

BRAIN RESEARCH FOUNDATION 2012-2013 ANNUAL REPORT

PROMISE

We believe in the promise of neuroscience: understanding the brain—everything that makes you, you.

Think of the countless ways you engage with the world and express yourself. Speech, memory, language, dreams. Touch, smell, sight, movement. Reasoning, judgment, learning. These few examples barely scratch the surface of all that is made possible through the wonders of the human nervous system—but not everyone enjoys its full benefits. Since 1953, the Brain Research Foundation (BRF) has been funding research by gifted scientists to better understand the intricate science that underlies neurological health. Research that is revealing the mysteries of the brain. Now, with a more diverse portfolio of grants and the ability to invite competitive proposals nationwide, the future is brighter than ever.

TABLE OF CONTENTS

2	LETTERS
4	THE PROMISE OF NEUROSCIENCE RESEARCH
6	SUPPORTING THE BEST RESEARCH
10	SEED GRANT AND AWARD STORIES
18	OUR PROMISE OF SUPPORT
24	PERSONAL STORIES
26	DONOR RECOGNITION
40	FINANCIAL SECTION
43	BRF BOARD OF TRUSTEES
44	BRF ASSOCIATE BOARD
45	SCIENTIFIC REVIEW COMMITTEE, BRF STAFF

To Our Brain Research Foundation Friends



I am so pleased to be writing this letter for the 2012-2013 Brain Research Foundation Annual Report. As 2013 marks our 60th anniversary, it is certainly a time to reflect on what the Foundation has done over the past six decades, but also, and even more importantly, it's a reason to reaffirm

our vision of funding the best neuroscience research that will advance the understanding of the brain.

The theme of this Annual Report is *promise*. Promise is a very strong word when you consider its meaning. It is a vow—a declaration made that something will definitely happen in the future. A promise is something that is not to be taken lightly or broken. When the Brain Research Foundation was established in 1953 we made a promise to promote research and knowledge concerning the human brain, and the prevention, correct diagnosis, effective treatment and cure of brain disorders. We have kept this promise for six decades and will continue to strive for its realization.

When I became the executive director of the BRF in 2002, it was because I had a passion for science and knew first-hand the extreme need for research funding. I made a promise to myself and the Foundation that I would do the best that I could at this very important job, and always lead with our mission and the intent of the donors in the forefront.

The Brain Research Foundation has a rich history, and for the past eleven years I have been part of two milestones—the BRF's 50th and 60th anniversaries. During those years there has been tremendous growth as we fund the best and the

brightest researchers. I am extremely proud to be leading this organization during this progression, as we created a new grant program (Scientific Innovations Award), developed more educational programs and expanded our research funding nationwide.

In my personal life during my time at the BRF, my family became one of the millions affected by neurological disorders. My grandmother was stricken with dementia and struggled for years until her death in 2009. And that same year, my mother was diagnosed with brain cancer. She bravely fought this terrible disease as it stole her ability to write, to read and eventually to talk. She finally succumbed to it 3 1/2 years later in 2013. She was 72.

I take comfort knowing that the research the BRF funds will play a role in helping people suffering with various neurological diseases such as the ones that afflicted my family. A miraculous outcome will not happen today...it will not happen tomorrow...and it did not happen in time to help them, but I know this organization is making a difference and I am so pleased to be part of its story.

Of course, the longevity and impact the Foundation has had, and continues to have, is only possible because of our trustees, friends and donors. Those who support our mission understand that funding neuroscience research and education will bring us closer to the day when better treatments and even cures are possible. The Foundation is proud of our accomplishments in 2012-2013 and we are excited about continuing on the promising path of discovery.

Sincerely,

A handwritten signature in black ink that reads "Terre A. Constantine". The signature is fluid and cursive, with a long horizontal flourish extending to the right.

Terre A. Constantine, Ph.D.
Executive Director

As the Brain Research Foundation commemorates its 60th anniversary milestone, this provides a great opportunity to thank all of our donors for your support over the years. Your generous gifts have created both an impressive history of achievements and a foundation for a promising future.

The history of the Brain Research Foundation is one of bold ideas and innovation. Our founders' vision was to better understand the brain, and in doing so, to make a difference across many diseases. Ultimately, many of our early trustees were driven by personal connections to individuals suffering from the debilitating effects of neurological diseases and disorders. The passion that drove our early years continues today with our current leadership, and many of you, our supporters.

Over the last 60 years the BRF has funded over \$10M of seed grant research and additional program investments toward this vision. As it becomes increasingly difficult to maintain innovative research through governmental and other public funding, organizations like ours continue to fill a gap. This Annual Report is a tribute to the founders' vision and also to our donors, who have sustained support to create a relevant and vibrant organization for the future.

As we look ahead, I speak on behalf of our trustees in saying that we have never been more excited about the BRF and the difference we are making in advancing neuroscience by funding breakthrough research. The Foundation is poised to meet an ever-growing unmet need of supporting innovative research, and does so from a position of strength.

- . Every dollar that is donated is used directly for research and educational programs.
- . Our leadership is strong both through our dynamic Board of Trustees and through our engaged Associate Board .
- . The Foundation staff combines professional management with a cost-effective infrastructure.

- . A growing and engaged list of donors and organizations validates our relevance and credibility.

Our efforts are making a difference in the scientific community through additional funding and publications, as well as scientific advancement and education.

As you review this Annual Report, we invite you to learn more about some of our most recent research programs and results. You will also see highlights of what has motivated some of our committed donors. Of additional note is the broadening reach of the Foundation in funding the best science across the country, through programs like our Scientific Innovations Award. Lastly, take note of the difference we are making in the community through our educational programs, for both the scientific and lay public.

Please join me and the rest of the organization in celebrating 60 years of accomplishment. On behalf of the trustees I offer a sincere thank you for your continued support. We are able to make a difference through the generous support that you, our donors, offer. While we celebrate our success, we are more encouraged by what lies ahead. Our theme of **promise** points to our expectation of what is yet to come. We look forward to that journey with all of you in the coming years.

Sincerely,



Nathan Hansen
President



BRF supports the best minds and the best ideas. That's where the promise of neuroscience research begins.

We've supported the best neuroscience
for 60 years.

We promise to continue.

Brain Research Foundation grants are offered on a competitive basis to accomplished neuroscientists in qualified research centers across the United States. Widely recognized as a mark of distinction, BRF grants are highly prized and well-regarded.

Candidates must be nominated. Each grant candidate is nominated by his or her academic institution and is required to submit a detailed research proposal.

Qualified proposals must pass peer review. Proposals that meet grant requirements go on to peer review, a process that determines their relative scientific merit. The peer review process is conducted by BRF's Scientific Review Committee, a multidisciplinary panel of experienced neuroscientists.

The best of the best emerge through deliberation. After the reviewers evaluate and score the proposals independently, they share their findings and reach consensus on the most deserving proposals.

In 2012 and 2013, we expanded our grants portfolio and our reach in the neuroscience community.

New grants to break new ground.

In January 2012, the Brain Research Foundation announced our first Scientific Innovations Awards (SIAs) to recognize and advance the work of world-class investigators at major U.S. research institutions. Each institution is invited to nominate one individual who is leading the way to highly innovative—possibly game-changing—responses to neurological disorders and diseases.

BRF is proud to add SIAs to our grants portfolio to encourage the work of some of our nation's most distinguished and productive neuroscientists.

BRF SCIENTIFIC INNOVATIONS AWARDS

PROVIDE \$150,000 OVER TWO YEARS TO HELP ESTABLISHED AND PRODUCTIVE NEUROSCIENCE INVESTIGATORS SUSTAIN INNOVATIVE RESEARCH PROJECTS THAT HAVE THE POTENTIAL TO YIELD SIGNIFICANT FINDINGS AND DEEPEN OUR UNDERSTANDING OF THE BRAIN.

BRF FAY/FRANK SEED GRANTS

PROVIDE \$50,000 OVER ONE YEAR TO HELP GIFTED NEUROSCIENTISTS PROGRESS THROUGH THE CRITICAL, EARLY STAGES OF RESEARCH, GIVING THEM TIME TO CONDUCT EXPERIMENTS DESIGNED TO MAKE THE CASE FOR PROJECT HYPOTHESES AND GENERATE DATA NECESSARY TO COMPETE FOR ADDITIONAL LARGE-SCALE FUNDING.

Broader reach, coast to coast.

The BRF set another milestone in fall 2012 with our first national call for **Fay/Frank Seed Grant** proposals. Initially focused on a single community of scientists at The University of Chicago, through the decades the BRF broadened our reach throughout the Midwest. Now, the Foundation receives grant proposals from investigators representing a growing number of research centers across the country.

By inviting more participation from the broader neuroscience community, the BRF will have more opportunities to identify and fund the most promising research.



Why do so many of us struggle with addiction or depression, or both? A BRF grant is helping Mitchell Roitman, Ph.D., look for answers.

“Our work has implications for disorders of reward-directed behavior like overeating that can lead to obesity—which is a huge problem in the United States—or consuming rewards that we shouldn’t, like some drugs.”

Human beings are wired to maximize what benefits us and minimize what harms us. That wiring is somehow crossed in people with diseases like obesity, addiction and depression. Can neuroscience uncover ways to help?

As he tells it, the thrill of learning drew Mitchell Roitman, Ph.D., to research. “I just loved being in the laboratory,” he said. “That’s how I learned best. I got hooked.”

Now Dr. Roitman is hooked on understanding the very reward system that sparked his commitment to research. He’s particularly interested in what drives us to seek harmful rewards, like overeating, drinking too much alcohol, or taking drugs that ultimately become addictive.

Cues and rewards: cause and effect?

Neurologically, Dr. Roitman explained, feelings of reward and reinforcement are triggered by a system that uses dopamine to help the brain send signals. When we sense cues that we associate with feelings of reward, the brain often produces a very brief spike in the level of dopamine.

“We think these dopamine spikes are important in establishing cue-reward associations,” he said. “But it’s still not known whether these are learned associations—bar sign means beer—or whether the associations actually promote behavior—bar sign means beer, bar sign activates dopamine, I go in and get a beer.”

The BRF awarded Dr. Roitman a 2012 seed grant to help him develop a new research tool to study the neurobiology of reward and aversion. The new tool combines highly advanced dopamine measurement, a particular strength of his lab at the University of Illinois at Chicago, with optigenetics.

Optigenetics is a technique that utilizes light and genetics to control the activity of neurons. Dr. Roitman is using optigenetics to install light-sensitive channels in dopamine neurons. Using brief pulses of light, dopamine neurons can be turned on or off to give scientists an opportunity to observe corresponding behaviors. For example, if dopamine neurons are off when an animal sees cues that mean sugar treat, does addictive behavior (consuming the treat) turn off as well?

What happened; what’s next?

Over the year of BRF funding, Dr. Roitman’s team proved the tool works. Specifically, they were able to cause brief pauses in dopamine signaling in an important region of the brain called the nucleus accumbens.

Next, the team will look for causal relationships in animals between brief changes in dopamine concentration and reward and aversion. Using preliminary data from the BRF study, Dr. Roitman has applied for additional funding from the National Institutes of Health to further his research.



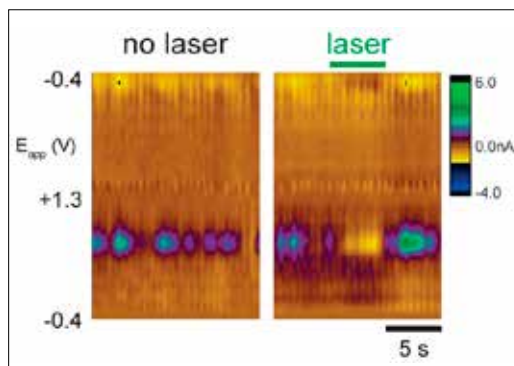
Mitchell Roitman, Ph.D., Department of Psychology, University of Illinois at Chicago

Future promise: decoding addiction and depression.

Another aspect of his research relates to loss of reward. Often, Dr. Roitman said, depressed individuals lose the ability to experience pleasure. This is called anhedonia, and may be linked to the absence of dopamine spikes. Using the new research tool, Dr. Roitman will attempt to establish a causal relationship between turning dopamine neurons off and this negative affect.

The opportunity?

Developing insights that will contribute to a deeper understanding of the neurobiology that underlies depression. This outcome, combined with the possibility of discovering a path to overcoming addiction, offers truly promising results.



Phasic dopamine ‘spikes’ evoked by drugs are suppressed by light administered to the ventral tegmental area—the site of dopamine cell bodies. Genetically engineered rats (courtesy of Drs. Karl Deisseroth and Ilana Witten) were made to express light-sensitive inhibitory channels. *Left:* Dopamine ‘spikes’—appearing as green ‘blobs’ occur often after administration of cocaine. *Right:* light emission suppresses dopamine ‘spikes’ but only while light is being emitted.

Current treatments for obsessive-compulsive disorder (OCD) can leave behind as many people as they help. With support from a BRF seed grant, Stephanie C. Dulawa, Ph.D., hopes to change that.

“This disorder is desperately in need of novel treatments. With more insight into the mechanisms of OCD, we may find new therapy ideas, or identify compounds or targets that would work better.”



Stephanie C. Dulawa, Ph.D., Department of Psychiatry and Behavioral Neuroscience, The University of Chicago

If we can discover how the genes linked to obsessive-compulsive disorder function at the molecular level, we can help more people reclaim their lives.

Human geneticists have been trying for years to identify genes responsible for OCD. Now they have. With a 2013 BRF seed grant, Stephanie C. Dulawa, Ph.D., is leading research that may reveal how the top two genes linked to OCD contribute to its development.

With the data that emerges, she hopes to convince the National Institutes of Health (NIH) of the potential for further research seeking new insights and more effective treatments. As it is, current therapies leave many unrelieved of OCD's haunting symptoms.

OCD: chronic and relentless.

OCD is a common brain disorder that is debilitating and, genetically, highly complex. People with OCD struggle daily with compulsions and obsessions such as unwanted thoughts or images and repetitive behaviors. A simple task that most people accomplish in minutes, like combing one's hair while looking into a mirror, for someone with OCD can turn into an inescapable ritual lasting hours.

Available treatments not always effective.

Some people successfully manage OCD through a type of cognitive behavioral therapy called exposure and response prevention, but not everyone can tolerate it. "Many patients experience high levels of distress with this therapy. But some are able to endure the process and achieve good results," Dr. Dulawa said.

Medication is often prescribed to treat OCD. Dr. Dulawa explained that a type of antidepressant called serotonin reuptake inhibitors seem to

work best, but are effective for roughly half of patients and, at best, reduce symptoms about 50%. "We really want to do better than that."

Doing better by understanding gene function.

Using techniques that Dr. Dulawa developed previously, the research team will observe two groups of mice: one that lacks the BTBD3 gene and one that lacks DLGAP1. They will look for phenotypes: any observable evidence that has relevance to OCD. For example, the team will watch for repetitive behavior, and neuron activity in specific brain regions that are known to be dysfunctional in persons with OCD.

Though the study is still in progress, Dr. Dulawa said the team has observed a number of behavioral differences that appear to be significant, and she feels the study offers real promise. All of which makes her hopeful of being able to write a compelling proposal for large-scale funding from the NIH to pursue the study further.

BRF funding: "absolutely essential."

Meanwhile, Dr. Dulawa emphasized, her 2013 BRF grant is all the more important. Despite her strong track record, including prior support from the National Institute of Mental Health and a 2007 BRF seed grant, and her status as a principle investigator (PI) at a major university, Dr. Dulawa finds it increasingly difficult to stay funded. This difficulty—experienced by so many researchers—is one of the reasons BRF remains firmly committed to funding innovative research.

"Suddenly, a lot of funding has dried up. It's extremely stressful," she said. "Most PIs today spend a ton of their time just writing grant proposals. It takes away from the science, unfortunately. That's why support from the BRF is absolutely essential."



Locomotor paths taken by individual mice in an open field are shown (over 30 min.). The paths taken by mice provide one means to study repetitive behaviors observed in certain neuropsychiatric disorders including OCD. The path taken by a control mouse (top) shows both straight and circumscribed movements during normal exploration. The path taken by a mouse receiving a drug challenge that worsens symptoms in OCD patients is highly repetitive and is characterized by straight movements.

It's heartbreaking to see people we care for struggle with dementia and not be able to help them. With support from the BRF, Aimee W. Kao, M.D., Ph.D., is on the path to new treatment options.

“Without the BRF grant, our team simply wouldn't be able to do this work. If we succeed, it would be a big advance in the sense that we will have a model very relevant to human disease that we can test.”

Subnormal levels of a protein called progranulin may cause microglia to attack, rather than protect cells responsible for brain function. Could faulty genes be reprogrammed to override mutations that cause this problem?

Alzheimer's, Huntington's and other neurodegenerative disorders behave in very different ways person to person. Likewise, symptoms for these conditions can vary widely making it difficult for physicians to reach a proper diagnosis and know what's best for each patient. The reasons why—and the path to effective treatments—appear to be hidden deep inside the workings of our brains, at the molecular level.

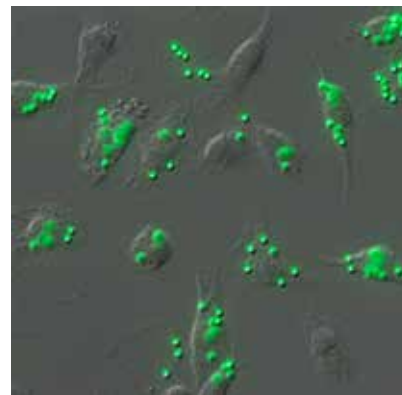
Since 2011, from her lab at the University of California, San Francisco, Aimee W. Kao, M.D., Ph.D., has been working to understand what contributes to frontotemporal lobar degeneration, a disease that robs people of functionality in the areas of the brain that make us most human.

As Dr. Kao described, the frontal lobes of the brain separate us from other animals on this planet. They give us our personality; our motivation; our ability to plan and multi-task, to appreciate art and music, to feel empathy and love. The temporal lobes also play a crucial role as the seat of language and our ability to navigate the world.

“Dementia affects so much of who we are,” she remarked, “and yet we don't know much about it.”

Dr. Kao's research team is focused on how a particular protein called progranulin contributes to loss of function in these key parts of the brain.

“We go through life every day managing a variety of environmental stressors like infection or bumps to the head,” Dr. Kao said. “As life goes on, the effects of stress can accumulate as changes in our cells and bodies. We postulate that over time, people who don't have enough progranulin develop aberrant responses to stress. This may be due to overly aggressive microglia—cells in the brain that become confused and start attacking neurons that they normally would protect.”



Macrophages lacking the progranulin gene avidly engulf fluorescent beads.



Aimee W. Kao, M.D., Ph.D., Department of Neurology, University of California, San Francisco

Climbing another rung.

With the help of a 2013 BRF Seed Grant, Dr. Kao's team will apply findings from prior work with plants and invertebrate and vertebrate animals to an examination of induced pluripotent stem cells, or iPSCs.

iPSCs are adult human cells that have been genetically reprogrammed to mimic characteristics of embryonic stem cells. Using advanced microscopy, the team will examine the iPSCs to understand in minute detail the effect of progranulin on interactions between neurons and microglia.

Millions of potential beneficiaries.

With enough knowledge, one possibility is finding a way to override mutations and prevent neurons from degenerating and dying.

In addition to a better understanding of frontotemporal lobar degeneration, Dr. Kao's research has the potential for changing the way scientists and physicians think about and treat Alzheimer's and Parkinson's disease as well as different forms of cancer. Her project is off to a very promising start.

Could a child with epilepsy be given a pill containing an all-but-invisible therapeutic device programmed to travel to specific cells in his brain and, once there, be “pinged” into action to repair the damaged cells? Quite possibly, says Brian Litt, M.D.

“We believe that treatments with nanodevices have the potential to dramatically improve treatment of some of the most common neurological and psychiatric illnesses by addressing the underlying causes.”



Brian Litt, M.D., Department of Neurology, University of Pennsylvania

The answer to epilepsy, depression and many other common brain disorders may be treatment at the molecular level, *inside* the specific cells responsible, through a nanodevice smaller than the period at the end of this sentence.

Epilepsy and many other common brain disorders are caused by localized problems in brain networks. And yet, most treatments today work at a macro level.

For example, a disease caused by a select group of neuronal cells in one area of the brain may routinely be treated with medication that floods every tissue in the body and can cause any number of side effects, some serious.

Some implantable treatment technologies have been developed, but they also have side effects and do little to slow the progress of the disease. This is because they are relatively non-specific and fail to focus on what scientists call disease mechanisms.

Getting to the root of the problem.

Neuroscientists now see a possibility for treating epilepsy and other neurological disorders where (and only where) the problems occur, without disturbing other areas in the body. Because treatments would be precisely targeted, to a specific location in the brain and inside certain cells, there is good reason to believe they would be far more effective.

Using nanodevices to repair damaged cells.

With support from a 2013 BRF Scientific Innovations Award, Brian Litt, M.D., is pursuing the treatment of epilepsy at the molecular level. Dr. Litt is using nanotechnology to develop devices that are smaller than you can see under a conventional microscope, and are programmed to travel to damaged cells and deliver therapeutic agents to repair them.

Mind-boggling possibilities.

Such a device could be coated with biologic particles to keep the immune system from attacking it, and with antibodies or surface protein receptors to guide the device to its destination. Once the target is reached, the device could be “pinged” to release the therapeutic agents it has carried to the damaged cells.

After making the repair, depending on how the device is fabricated it might dissolve; or be engulfed and consumed by micro-organisms preloaded on the device and preprogrammed to clean up; or be excreted.

Implications for multiple diseases.

At this stage, Dr. Litt’s team is concentrating on the potential for nanodevices designed to treat epilepsy. If successful, he believes much of what his research team learns will be applicable to treatments for depression, movement disorders like Parkinson’s and Huntington’s disease, schizophrenia, and possibly even some types of brain tumors.

The promise of neuroscience continues to amaze.

Our promise of support spans
innovative research, education and
prevention for better brain health.

Our promise to support research.

Every year, the Brain Research Foundation receives numerous grant proposals from gifted neuroscientists. Guided by the high standards set by BRF's Scientific Review Committee, the highest-scoring proposals are peer-reviewed by committee members. Those that reflect the best science and offer the greatest potential for producing breakthrough results are given financial support.

2012 Fay/Frank Seed Grant Winners

Depression

Dane Chetkovich, M.D., Ph.D.
Department of Neurology, Northwestern University
Novel HCN channel inhibitors for treatment of depression

Learning and memory

David Freedman, Ph.D.
Department of Neurobiology, The University of Chicago
Cortical circuit mechanisms for visual categorization and category learning

Associate Board Grant

Parkinson's disease

Yong-Chao Ma, Ph.D.
Department of Pediatrics, Northwestern University/Children's Hospital Research Center
Regulation of dopaminergic neuron fate specification by neurogenin 2

Age-related macular degeneration (AMD)

Agnella Matic, Ph.D.
Department of Otolaryngology, Northwestern University
Characterization of infrared neural stimulation in the retina

Visual processing

Leslie Osborne, Ph.D.
Department of Neurobiology, The University of Chicago
Neural mechanisms of efficient coding in the primate visual cortex

Hormonal effects on neurons

Raphael Pinaud, Ph.D.
Department of Neurobiology, Northwestern University
Modulation of visual cortical processing by brain-generated estrogen

Neural stem cells

Murali Prakriya, Ph.D.
Department of Molecular Pharmacology and Biological Chemistry, Northwestern University
CRAC channel function and calcium dynamics in neural stem cells.

Addiction and depression

Mitchell Roitman, Ph.D.
Department of Psychology, University of Illinois at Chicago
Optogenetic induction of phasic pauses in dopamine signaling in awake, behaving rats

Neuronal activity

Geoffrey Swanson, Ph.D.
Department of Molecular Pharmacology and Biological Chemistry, Northwestern University
Galectin modulation of excitatory transmission and neuronal function

Schizophrenia

Kuei Tseng, M.D., Ph.D.
Department of Cellular and Molecular Pharmacology, Rosalind Franklin University
CB1 cannabinoid receptor activation during adolescence impairs maturation of prefrontal GABAergic circuits

2013 Fay/Frank Seed Grant Winners

Autism, epilepsy, schizophrenia

Brian Y. Chow, Ph.D.
Department of Bioengineering, University of Pennsylvania
Non-invasive optogenetic engineering of cortical dynamics

Obsessive-compulsive disorder (OCD)

Stephanie C. Dulawa, Ph.D.
Department of Psychiatry and Behavioral Neuroscience, The University of Chicago
Functional characterization of genes associated with obsessive-compulsive disorder using mouse models

Huntington's disease, OCD, Parkinson's disease

Xin Jin, Ph.D.
Department of Molecular Neurobiology Laboratory, The Salk Institute for Biological Studies
Optogenetic dissection of the striatal subcircuits during action sequence learning

Autism, schizophrenia

Pascal S. Kaeser, M.D.
Department of Neurobiology, Harvard University
Activity-induced adaptations in the molecular machines that control neurotransmitter release

Alzheimer's disease

Aimee W. Kao, M.D., Ph.D.
Department of Neurology, University of California, San Francisco
Progranulin deficiency and neuronal-microglial interactions in the pathogenesis of neurodegenerative disease

Alzheimer's disease, autism, schizophrenia

Matthew J. Kennedy, Ph.D.
Department of Pharmacology, University of Colorado
Controlling synaptic function with light

Autism

Yingxi Lin, Ph.D.
Department of Brain and Cognitive Sciences, Massachusetts Institute of Technology
A systems biology approach to identify transcription regulatory networks in healthy and diseased neurons

Itch perception

Qin Liu, Ph.D.
Department of Anesthesiology, Washington University in St. Louis
The molecular and neural basis of itch sensation

Schizophrenia

Axel Nimmerjahn, Ph.D.
Department of Biophotonics, The Salk Institute for Biological Studies
The role of astrocyte-neuron communication in normal brain function and mental disorder

Autism, intellectual disability

Debra L. Silver, Ph.D.
Department of Molecular Genetics and Microbiology, Duke University
Distal mRNA localization and translation in neural stem cells during mammalian cortical development

Associate Board Grant

Alzheimer's disease

Yongli Zhang, Ph.D.
Department of Cell Biology, Yale University
Structures, stabilities and formation kinetics of amyloid beta precursors and oligomers

Neural plasticity, neurodevelopment

Zhaolan Zhou, Ph.D.
Department of Genetics, University of Pennsylvania
Epigenetic control of experience-dependent neural plasticity

2012-13 Scientific Innovations Award Winners

Cortical function and behavior

W. Martin Usrey, Ph.D.
Center for Neuroscience, University of California, Davis
Optogenetics in the behaving primate: using light to study cortical function *2012 Scientific Innovations Award, \$150,000*

Neuron replacement: improved cognitive function

Jean M. Hébert, Ph.D.
Department of Neuroscience, Albert Einstein College of Medicine
How receptive is the adult neocortex to incorporating new projection neurons? *2013 Scientific Innovations Award, \$150,000*

Targeted therapy with Nanodevices

Brian Litt, M.D.
Department of Neurology, University of Pennsylvania
Nanodevices to treat neurological diseases *2013 Scientific Innovations Award, \$150,000*

Seeing the promise of neuroscience research through other lenses.

Brain research is at a unique point in its evolution. The opportunities to do highly impactful research have never been greater, but funding is shrinking. As a result, scientists are being asked to work with a fraction of the resources they require.

Unless more resources are offered—by companies, foundations, private research institutions, the government and individuals—the pace of discovery will slow, and so will our progress toward solutions that will help address brain disorders and diseases.

By building public awareness through educational initiatives, the BRF is calling attention to the need for increased funding for neuroscience.

PROTECTING CHILDREN FROM CONCUSSIVE EVENTS.

CHILDREN HAVE ALWAYS BEEN AN IMPORTANT CONSTITUENCY FOR THE BRF. THIS IS PARTICULARLY TRUE FOR OUR ASSOCIATE BOARD OF YOUNG PROFESSIONALS WHO ARE COMMITTED TO MAKING A POSITIVE DIFFERENCE IN THE LIVES OF CHILDREN WITH BRAIN DISORDERS.

ONE FOCUS OF BRF EFFORTS TO PROTECT CHILDREN'S BRAIN HEALTH IS PREVENTING SPORTS-RELATED INJURIES IN YOUTH, ESPECIALLY CONCUSSIONS. THIS FOCUS IS VALIDATED BY DATA GATHERED BY THE CENTERS FOR DISEASE CONTROL AND PREVENTION, AND BY QUALITATIVE RESEARCH WE CONDUCTED LATE IN 2010 THAT POINTED TO THE NEED TO ENGAGE COACHES, TRAINERS AND PHYSICIANS AS WELL AS PARENTS.

SCHOOL ATHLETIC PROFESSIONALS WITH A \$50,000 BRF GRANT, SPORTS LEGACY INSTITUTE IS EQUIPPING COACHES AND TRAINERS WITH MORE INFORMATION ABOUT THE RISKS AND DANGERS OF CONCUSSIONS AND BETTER WAYS TO RESPOND AS STUDENTS SUFFER AND RECOVER FROM HEAD INJURIES.

PEDIATRICIANS A \$30,000 BRF GRANT IS FUNDING RESEARCH TO IDENTIFY VALID AND RELIABLE CLINICAL TOOLS THAT PEDIATRICIANS CAN USE TO MEASURE CONCUSSIVE RECOVERY IN YOUNG CHILDREN.

PARENTS AND THE PUBLIC AT LARGE IN 2013, THE BRF HELPED SPONSOR BICYCLING EVENTS HOSTED BY PRAIRIE STATE CYCLING SERIES IN EIGHT LOCATIONS THROUGHOUT METROPOLITAN CHICAGO. THOUSANDS TURNED OUT FOR BRAIN-HEALTHY EXERCISE AND LINED UP TO LEARN ABOUT THE CRUCIAL ROLE OF HELMETS IN BIKE SAFETY. PARTICIPANTS WHO SHARED STORIES ABOUT BRAIN HEALTH ISSUES PROVED A WELL-ESTABLISHED BRF AXIOM: EVERY LIFE IS TOUCHED BY NEUROLOGICAL PROBLEMS (MORE ABOUT THIS ON PAGES 24-25).

A SPOTLIGHT ON AUTISM.

TWO YEARS AGO, FOUNDATION INVESTIGATORS YOUNG SHIN KIM, M.D., PH.D., M.P.H, AND BENNETT L. LEVENTHAL, M.D., LED A FIVE-YEAR SCREENING STUDY THAT YIELDED SURPRISING RESULTS. FUNDED PRIMARILY BY THE BRF, THE STUDY FOUND AUTISM IN CHILDREN AGES 7-12 TO BE ALMOST THREE TIMES HIGHER THAN PREVIOUSLY THOUGHT.

THIS RESULT EMPHASIZED THE NEED TO INCREASE RESEARCH, ESPECIALLY EARLY-STAGE INVESTIGATIONS, AND LED US TO CHOOSE AUTISM AS THE FOCUS FOR THE LECTURE SEGMENT OF BRF'S 13TH ANNUAL NEUROSCIENCE DAY IN JANUARY 2013.

NEUROSCIENCE DAY.

EACH YEAR FOR NEUROSCIENCE DAY, GRADUATE AND POSTDOCTORAL STUDENTS PREPARE AND PRESENT INTERACTIVE SCIENTIFIC POSTERS THAT DOCUMENT THEIR RESEARCH IN THEIR AREAS OF STUDY. PROMINENT RESEARCHERS FROM CHICAGO-AREA INSTITUTIONS JUDGE THE PRESENTATIONS AND HONOR THE TOP TWO PRESENTERS FROM EACH GROUP WITH CASH AWARDS. AFTER THE POSTER COMPETITION, PARTICIPANTS ATTEND A FULL AFTERNOON OF LECTURES.

Slowing cognitive decline—the time is now.

As members of the baby-boom generation move into their 60s, we desperately need more education—and more research—on ways to slow cognitive decline.

Early-stage research has established a link between physical exercise and more stable mental acuity in middle-aged and older adults. As yet though, we are far from having a plan of action. Definitive research is needed to understand how cognitive abilities begin to stall, and how to intervene.

Raising public awareness, encouraging regular exercise beginning in early adulthood, and a stepping up of investment in research to expand knowledge of cognitive decline promise to improve the quality of all our lives. We have an opportunity to redefine aging.

WHAT WE KNOW ABOUT SLOWING COGNITIVE DECLINE ONLY SCRATCHES THE SURFACE. HERE ARE A FEW EXAMPLES:

- . AEROBIC EXERCISE INCREASES BRAIN VOLUME IN PEOPLE AGED 60-79.
- . EXERCISING REGULARLY IN OLD AGE MAY BETTER PROTECT AGAINST BRAIN SHRINKAGE THAN ENGAGING IN MENTAL OR SOCIAL ACTIVITIES.
- . DAILY PHYSICAL EXERCISE, INCLUDING ACTIVITIES LIKE HOUSE CLEANING, MAY REDUCE THE RISK OF ALZHEIMER'S DISEASE, EVEN IN PEOPLE OVER AGE 80.

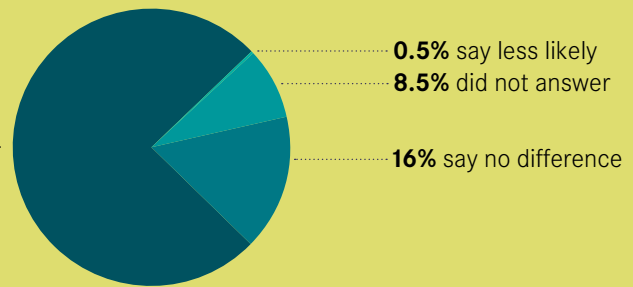
Shining a light on cognitive decline and exercise.

The cycling event we helped sponsor throughout metropolitan Chicago in 2013 offered a platform to begin a public conversation on slowing cognitive decline by increasing exercise levels. The BRF surveyed 225 people on activity levels and habits, and personal concerns about losing cognitive functioning. The concept for the survey came from our discussions with scientists and the identification of a gap in research on this subject.

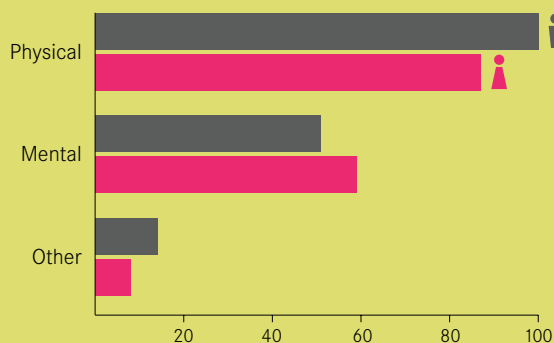
Results from the survey indicate a prime opportunity exists to both fill the research void and motivate people to improve their physical health through exercise, so that they are better able to sustain mental acuity.

HOW DOES KNOWING THAT EXERCISE SLOWS MENTAL DECLINE IMPACT HOW MUCH YOU EXERCISE?

75% more likely to exercise if it slows mental decline

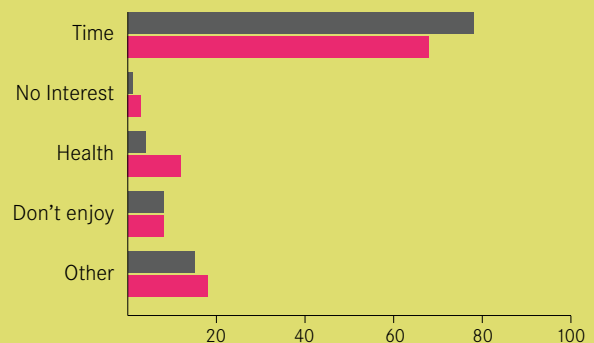


WHY DO YOU EXERCISE?



82% exercise to maintain physical health
48% exercise to maintain mental health

WHY DON'T YOU EXERCISE?



64% indicate time prevents them from exercising

DO YOU KNOW SOMEONE WHO HAS SUFFERED COGNITIVE DECLINE SUCH AS DEMENTIA OR ALZHEIMER'S?



63% have known someone who has suffered cognitive decline such as dementia or Alzheimer's

ARE YOU PERSONALLY CONCERNED ABOUT DEVELOPING COGNITIVE DECLINE SUCH AS DEMENTIA OR ALZHEIMER'S?



54% are concerned about the possibility of developing dementia or Alzheimer's

A mother with dementia. A friend with ALS. A child with autism. We all know someone who has a story.

When you think about it, it's astonishing—and disheartening—to realize that we all have stories about someone whose life has been impacted by a neurological disorder. We recently sponsored a cycling event that gave us an opportunity to hear some of those stories.

These are just a few.



↑ Graphic designer Vernon Lockhart insisted that a friend “mess up her hair” and wear a cycling helmet. A fall caused a deep gash in her helmet—but her hair was just fine.



↑ When she was a small child, Stephanie Weber experienced a devastating brain injury caused by 2,000 pounds of sheet rock falling on her. Because of neurological advances that helped repair her skull, restore most of her hearing and eliminate double vision, Stephanie today leads the active life of a young adult.



↑ Esther Gutierrez watched her grandmother, and is now watching her mother, struggle with the devastating effects of dementia. She wonders if there is a genetic component and if a similar struggle is in her future as well.



↑ A competitive cyclist from New Zealand, Haley Criddens relies on helmets to protect her from crashes. And she questions the sanity of motorcyclists who ride without them.



← Walt Sloan is watching the slow progression of his mother-in-law's dementia and advocates increased funding for brain research. “We have to act today—in order to ensure that we have answers tomorrow.”



← Mae Rosenthal, a physical therapist, works closely with Parkinson's patients struggling with the disease's devastating effects. For many of these patients, loss of mobility and speech are typically accompanied by depression and anxiety.



← Catherine Majeske, a retired school teacher, is slowly losing a friend to brain cancer. A once-vital and engaged woman, “she's there, but it's not her.”

Thank you for helping us achieve so much these past 60 years.

Memory loss. Disorientation. Impaired speech. Inability to reason. Inappropriate behavior. Any one of the symptoms associated with dementia would be difficult to live with. The experience of watching a person lose his or her ability to think and relate to others as dementia symptoms stack up and intensify is devastating. Watching family and friends give up on that person is excruciating.

Cindy Macfarland described her father as a classic people person, a man who loved spending time with his family and many circles of friends, and interacting with his clients. But then memory problems set in and all that changed.

Cherish people you care for, no matter what.

Little by little, Cindy's father was less and less able to be himself. In large groups of people he would withdraw and not engage—something that was so unlike his typical enthusiasm for people. This led to fewer social interactions.

The gift of time.

“At dinners and parties,” Cindy recalled, “My mom and I would always sit myself next to my dad and take whatever time he needed in a conversation. When someone has dementia or memory loss, you need to be patient and wait for him.”

Sadly, Cindy, like so many others, has had an opportunity to not only help her father through his dementia, but also other family members and friends. Moreover, she feels confident that more and more people will share her experience as we continue to live longer.

What we can do...now.

More than advice, Cindy has two urgent invitations. One, stay connected to people who have dementia to help them through the journey. Two, support neuroscience research through the BRF.

“I give every year because we have to find our way to a cure, or at least something that stalls the process,” she said. “I understand that the BRF was one of the first organizations to believe in the potential for neurological research. I know they've convinced me through their progress and determination.”

“We need to ensure that the BRF can continue to deliver on the promise it made 60 years ago to fund leading-edge neuroscience research.”

DEMENTIA IS A CONDITION CHARACTERIZED BY IMPAIRED THINKING AND SOCIAL SKILLS. IT STEMS FROM DAMAGED NERVE CELLS IN THE BRAIN AND AFFECTS PEOPLE DIFFERENTLY, DEPENDING ON WHICH AREA OF THE BRAIN IS AFFECTED. SOME DEMENTIA IS REVERSIBLE WITH TREATMENT. SOME DEMENTIA IS PROGRESSIVE, SUCH AS THAT ASSOCIATED WITH ALZHEIMER'S DISEASE, AND SOME IS LINKED TO COMPLEX INJURY OR DISEASE, SUCH AS TRAUMATIC BRAIN INJURY, PARKINSON'S AND HUNTINGTON'S DISEASE.

*“On top of stealing memories, dementia takes a person’s dignity.
We need to help BRF fund more research.”*



BRF donor Cynthia J. Macfarland



BRF donors Marc and Barbara Posner

CREUTZFELDT-JAKOB DISEASE (CJD) IS A FAST-MOVING BRAIN DISORDER THAT LEADS TO DEMENTIA AND DEATH. IT IS ASSOCIATED WITH ABNORMALITIES IN PRION, A NORMALLY HARMLESS BRAIN PROTEIN. IN MOST CASES, CJD DEVELOPS SPONTANEOUSLY, FOR NO APPARENT REASON. A SMALL NUMBER OF CASES ARE INHERITED, CAUSED BY A GENETIC MUTATION, OR ARE DUE TO CONTAMINATION.

“We decided to support BRF because it supports the best neurological research.”

Barbara and Marc Posner were introduced to neuroscience research through a crisis. Without warning, Barbara’s father, John McGrath, a healthy and vigorous 63-year-old banking executive, began behaving strangely. Eating with his left hand, rather than his right. Stuffing clothes behind his dresser, rather than placing them inside the drawers.

Soon, he was diagnosed with Creutzfeldt-Jakob disease (CJD). Unfortunately there was, and still is, no cure for CJD. Six weeks after diagnosis, Barbara’s father died.

Gifts of gratitude.

This experience heightened Marc and Barbara’s interest in neuroscience research and propelled them to contact Dr. Raymond Roos, under whose care Barbara’s father had been during his illness. They learned of the work that he and Dr. James Mastrianni were doing at The University of Chicago, and the grants the doctors had received from the BRF. Through periodic gifts to the Foundation, the Posners began supporting their research.

3 family households, 2 generations, 1 goal.

The periodic gifts led to an expanded five-year financial commitment to the Foundation that also included contributions by Marc’s parents,

now deceased, and Barbara’s mother, Gloria, and later to a significant legacy gift by Marc and Barbara. This gift embodies their shared experiences and values and serves one goal: realizing the promise of neuroscience research.

Giving to make a lasting difference.

Recently, Barbara and Marc decided to focus their philanthropy with the Foundation and take a long-term approach. Through a substantial estate gift designated to prion research, the Posners will make a bigger impact by increasing their support to expand understanding of not only CJD, but many neurological disorders.

“While my Dad died of CJD (so rare that it strikes one in a million),” Barbara said, “prion research stands to impact Parkinson’s, ALS, Alzheimer’s and other neurodegenerative diseases.”

The same is true for other research that the BRF funds. Any breakthroughs from one study will benefit a far greater number of people.

Contributing to the BRF began as a very personal decision for Barbara and Marc and, they said, still represents a way to honor Barbara’s father. But, as she explained, knowing their gifts will help create a more promising future for generations to come is its own gift.

JAMES A. MASTRIANNI, M.D., PH.D., HAS RECEIVED SIX BRF SEED GRANTS TO ADVANCE HIS RESEARCH. IN ADDITION, DR. MASTRIANNI WAS ONE OF TWO NEUROSCIENTISTS INVITED BY BRF TO ADDRESS 2012 DISCOVERY DINNER ATTENDEES ON THE RISK OF COGNITIVE IMPAIRMENT.

RAYMOND P. ROOS, M.D., HAS RECEIVED ELEVEN BRF SEED GRANTS TO ADVANCE HIS RESEARCH ON NEURODEGENERATIVE DISEASES.



BRF donor Diana Healy

“I find comfort and hope in the fact that even though it’s too late to help some of those I love, I may still help others.”

When Diana Healy heard her husband’s diagnosis, she was overcome with shock: ALS, often called Lou Gehrig’s disease, is a devastating progressive neurodegenerative illness that has no cure.

She said she never felt so hopeless.

“My immediate thought was: What do we do to fight this? Then I learned that there wasn’t much to do, there was not one medication available that could give us even a little bit of hope. That’s when I really became scared.”

“I was stunned to be in this situation again. This is not the first time in my life that I’ve had to deal with a loved one struggling with a disease of the brain or nervous system. In fact, I’ve had five family members afflicted with various illnesses, including two who suffered from depression and ultimately took their own life. My younger daughter is mentally handicapped and my older daughter’s husband is living with brain cancer. I knew I had to act.”

Knowledge: the key to hope.

The months following the ALS diagnosis went quickly and Diana’s husband succumbed to his illness in less than a year. Once he passed, she

was determined to find out as much as she could about current neuroscience research. In January 2013 she discovered the BRF and was profoundly impacted by our mission. Diana decided that BRF would be the best custodian for her philanthropy and made her first gift that month. She said she is committed to supporting the Foundation as long as she can.

So many stand to benefit.

“I find comfort and hope in the fact that even though it’s too late to help some of those I love, I may still help others. My donation, regardless of size, may actually be a part of a cure.”

Why BRF?

“In my opinion,” Diana said, “the Brain Research Foundation is the organization doing the most to support research. Their approach makes sense to me: supporting different research topics and scientists at different institutions who come at the problems from different angles. And the fact that 100% of my donation goes directly to scientific studies is something no other organization I researched was able to promise.”

We promise we’ll do everything possible to earn the confidence that Diana has placed in us.

AMYOTROPHIC LATERAL SCLEROSIS, OR MOTOR NEURON DISEASE, IS A FATAL NEUROLOGICAL DISEASE THAT CAUSES MUSCLES TO WEAKEN AND, ULTIMATELY, BECOME UNCONTROLLABLE. OFTEN, IT BEGINS WITH SLURRED SPEECH OR TWITCHING AND WEAKNESS IN ARM OR LEG MUSCLES. AS THE DISEASE PROGRESSES, IT AFFECTS THE ABILITY TO CONTROL MUSCLES NEEDED FOR MOVEMENT, SPEECH, EATING AND BREATHING.

DEPRESSION IS A SERIOUS ILLNESS THAT IS COMMON, CHRONIC AND USUALLY TREATABLE, AND AFFECTS EMOTIONS AND BEHAVIOR. DEPENDING ON TYPE AND SEVERITY, DEPRESSION CAN BE DISABLING AND MAY BE TRIGGERED OR ACCOMPANIED BY OTHER ILLNESSES. MANY PEOPLE WITH DEPRESSION ARE HELPED BY MEDICATION, PSYCHOTHERAPY AND OTHER METHODS, THOUGH SOME NEVER SEEK TREATMENT.

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Letter from the Treasurer

The Brain Research Foundation continues to fund ground-breaking research thanks to the increasingly generous contributions of our donors and our superior investment performance. During the last two years, more than 100% of our annual donor contributions have been used to fund research and educational programs in neuroscience. Our recently completed strategic plan established the objective of 100% of annual donations being used to fund research and we are proud of having achieved these results in each of the last three years.

Strategically, the BRF decided to extend our reach for our research grant programs to worthy projects throughout the United States. This allowed us to back exciting research projects throughout the country that can make a difference. We funded about \$2.4 million of projects in the last two years. That amount exceeded our contributions by \$350,000, thus meeting our objective of investing at least 100% of our annual support in worthy neuroscience programs. We are proud of our ability to be good stewards of our donor dollars.

We ended the two-year period since our last report with over \$15 million of net assets which positions us well for continuing our aggressive program of investments in worthy research projects. We have included a summary of our income and major expenses and a condensed balance sheet for fiscal years 2012 and 2013. We encourage you to review our audited financial statements on our website or by contacting the BRF office.

The Board of Trustees and staff continue to work hard to sustain your support to fulfill our mission. As we start our 61st year, we look forward to building our donor base and funding more researchers who are focused on improving life through innovative neuroscience research.

Sincerely,

A handwritten signature in black ink that reads "David H. Fishburn". The signature is written in a cursive, flowing style.

David H. Fishburn
Treasurer

Financial Statements

Statement of Activities and Changes in Net Assets

Highlights of Income Statement year ended June 30, 2012 and 2013

	2013		2012	
Beginning Net Assets	\$	13,918,410	\$	14,164,726
Contributions		1,161,346		898,740
Interest and dividends		553,056		448,437
Net realized gain (loss) on investments		653,590		335,210
Change in net unrealized gain on investments		408,708		(371,819)
Total	\$	16,695,110	\$	15,475,294
Expenses				
Program services	\$	1,266,474	\$	1,150,873
Supporting services		427,365		406,011
Total		1,693,839		1,556,884
Total net assets	\$	15,001,271	\$	13,918,410

Financial Statements (continued)

Statement of Financial Position

As of June 30, 2012 and 2013

Assets	2013		2012	
Cash	\$	11,684	\$	13,658
Current prepaid expenses and deposits		25,120		14,908
Investments		15,156,190		13,934,809
Contributions receivable		-		25,000
Property and Equipment - Net		9,204		15,224
Other assets		5,200		5,200
Total	\$	15,207,398	\$	14,008,799
Liabilities and Net Assets	2013		2012	
Liabilities				
Accounts Payable and Accrued Expenses	\$	206,127	\$	90,389
Total liabilities	\$	206,127	\$	90,389
Net Assets				
Unrestricted	\$	9,282,257	\$	8,732,706
Unrestricted - Board-designated		3,530,040		3,229,632
Temporarily restricted		688,974		456,072
Permanently restricted		1,500,000		1,500,000
Total net assets	\$	15,001,271	\$	13,918,410
Total	\$	15,207,398	\$	14,008,799

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