates
The preparation of financial statements in conformity with accounting principles generally accepted in the United States of America requires management to make estimates and assumptions that affect the reported amounts of assets and liabilities and disclosure of contingent assets and liabilities as of the date of the financial statements and the reported amounts of revenues and expenses during the reporting period. Actual results could differ from those estimates.

Note 2 —Tax-Exempt Status

The Foundation is a not-for-profit organization that is exempt from income taxes under Section 501(c)(3) of the Internal Revenue Code. Accordingly, the accompanying financial statements do not reflect income taxes.

Note 3— Cash and Cash Equivalents

Cash and cash equivalents consist of the following:

	2007	2006
Cash	$ 20,941	$ 1,371
Money market funds	$ 341,847	$ 363,442
	$ 362,788	**$ 364,813**

The Foundation maintains its cash and cash equivalents in bank accounts, which at times may exceed federally insured limits. The Foundation has not experienced any losses in such accounts. The Foundation believes it is not exposed to any significant credit risk on cash and cash equivalents.

Note 4 — Investments

Investments are recorded at fair value. Investments consist of the following as of June 30, 2007 and 2006:

	2007	2006
Unrestricted investments		
Common and preferred stock	$ 6,277,073	$ 6,166,576
Corporate bonds	829,304	966,141
Government bonds	749,470	601,406
Total Unrestricted	**7,855,847**	**7,734,123**
Temporarily restricted investments		
Common and preferred stock	549,605	601,294
Corporate bonds	332,304	326,360
Government bonds	548,812	465,694
Certificates of deposit	48,834	47,963
Total Temporarily Restricted	**1,479,555**	**1,441,311**
Total Investments	**$ 9,335,402**	**$ 9,175,434**

Total investment returns reported as income from investing activities in the statements of activities amounted to $1,178,527 and $386,303, with related expenses of $37,252 and $40,369 for the years ended June 30, 2007 and 2006, respectively.

Note 5 — Board-Designated Assets

The Foundation has set aside assets for the following purposes:

	2007	2006
Seed Grant Awards	$ 1,838,402	$ 1,615,754
Neuroscience Professorship Payable	400,000	819,698
	$ 2,238,402	**$ 2,435,452**

The assets consist of money market funds, common and preferred stock and government bonds and are reported as Board-designated investments on the statement of financial position. See Note 1 and Note 7 for additional Seed Grant disclosures.

Notes to Financial Statements (continued)

Note 6 — Neuroscience Professorship Payable

During May 2004, the Foundation entered into a gift agreement with the Division of Biological Sciences at the University of Chicago. Per the agreement, the Foundation pledged to give an aggregate amount of not less than $2,000,000 to the Division of Biological Sciences at the University of Chicago to establish and endow the Brain Research Foundation Professorship. (See Note 5 for an additional disclosure.) The pledge will be satisfied over a five-year period. The Foundation has properly recorded an expense for the entire payable, measured at the present value of the future payments.

Maturities on pledges payable as of June 30, 2007 are as follows:

Fiscal Year Ending:	
2008	$ 400,000
2009	400,000
	800,000
Less amount representing interest	(15,083)
Present Value of Professorship Payable	**$ 784,917**

A discount rate of 3.85%, derived from the July 1, 2004 treasury note interest rate, with a five-year maturity, was used to calculate the present value of the pledge.

Note 7—Temporarily Restricted Net Assets

The temporarily restricted fund represents contributions received by the Foundation where the donor has specified the purpose for which the contribution may be used plus the accumulated investment returns on the restricted contributions.

Temporarily restricted net assets as of June 30, 2007 and 2006 include the following:

	2007	2006
Fay/Frank Seed Grant Fund	$ 1,233,025	$ 1,120,328
Women's Council	389,544	343,190
Special Fund	28,558	59,074
	$ 1,651,127	**$ 1,522,592**

See Note 1 and Note 5 for additional Seed Grant disclosures.

Note 8 — Lease Commitments

The Foundation had been leasing office space from the University of Chicago at below-market rates since August 2002. The operating lease was due to expire on August 31, 2007; however, the Foundation canceled this agreement and entered into a lease for new office space effective June 1, 2007. This lease expires on May 31, 2011.

Rent expense amounted to $157,366 and $165,999 for the years ended June 30, 2007 and 2006, consisting primarily of donated rent as follows:

	2007	2006
Fair market value of donated rent	$ 137,445	$ 149,940
Rent paid to University of Chicago	16,620	16,059
Rent paid on new lease	3,301	—
Total rent expense	**$ 157,366**	**$ 165,999**

Future minimum rent payments required under the new lease as of June 30, 2007 are as follows:

Year Ending June 30:	
2008	$ 38,755
2009	39,598
2010	40,440
2011	41,283
Total Minimum Payments Required	**$ 160,076**

Note 9— 401(k) Retirement Plan

The Foundation has a 401(k) Retirement Plan (the Plan). Substantially all of the employees are eligible to make contributions at their own discretion. Upon the date an employee commences employment, they are immediately eligible to make pretax contributions to the Plan. Employees may annually contribute up to 6% of their compensation on a pretax basis up to the limits imposed by the current IRS regulations.

All employees become eligible after one year of service to receive employer-matching contributions equal to $2.67 for every one dollar an employee defers. In addition, the Foundation may elect to make discretionary contributions to the Plan as determined by the Board of Directors. Employees are 100% vested in all their accounts in the Plan.

The Foundation contributed $30,097 and $30,084 for the years ended June 30, 2007 and 2006, respectively.

Ways of Giving to the Brain Research Foundation

There are several ways in which donors can participate in the work of the Brain Research Foundation.

General Support
Unrestricted gifts are applied to the general work of the Foundation.

Restricted Gifts
Gifts designated for specific purposes established by the donor.

Stock
Gifts of stock may be given to the Brain Research Foundation.

Matching Gifts
You may be employed by one of the growing number of companies with a Matching Gift Program, so that the amount of your gift is multiplied. Please check with your Human Resources Office to see if your company offers this benefit.

Planned Giving
Long-range estate and financial planning can enable you to make a substantial contribution to the Brain Research Foundation. Some examples of planned gifts include bequests, life insurance policies, charitable remainder trusts, charitable lead trusts and charitable gift annuities.

Memorial and Honorary Gifts
You may designate the Brain Research Foundation by a donation in memory of someone, or give a gift in honor of a special person.

Donors to the Brain Research Foundation

$250,000 and above

Dr. Ralph and Marian Falk Medical Research Trust

Mr. and Mrs. Charles W. Palmer

$50,000–$249,999

Leonore and Ernest Alschuler Fund

Mr. and Mrs. Patrick J. Haynes III

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Peregrine Charities

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$5,000–$49,999

Anonymous

Anonymous

Anonymous

Anonymous

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Thomas W. Dower Foundation

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