Studying Cellular Motors with Fruit Flies in the Gelfand Lab

“Curiosity drives basic science,” says Vladimir Gelfand, PhD, Leslie B. Arey Professor of Cell, Molecular, and Anatomical Sciences. Four years ago, curiosity led Gelfand, then a classical cell biologist, to move from studying mammalian cells to working with fruit flies and fly neuron cell cultures to better understand how cells function and how molecules move within them.

“We realized we needed a lot of molecular tools to look into cellular interactions,” he says. “When we looked at organisms, flies were a reasonable compromise. Once we started working with fly cells, we started to have interesting findings, and we wanted to see how mechanisms play out in the fly as a whole organism.”

Gelfand’s research focuses on how components inside the cell, such as organelles and vesicles, are distributed. This distribution depends on the function of motor proteins, molecules that are able to move along cellular highways known as microtubules. More than 40 different motors move proteins along microtubules, and scientists understand very little about how those motors recognize their cargo and how their activities are regulated.

“My research is relevant to many aspects of diseases,” Gelfand says. “Familial neurological diseases like amyotrophic lateral sclerosis (ALS), for instance, are associated with defects in motor proteins. In neurons, motor proteins have to deliver cargo from the cell body all the way to the ends of the synapses. Sometimes the cargo has to travel over a meter from...”

Continued on pg. 2
While Gelfand uses a number of models to study motor proteins and microtubules, his main model is fruit fly, or drosophila, cells. After observing a mechanism in tissue cells, he tests how that mechanism alters the development and behavior of the fly as a whole organism. Gelfand says he uses drosophila because they are easy to study and manipulate, and many genes present in these flies are conserved in and serve similar functions in humans.

“When we select something to study, we are careful to pick something common in drosophila and human cells,” Gelfand explains. “This way what we study is important for all aspects of human diseases. Moreover, if a mechanism is conserved from flies to humans, than it is important enough that we want to understand how it works.”

Recent Discoveries
In a recently published paper, Gelfand discovered for the first time that kinesin-1, a major motor that drives transport of cellular cargo through the cell, requires the regulatory protein ensconsin to function in neurons and non-neuronal cells.

By analyzing the movement of cellular cargo in two types of cultured cells, the scientists determined that when cells were depleted of ensconsin, the movement of the cargo along microtubules, or the tracks for transport, was severely impaired. They also found that the depletion of ensconsin impaired the ability of the kinesin-1 motor to assist in the moving of microtubules against each other, also known as microtubule sliding.

“Scientists have long suspected this is going on, but we published the first paper to actually show it,” said Gelfand.

In addition to showing its role in cell tissue function, Gelfand explored how ensconsin affects an entire organism by studying it in fruit flies. He found that flies without this protein do not survive into adulthood.

Gelfand has also discovered a completely new mechanism: the growth of neurites, or projections from the cell body of a neuron such as dendrites or axons, during neuronal differentiation. Gelfand showed that microtubules actually slide against each other and that sliding pushes the neurite out and drives growth.

“This is a new mechanism that no one has ever seen before,” he says. “It is now clear that microtubules can create neurite extension, and the implications are pretty big.”

Gelfand plans to continue exploring the mechanism that regulates the microtubule sliding. While he has found that the sliding drives growth in neurons, that process is shut down when neurons mature. If Gelfand can determine what shuts the process down, that same mechanism might reactivate neuron growth.

“If you know what shuts the mechanism that drives growth in neurons down, you can probably activate it. Understanding this mechanism could lead to therapeutics that might be used for patients with spinal cord injury, stroke, or any neurological disease.”

Collaboration and Curiosity
Gelfand is actively collaborating with other scientists at Feinberg.

He has worked with Robert Goldman, PhD, chair of the Department of Cell and Molecular Biology, studying the regulation and function of intermediate filaments in cells and Jacob I Sznajder, MD, chief of the Division of Medicine-Pulmonary, to study the roles of motor proteins in alveolar epithelial cells.

“These collaborations have led to greater findings and novel techniques,” he says. “Science is not always a linear track; you don’t go from one experiment to the next. Sometimes things going on in labs are beyond your control, and you follow your curiosity.”

In Gelfand’s case, that curiosity has led to a world of discovery.
Research Day Boasts Record Number of Posters

The ninth annual Lewis Landsberg Research Day, held April 4, 2013, brought together 318 poster presenters representing all 26 Feinberg departments for a celebration of science on the Chicago campus.

“Research Day is a great venue for learning what research is going on at the medical school, and we get to see a lot of different areas of science,” said Michelle Oliveira-Fernandes, a first-time presenter and first place winner for her basic science poster. “Being in the basic sciences, it is nice to see posters from the clinical sciences and public health.”

Faculty, graduate students, medical-scientist students, medical students, postdoctoral fellows, clinical residents and fellows, and research staff participated in the campus-wide event, which promotes faculty and trainee development through networking and the sharing of research.

The day kicked off with keynote speaker Ronald D. Vale, PhD, professor in the Department of Cellular and Molecular Pharmacology at the University of California-San Francisco and investigator in the Howard Hughes Medical Institute. His presentation, “Building a Mitotic Spindle,” focused on his breakthroughs and setbacks in the scientific process.

Faculty Mentor of the Year awards were provided to Stephen Miller, PhD, Judy Gugenheim Research Professor of Microbiology-Immunology, and Jack Kessler, MD, Ken and Ruth Davee Professor of Stem Cell Biology.

At the conclusion of Research Day, awards were announced for 12 individual presenters in the categories of basic science, clinical research, public health and medical social sciences, and women's health. More than 30 judges, comprised of senior Feinberg faculty, evaluated abstracts and posters based on their potential for contributing to the advancement of medical science and healthcare. In addition, the Alliance for Research in Chicagoland Communities presented the second annual Community Engaged Research Partnership Award to South Asian Healthy Lifestyle Initiative. The Tripartite Legacy Faculty Prize was awarded to Donald Lloyd-Jones, MD, MPH, Eileen M. Foell Professor of Medicine, senior associate dean for clinical and translational research, chair of the Department of Preventive Medicine, and director of NUCATS.

“This is the biggest Research Day I have been a part of. I think it is great to see more faces from different parts of campus,” said Ron Ackermann, MD, MPH, director of the Center for Community Health, associate professor of medicine, and Research Day judge. “As a judge, I spend a lot of time reading abstracts before the event. It is great to meet the people who have done the research, and I enjoy taking a closer look at the posters.”

Video recap on YouTube

2013 Research Day Winners

**Basic Science Research**
- First Place: Michelle Oliveira-Fernandes, PhD candidate
- Second Place: Laura A. Sena, MD/PhD student
- Third Place: Relja Popovic, PhD

**Clinical Research**
- First Place: Reeti Chawla, MD
- Second Place: Daniel C. Lee, MD
- Third Place: Aksharananda Rambachan, MD/MPH student

**Public Health and Social Sciences Research**
- First Place: Jennifer M. Duncan, PsyD
- Second Place: Lisa B. VanWagner, MD, MS
- Third Place: Jennifer A. Hershfield, MA, MS

**Women’s Health Research**
- Basic Science: Ann Marie Carias, PhD student
- Clinical Research: Jun Wang, PhD
- Public Health/Social Sciences: Javiera Pumarino, BA

**Community Engaged Research Partnership Award**
- South Asian Healthy Lifestyle Initiative

Complete list of winners, PIs, and project titles (PDF).
Waiting outside of his office sits more than 311 million Americans.

For David Baker, MD, MPH, investigating the way in which health services function provides an opportunity to affect an entire country, not just individual patients.

With roles at Feinberg that include chief of general internal medicine and geriatrics, deputy director of the Institute for Public Health and Medicine, and principal investigator of a multimillion dollar grant that established the Center for Advancing Equity in Clinical Preventive Services, Baker has dedicated his career to examining ways to improve population health.

“In a nutshell, I examine problems in the healthcare system and try to design solutions. My goal is to not only improve the health of individual patients, but of the larger population as well,” Baker said.

Twenty years ago, as the term “health literacy” emerged, few people were examining its meaning, and its impact on health care was unknown. Today, it’s a part of the lexicon of health care and health care policy, as researchers like Baker work to explain the vital role it plays in Americans’ health.

“My research is quite pragmatic; much of it has examined how patients’ reading ability influences their healthcare and health outcomes,” Baker said. “Now we are working to identify ways to overcome the barriers posed by low health literacy and improve quality of care and outcomes.”

Q&A

How does your research advance medical knowledge?

Most of my work has focused on understanding and addressing disparities, including health literacy, language barriers, and racial and ethnic disparities. I have led studies demonstrating that patients with low health literacy have higher hospitalization rates and higher mortality. This work has led to a large number of studies examining the causal pathways between literacy and health outcomes, along with research and evaluation studies examining strategies to mitigate the negative impact of low health literacy.

At what point in your life did you become interested in medicine?

I first became interested in medicine in high school. I always loved science, and I had some personal experiences that exposed me to medicine before I set my sights on this as a career. But where I am now is quite different than that original path.

During college, I worked with a volunteer public health program called Amigos de las Americas. I volunteered with a vaccination team in Paraguay and a dental program in Guatemala. These experiences got me interested in public health and health care policy. During my internal medicine residency in Los Angeles, these interests resurfaced because of the tremendous problems the public health care system was facing. This led me to pursue a research career focusing on health and health care disparities.

What types of collaborations are you engaged in?

Over the last two decades I have worked closely with Mark Williams, MD, chief of hospital medicine at Northwestern, and Ruth Parker, MD, professor of medicine at Emory University, on my health literacy research. Since coming to Northwestern, I have had the privilege of working with Michael Wolf, PhD, MPH, associate division chief for research in general internal medicine, on research examining health literacy and the association between health literacy and cognitive functioning. As director of the Center for Advancing Equity in Clinical Preventive Services, one of three centers for excellence nationally, we are working with investigators at the University of Colorado and the University of North Carolina at Chapel Hill.

What would you consider your defining characteristics outside of medicine?

My family is the center of my life. My wife, Ann, and I have three children who are the joy of my life and, simultaneously, the cause of my graying hair. I also love the outdoors and enjoy skiing, backpacking, kayaking, and cycling. My youngest son is a very talented basketball player who has finally taught me how to shoot a jump shot. So, basketball is now a very big part of my life.

Who has been the biggest influence on your career?

I would have to say Robert Brook, MD, ScD. He was my attending as a resident at the University of California—Los Angeles (UCLA). At the time, I was very involved in efforts to avert cutbacks in healthcare proposed by the Los Angeles...
Staff Profile: Lanty O’Connor
Program Manager, Northwestern Simulation

Where are you originally from?
I am originally from Long Grove, Ill.

What is your educational background?
I received a bachelor of arts degree, cum laude, in philosophy from University of Colorado at Boulder, and a master’s degree in health-care quality and safety from Northwestern University.

Tell us about your professional background.
I previously worked as a strength and conditioning coach with the University of Colorado and the Chicago Bulls. I’ve worked at Northwestern for four years, coming on board in 2008 to help build the simulation program.

Why did you choose to work at Northwestern?
I was recruited by John Vozenilek, MD, someone for whom I have profound respect and admiration. Through him, I was offered the opportunity to work at a well-respected university with a team of entrepreneurially minded people. It was an opportunity that I was enthusiastic about.

What is your role at the medical school?
I supervise the medical simulation program and the innovations lab in the Center for Education and Medicine’s Simulation Technology and Immersive Learning (STIL) program. The STIL laboratory houses approximately 40 simulation devices with hundreds of procedural practice opportunities. The innovations lab creates and produces medical training devices using state-of-the-art materials and techniques, including modern polymers, 3-D printing, and Computer-Aided Design (CAD), in collaboration with engineers with the McCormick School of Engineering.

Our work aims to improve the reliability of healthcare through education and research. The goal is to translate the work from the lab into the clinical environment in order to improve patient outcomes.

How do you contribute to the research mission here?
Our innovations lab is a great example of the collaborative nature of our education and research work. We work with teaching faculty to create novel educational devices to address areas of educational deficiency. Among others, we’ve created training models for facial plastic surgery, neonatal chest tube insertion, pediatric trans-esophageal fistula repair, paracentesis, and melanoma diagnosis.

We research the validity of these models and then use them as part of an educational intervention aimed at improving both the quality of training and patient outcomes.

What do you like to do in your spare time?
You can usually find me at the gym doing something active. I used to swim competitively and I still swim several times per week. In addition to being active on my own, I also train clients at HardPressed High Intensity Workouts, a boutique gym in River North.

Anything else we should know about you?
My full name is Maelaghlin Moore O’Connor. It’s an old Irish king’s name. Yes, junior high was a real treat.

► Connect with Lanty on LinkedIn.

Which honors are you most proud of and why?
In April, I will receive the Alvan R. Feinstein Award for Patient Care in the Field of Clinical Epidemiology from the American College of Physicians (ACP). This is a tremendous honor for me because it is one of ACP’s highest awards and because I knew Dr. Feinstein when I was a research fellow. He was a giant in the field, and to receive an award named in his honor is really special for me.

County Board of Supervisors. Bob and I had several long discussions, and he encouraged me to do a research fellowship. A year later, he accepted me into the UCLA Robert Wood Johnson Clinical Scholars program and he served as one of my key mentors. He is really one of the great thinkers in health services research and he taught me so many things. Most importantly, he helped me learn how to ask important questions and design studies that would give policy-relevant answers.
Student Profile: Amelia Ashley Mutso
Northwestern University Interdepartmental Neuroscience Program

Where is your hometown?
I grew up in Lancaster, Penn.

What is your educational background?
I graduated in 2008 from Haverford College with a major in psychology, a minor in German, and a concentration in neural and behavioral studies. As an undergrad at Haverford, I did my undergraduate thesis in biological psychology investigating neuroendocrine factors on empathy for pain in mouse models. Haverford was a great school for undergraduate researchers. Since there were no graduate students or postdocs, all the research was done by the principal investigators (PIs) and undergrads. This meant I was able to experience “real” research at an early point in my career and realize it was something I loved to do. I also spent each summer doing undergraduate research in the psychology departments of both Haverford and Northwestern University.

What are your research interests?
We know that chronic pain patients exhibit increased anxiety, depression, and deficits in learning and memory. Yet how persistent pain affects the key brain area regulating these behaviors, the hippocampus, has not been investigated. My main interest is to determine how the hippocampus is impacted by, and is involved in, the evolution of chronic pain. I’m also generally interested in cortical involvement in pain processing, interactions between stress, anxiety, and pain, as well as hippocampal neurogenesis.

What exciting projects are you working on?
I think everything I’ve worked on is exciting, but my current project is investigating the impact of hippocampal neurogenesis on persistent pain behavior. This stems from a study I published last spring showing that hippocampal neurogenesis is downregulated in animals with chronic neuropathic pain. Furthermore, what is great about my lab is that research takes place in both animal models and human pain patients. Because of this, I can extend what I find in animal models to human subjects.

I recently finished a study determining how hippocampal functional connectivity in back pain patients progresses differently in patients whose pain persists and those who recover.

What attracted you to the NUIN program?
NUIN was unique from other programs in several ways. First, having the ability to rotate through labs before deciding on a thesis lab was very appealing to me. It provided an opportunity to learn new techniques and delve into various research topics before finding one that was right. Second, I was really attracted to NUIN because the program accepts students from a variety of undergraduate majors and experiences, and then has everyone take a first-year curriculum of basic neuroscience so everyone is equipped with similar background. I knew that when graduating from this program, I would have a strong fundamental knowledge of the larger field of neuroscience, not just in my sub-specialty of chronic pain. Lastly, the strong collaborative research environment and strength of the research faculty was very attractive.

What has been your best experience at Feinberg?
I would say the best overall experience is the constant collaboration between labs. For my thesis, I am actively working across four different labs in several departments to complete my research, in addition to my home lab. Because of the positive collaborative environment, my thesis research is greatly strengthened from the insight, techniques, and support from these collaborators.

How would you describe the faculty at Feinberg?
They are at the top of their field, supportive, and collaborative.

What do you do in your free time?
In my free time, I love to ice skate, foster kittens for PAWS Chicago, cheer on the Cubs, and explore new and exciting restaurants throughout Chicago.

What are your plans after graduation?
I will be graduating this spring quarter and am currently looking for either postdoctoral opportunities or staff scientist positions in the Chicago area. I want to continue to be hands-on with research, but I also love the administration and management side of science (which seems to be rare) and would like to find a way to incorporate this into what I do. I would welcome any opportunities forwarded my way!

► Connect with Amelia on LinkedIn.
Sponsored Research

Jacob Sznajder, MD  
Chief, Division of Medicine-Pulmonary, Ernest S. Bazley Professor of Asthma and Related Disorders, and Professor of Medicine-Pulmonary and Cell and Molecular Biology

Project title: Role and Regulation of Sodium, Potassium ATPase in Lung Epithelium

Sponsor: National Heart, Lung, and Blood Institute

Patients with acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) have their lungs filled with fluid (edema accumulation) that leads to impaired alveolar gas exchange and low oxygen levels in the blood, which is associated with high mortality. Alveolar fluid reabsorption is affected by vectorial Na+ transport via apical Na+ channels and basolateral Na,K-ATPase of the alveolar epithelium. In patients with lung injury, the Na,K-ATPase and alveolar epithelial are impaired, which leads to edema accumulation.

Sznajder has previously described that in models of lung injury mechanical ventilation with low tidal volumes leads to improved epithelial function. These findings were translated into clinical trials, and the National Institutes of Health-sponsored ARDS network reported that indeed patients with ARDS ventilated with smaller tidal volumes and had significantly better survival.

More recently, in studies supported by this grant, Sznajder has described that dopamine and ß-adrenergic agonists via specific signal transduction pathways increase the Na,K-ATPase function by upregulating its protein abundance in lung epithelial cells, which resulted in increased alveolar fluid clearance in preclinical models of lung injury. However, a recent clinical trial in patients given aerosolized ß2-agonist for treatment in patients with ARDS was not beneficial. These findings raise questions of how to better stimulate and repair the alveolar epithelium and restore normal lung function for these patients to recover from acute lung injury.

Sznajder is now working with a team of investigators in the Pulmonary and Critical Care Division on elucidating pathways regulating the Na,K-ATPase and improving the lung epithelial function for patients to breathe better. The role and regulation of the Na,K-ATPase in the lungs is of paramount importance not only for ionic transport and cell homeostasis, but also for its cell-cell interaction and anchoring function as well as in cellular adaptation to stressful environments such as hypoxia and hypercapnia, which occurs in lung diseases.

"We have been working on the themes of this grant for almost 20 years with the focus on patients with lung diseases," Sznajder says. "For these patients to breathe normally, the alveolar epithelial function needs to recover from acute lung injury, lung infections, cystic fibrosis, asthma, chronic obstructive pulmonary disease, and lung cancer."

Conducting scientific experiments, says Sznajder, is a team endeavor which incorporates interdisciplinary tools to discover complex structures and specialized cellular functions.

"I have been fortunate to work with a team of inspiring individuals focused on scientific discovery which is very stimulating and rewarding," he says. "I am grateful to the wonderful colleagues in our division and collaborators from other Northwestern University departments, as well as our scientific collaborators from Argentina, Brazil, Canada, China, Germany, Greece, Israel, Japan, Mexico, Peru, Spain, Sweden, and Uruguay."

Teepu Siddique, MD  
Les Turner ALS Foundation/Herbert C. Wenske Foundation Professor, Professor in Ken and Ruth Davee Department of Neurology and Cell and Molecular Biology

Project title: Disease Mechanisms in Human Ubiquilinopathy

Sponsor: National Institute of Neurological Disorders and Stroke

Amyotrophic lateral sclerosis (ALS) is a neurodegenerative disorder affecting an estimated 350,000 individuals worldwide. It is characterized by degeneration of large motor neurons in the brain and spinal cord, resulting in progressive wasting and paralysis of voluntary muscles, respiratory failure and ultimately death. For 50 percent of patients this occurs within three years of symptom onset. A significant subset of patients experience cognitive changes that range from mild behavioral changes to frontotemporal dementia. The financial burden to the family approaches $200,000 per year in the final years of illness and its emotional toll is incalculable. At present the disease defies all treatment.

Familial ALS (FALS) accounts for around 10 percent of all ALS cases, with the majority (90 percent) of the disease presenting as sporadic ALS (SALS). Discovery of diverse genetic causes of ALS has been the driving...
Sponsored awards, continued from pg. 7

force in ALS research for the last 20 years, providing insight into mechanisms of disease. However, in spite of this effort, global treatments have not been possible for ALS because a common molecular mechanism downstream from the distinct etiologies has not become apparent. It is clear that discovery of additional etiologies and common downstream mechanisms will greatly advance the goals for a molecular understanding of ALS and in finding appropriate treatments.

Siddique has recently identified mutations in the gene UBQLN2 as the cause X-linked inherited ALS and ALS/dementia. The pathology associated with these patients is novel, distinct and unique, with ubiquilin2 positive inclusions in the spinal cord and cortex of patients with ALS alone, and additional inclusions in the hippocampus of patients with ALS and frontotemporal dementia (FTD). Additionally, to the surprise of Siddique, he has found similar ubiquilin2 positive inclusions in all cases of SALS, ALS/FTD and FALS (regardless of genetic cause) studied thus far. None of these additional cases had mutations in UBQLN2, implying there is a posttranslational role for ubiquilin2 in ALS as a whole.

Defects in the clearance of the ubiquinated proteins have long been speculated to cause neurodegenerative disorders, but until now direct evidence has been very limited.

Siddique’s studies of UBQLN2 in human patients (Deng et al, Nature 2011) and now in transgenic mice represent major breakthroughs in neurodegenerative research, providing to date the most robust in vivo evidence directly linking impaired protein degradation to protein aggregation, neuronal dysfunction and degeneration. Initial in vitro and in vivo data have implicated dysfunction in both the ubiquitin proteasome system (UPS) and autophagy in ALS and FTD. The data suggest that impaired autophagy is a mechanistic cause at the cellular level for ALS and FTD, directly linking impaired protein degradation to protein aggregation, neuronal dysfunction and degeneration. Mutant UBQLN2 transgenic mice developed UBQLN2/ubiquitin/p62-positive pathologic inclusions in the brain, especially in the hippocampus and cortex.

Siddique has also identified mutations in SQSTM1, the gene that encodes p62, in a subset of FALS patients. These inclusions are characterized by a novel “dendritic spinopathy,” observable with electron microscopy, which is associated with impaired transmission of presynaptic signals to post-synaptic neurons. To understand the molecular mechanism of inclusion formation in the dendritic spines, his team developed double transgenic mice expressing both mutant UBQLN2 and a representative ubiquitinated protein substrate of the proteasome, Ub-G76V–GFP.

In preliminary experiments, they found that UbG76V–GFP degradation is remarkably impaired and the inclusions in the dendritic spines are protein complexes composed of key components of ubiquitinated protein degradation machinery, including ubiquitin, p62, UBQLN2, proteasome subunits and Ub-G76V–GFP. They also demonstrated for the first time that autophagosome clearance is the step at which ALS-FTD-linked mutant UBQLN2 inhibits autophagy. Understanding the step(s) affected in the autophagic process is essential to developing targeted therapeutic approaches based on modulation of autophagy. At the same time, the data has implications for the wider role of UBQLN2 mediated protein recycling in diseases characterized by widespread UBQLN2 pathology even in the absence of UBQLN2 genetic mutations, like Alzheimer’s disease, Parkinson’s disease and non-UBQLN2 linked ALS and FTD.

Therefore, in this project Siddique uses several strategies to understand the disease-causing mechanism(s) of mutant ubiquilin2. He will further develop mouse models relevant to UBQLN2-linked ALS and ALS/dementia and establish pathological, cognitive, behavioral and motoric phenotypes and their electrophysiological correlates. The team will use their already established ubqln2 knockout mouse model and develop ubqln1 knockout and double knockouts of ubqln1/ubqln2 to test if mutant ubiquilin2 defects are the manifestation of a loss of function of ubiquilin2 in relation to the UPS and autophagy systems or whether mutant ubiquilin2 exhibits a novel toxic property and characterize both the of their phenotypes, as well as their pathology. Additional work will include characterization of the electrophysiological and morphological phenotype of hippocampal neurons in these models.

Successful completion of this project will not only provide insight into understanding the pathogenic mechanism of X-linked ALS, but also rapidly provide the wider research community with useful reagents for future studies and for screening and testing potential therapies. Results of this project will also have important implications in the understanding of the pathogenesis and treatment of other neurodegenerative diseases.
NIH News

Sally Rockey, PhD, NIH deputy director for extramural research, posted data that shows that the number of institutions competing for R01 applications has been relatively stable, but the number of institutions submitting Research Project Grant applications has increased.

NIH issued a new set of frequently asked questions related to NIH-supported core facilities and their operation in compliance with the terms of award, including applicable federal cost principles.

An information notice concerning plans to reissue a Funding Opportunity Notice regarding the NIH Pathway to Independence Award (K99/R00) has been posted. NIH is planning to make major changes to the award, including the eligibility criteria.

NIH has issued a request for applications for the next round of the Lasker Clinical Research Scholars program. The program is designed for early stage clinical researchers, within 10 years of completing their initial residency program. Lasker Scholars spend five years as funded, independent investigators in the NIH intramural program, then have the opportunity for additional independent financial support either at the NIH or at an extramural research institution. The application deadline is June 24, 2013, for positions that will begin in 2014.

A new All About Grants podcast has been posted to the NIH web site. The podcast, “Who Should I Contact at NIH? Roles of NIH Staff and How They Work Together,” is now available.

Welcome New Faculty

Jordan Grafman, PhD, joins as professor in the Department of Physical Medicine and Rehabilitation, Cognitive Neurology and Alzheimer's Disease Center, Psychiatry and Behavioral Sciences, and Weinberg College of Arts and Sciences. He is the Coleman Chair of Rehabilitation Medicine at the Rehabilitation Institute of Chicago.

Grafman was most recently director of the Kessler Foundation's Traumatic Brain Injury Research Laboratory, in West Orange, N.J. He previously was chief of the National Institute Neurological Diseases and Stroke's Cognitive Neuroscience Section and before that, senior staff fellow for the National Institute of Neurological and Communicative Disorders and Stroke's Clinical Neuropsychology Section in Bethesda, Md. Prior, he was director of human neuropsychology from the University of Wisconsin—Madison.

His primary interest is in understanding the functions of the human prefrontal cortex and improving those functions after brain damage. He has additional interests in neuroplasticity, memory, executive functions, and social neuroscience. Grafman uses structural and functional MRI, non-invasive brain stimulation, psychophysiological recordings, and genetics as tools in this research effort.

Joshua Meeks, MD, PhD, joins as assistant professor in the Department of Urology and section chief of robotic surgery at the Jesse Brown VA Medical Center.

Meeks received his doctor of medicine degree and his doctorate degree in tumor biology from Northwestern University, in Chicago, where J. Larry Jameson, MD, PhD, served as his graduate and postdoc mentor. While at Northwestern, he also completed two postdoctoral research fellowships, an internship in general surgery, and clinical residency in urology, and served as research assistant professor. He recently completed a urologic oncology fellowship at Memorial Sloan-Kettering Cancer Center in New York.

His research interests focus on both the epigenetics and genetic mutations associated with cancer biology. Meeks has joined the lab of Jonathan Licht, PhD, chief of the Division of Medicine-Hematology/Oncology and Johanna Dobe Professor of Hematology/Oncology, and will be studying how chromatin remodeling genes play a role in bladder cancer. In addition, he is studying the “driver” mutations found in bladder cancer. In the future, he hopes to develop novel systemic and intravesical therapies to improve survival of patients with bladder cancer.
Research in
the News

FOXNews.com March 27
Antidepressants not tied to stunted infant growth
Katherine Wisner’s research was featured.
▶ Study was also mentioned in US News & World Report, Huffington Post

New York Times March 26
New prostate cancer tests could reduce false alarms
William Catalona was quoted.

Chicago Tribune March 26
Prescription for nutrition
Clyde Yancy was quoted.

New York Times March 25
Looking for evidence that therapy works
Bonnie Spring was quoted.

Time Magazine March 19
How a healthy heart can lower risk of cancer
Laura Rasmussen-Torvik’s research was featured.
▶ Study was also mentioned in CBS News, US News & World Report

Reuters March 19
Emergency room symptoms may not predict health care needs: study
James Adams’ research was featured.
▶ Study was also mentioned in US News & World Report

Wall Street Journal March 11
Hard math: Adding up just how little we actually move
Bonnie Spring was quoted.

New York Times March 2
Experts want more studies of diet’s role for the heart
Neil J. Stone was quoted.

More headlines

High Impact Factor Research
February 2013


Help Feinberg Track Journals
The Feinberg Research Office regularly tracks research published by Feinberg investigators. The citations are used on web pages, in newsletters and social media, for internal reporting, and more. To more accurately track these journals, the Research Office asks that Feinberg investigators use the following institution name in the address field when publishing in peer-reviewed journals: “Northwestern University Feinberg School of Medicine.”
Funding Opportunities

Specialized Programs of Research Excellence (SPOREs) in Human Cancer for Years 2013 and 2014 (P50)

More information

Sponsors: United States Department of Health and Human Services (HHS), National Institutes of Health (NIH)
Submission Deadline: May 21
Upper Amount: $12.5 million

Synopsis: This opportunity will fund five-year P50 SPORE grants to support state-of-the-art investigator-initiated translational research that will contribute to improved prevention, early detection, diagnosis, and treatment of an organ-specific cancer (or a related group of cancers). SPOREs are expected to conduct a wide spectrum of research activities and contribute significantly to the development of specialized shared resource core facilities, improved research model systems, and collaborative research projects with other institutions. The research supported through this program must be translational in nature and focused on knowledge of human biology stemming from research using cellular, molecular, structural, biochemical, and/or genetic experimental approaches with the goal of a translational human endpoint within the five-year term of the grant. SPOREs must include both a developmental research program for pilot studies and a career development program to foster careers in organ-based translational science.

NICHD Program Project Grant (P01)

More information

Sponsor: United States Department of Health and Human Services (HHS), National Institutes of Health (NIH), Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD)
Submission Deadline: May 25
Upper Amount: $4 million

Synopsis: This opportunity encourages innovative, multidisciplinary, interactive, and synergistic program project grant applications from institutions that propose to conduct research on reproductive, developmental, behavioral, social, and rehabilitative processes that determine the health or functioning of newborns, infants, children, adults, families, and populations. The purpose of the P01 mechanism is to encourage investigation of complex problems relevant to NICHD’s mission and to facilitate economy of effort, space, and equipment. Under appropriate circumstances, the collaborative research effort of a program project can accelerate the acquisition of knowledge more effectively than a simple aggregate of research projects without thematic integration.

View more funding opportunities

Featured Events

4.16 IBNAM Colloquium
“Bio-inspired materials for applications in medicine and surgery,” presented by Elliot L. Chaikof, MD, PhD, Harvard Medical Center
Date: Tuesday, April 16, 4:30 to 5:30 p.m.
Location: Lurie Research Center — Baldwin 303 E. Superior St. (Chicago campus)
Contact: jill-johnson@northwestern.edu
More information

4.18 Thursday Seminar Series
“An online social support ART adherence intervention: The ‘Thrive with Me’ study,” presented by Keith J. Horvath, PhD, Medical College of Wisconsin
Date: Thursday, April 18, Noon to 1 p.m.
Location: Lurie Research Center — Baldwin 303 E. Superior St. (Chicago campus)
Contact: emily.stambaugh@northwestern.edu
More information

4.25 Lurie Cancer Center Tumor Cell Biology Seminar
“Mitochondria: cancer’s crystal ball,” presented by Anthony Letai, MD, PhD, Dana-Farber Cancer Institute, Boston
Date: Thursday, April 25, 1 to 2 p.m.
Location: Lurie Research Center — Searle 303 E. Superior St. (Chicago campus)
Contact: cancer@northwestern.edu
More information

4.25 Cell & Molecular Biology Seminar
Presented by Sergei Y. Sokol, PhD, Mount Sinai Hospital, New York
Date: Thursday, April 25, 4 to 5 p.m.
Location: Ward Building, 4-075 CMB Conf. Rm. 303 E. Chicago Ave. (Chicago campus)
Contact: b-jaron@northwestern.edu
More information

5.6 Biostat Seminar
“Coming to our sensors: Why body language is harder to decode than natural language,” presented by Ciprian Crainiceanu, PhD, Johns Hopkins Bloomberg School of Public Health
Date: Monday, May 6, 3 to 4:30 p.m.
Location: 680 N. Lake Shore Drive Stamler Conf. Rm. (Chicago campus)
Contact: f-nichols@northwestern.edu
More information

More events

Event organizers are encouraged to submit calendar items on Plan-It Purple for consideration. Please contact the Research Office with further questions.