

The NICHD Connection

June 2016

INSIDE THIS ISSUE

Twelfth Annual Fellows Retreat Recap	1
Letter from the Editor	2
Life Outside Lab: 2016 Fellows Retreat	13
Meet Our New Fellow	14
June Announcements	15
June Events	19

EDITOR IN CHIEF

Shana R. Spindler, PhD
Shana.Spindler@gmail.com

LAYOUT & DESIGN

Nichole Swan

BACKGROUND PHOTOGRAPHY

Unsplash
Pixabay
Life Outside Lab photos by
Jeremy Swan

CONTRIBUTORS

Smriti Aryal A C, BDS, PhD
Arnab Datta, PhD
Courtney Kurtyka, PhD
Pushpanathan Muthuirulan, PhD
Uma Neelathi, PhD
Neelam Dabas Sen, PhD

Twelfth Annual Fellows Retreat Recap

The NICHD Twelfth Annual Meeting of Postdoctoral, Clinical, and Visiting Fellows and Graduate Students took place on April 22, 2016 at the National Museum of the American Indian in Washington, D.C. Fellows gathered to share research findings, learn from top-notch scientists (including a Nobel Laureate), and chat over snacks and well-designed posters. The creator of wildly popular *PhD Comics*, Dr. Jorge Cham, even made a guest appearance, expounding upon the benefits of—what else—procrastination in his keynote address.

Several fellows who attended the event recapped the talks for *The NICHD Connection*. We are excited to bring you the 2016 Annual Fellows Retreat presentations. Enjoy!

SECRETS BEHIND GREAT SUCCESS *By Pushpanathan Muthuirulan, PhD*

“Do what you love to do, and do it with passion,” Dr. Eric Betzig said during his keynote address to fellows at the Twelfth Annual NICHD Fellows Meeting. Betzig, 2014 Nobel Prize Laureate in Chemistry, is a group leader at HHMI’s Janelia Research Campus, where he develops optical imaging technologies. His talk recalled his transition from early career successes to a mid-career setback, followed by a groundbreaking scientific discovery.



Dr. Eric Betzig

During Betzig’s PhD studies at Cornell University (1985–1988), he focused on developing high-resolution optical microscopes that could see preserved cells at the theoretical limit of 0.2 micrometers. Later, he was recruited by AT&T Bell Laboratories, and in 1992, he became the first person to image individual fluorescent molecules at room temperature, localizing their positions to within 0.2 micrometers.

Despite his early string of successes, Betzig became frustrated with the academic community and corporate structure. In 1994, he voluntarily left his positions.

(continued on page 3)

Letter from the Editor

Many of you are running out the door on vacation, so I will keep my words brief! I'd rather focus your attention to the outstanding articles by NICHD fellows who graciously volunteered to recap this year's Annual Fellows Retreat. I can't think of a better way to celebrate the six-year anniversary of this newsletter than to focus on you, the fellows, and all that you do.

Cheers!
Shana R. Spindler, PhD

Please send questions and comments to Shana.Spindler@gmail.com.



Twelfth Annual Fellows Retreat Recap

(continued from page 1)

Betzig spent some years without a job before he became Vice President of Research and Development at Ann Arbor Machine Company (owned by his father), where he developed Flexible Adaptive Servohydraulic Technology (FAST). After spending millions of dollars on its development, they sold a total of two devices that failed to meet their expectations or achieve commercial success. Betzig referred to this time as the “dark side” of his life. The unfortunate circumstances made him realize the importance of customer satisfaction. He learned that satisfying an urgent need was the underlying secret behind great success.

In 2002, Betzig went back to his field of microscopy by founding a firm known as New Millennium Research in Okemos, Michigan. Having deep admiration for the work of Mike Davidson and his fluorescent proteins, he teamed up with his former colleague Harald Hess to develop photoactivated localization microscopy (PALM), a method of controlling fluorescent proteins using pulses of light.

Betzig considered 2005, the year he and Hess began developing PALM, as the luckiest year in his life. Over the course of the year, he built—literally—his invention and career with support from Hess and other collaborators, including Dr. Jennifer Lippincott-Schwartz. After Betzig and Hess developed the PALM-capable microscope, Lippincott-Schwartz hosted their work and housed their creation at the NICHD. With her help, Betzig and Hess created images with a higher resolution than was previously thought possible.

The next year, Betzig and Hess joined the HHMI’s Janelia Farm Research Campus as a group leader to continue work on super high-resolution fluorescence microscopy techniques. There, he used his technique to study cell divisions in human embryos. In 2014, Betzig was awarded the Nobel Prize in Chemistry for the development of super-resolved fluorescence microscopy.

Beyond the Nobel Prize, Betzig continued to push the boundaries of microscopy with innovative technologies. His latest work allows scientists to look at mitosis in a developing zebrafish eye! Betzig ended his inspirational talk with advice on how to succeed in science without really trying. He said:

- » Do what you love to do, and do it with passion
- » Have a good reason for doing what you do
- » Don't be afraid to make a change
- » Failure is painful, but a great teacher (pain is the best teacher)
- » Live for yourself and live for your needs
- » Your reputation is your most important asset
- » Don't let others dilute your convictions
- » Be your own toughest critic
- » Work hard, be a bit scared
- » You have a responsibility to others who support you

Just one among many in the audience, I was highly motivated by Dr. Eric Betzig. I believe that his words can inspire all researchers to put their ideas into action and battle through failures in order to attain great success.

(continued on page 4)

Twelfth Annual Fellows Retreat Recap (continued from page 3)

MAINTAINING THE CELL'S RECYCLING CENTER By Neelam Dabas Sen, PhD

Lysosomes are known as the cell's recycling center. They recycle macromolecules like carbohydrates, lipids, proteins, DNA, and RNA in cells by digesting them. This is achieved by the activity of hundreds of enzymes called hydrolases in the acidic lumen of lysosomes. Dr. Maria Bagh, a research fellow in Dr. Anil Mukherjee's lab, began the morning fellow talks by describing the central role played by lysosomes in intracellular digestion, the importance of acidic pH in ensuring this role, and the diseases developed if this function is compromised.

Bagh studies a group of hereditary childhood neurodegenerative lysosomal storage diseases called neuronal ceroid lipofuscinoses (NCLs), estimated to occur at a frequency of 1 in 100,000 individuals worldwide. Symptoms of these diseases include progressive, permanent loss of motor and psychological ability with a severe intracellular accumulation of lipid-containing residue from lysosomal digestion. Currently there is no effective treatment of NCLs.

Lysosomal storage disorders can occur when a particular hydrolase is defective in the lysosome, resulting

in excess accumulation of unwanted macromolecules. Infantile NCL is caused by inactivating mutations in the gene encoding palmitoyl-protein-thioesterase-1 (Ppt1), a lysosomal enzyme that catalyzes the removal of palmitoyl groups from proteins. Without Ppt1, palmitoylated proteins accumulate in the lysosome. Children with Infantile NCL are normal at birth but start to exhibit symptoms by 6 to 24 months old. Symptoms progress rapidly, causing psychomotor retardation, blindness, abnormally increased muscle spasm, seizures, and no brain activity. These children usually die before age 5.

Using pH sensitive dye, Bagh showed that lysosomal pH was increased in mice lacking Ppt1. To understand the lysosomal acidification defect, she looked into the activity of v-ATPase, a multi-subunit proton pump that maintains lysosomal pH. Bagh found that v-ATPase assembly and disassembly required dynamic palmitoylation of an important v-ATPase subunit. Without Ppt1, the subunit was misrouted in the cell,



Dr. Maria Bagh

(continued on page 5)

Twelfth Annual Fellows Retreat Recap (continued from page 4)

which led to defective lysosomal acidification. Treatment with a drug that mimicked the job of PptI restored lysosomal pH to near normal levels.

Bagh's fascinating work reveals an unanticipated role of PptI in regulating lysosomal pH and may lead to better understanding of lysosomal acidification defects in other lysosomal storage disorders or common neurodegenerative disorders like Alzheimer's and Parkinson's disease.

PI4KA: A NEW TARGET FOR DEMYELINATING DISEASES OF PERIPHERAL NERVOUS SYSTEM?

By *Arnab Datta, PhD*



Dr. Alejandro Alvarez-Prats

"Myelination is a striking process," said Dr. Alejandro Alvarez-Prats, visiting fellow in the Section on Molecular Signal Transduction. Alvarez-Prats studies the role of Phosphatidylinositol 4-kinase III-alpha (PI4KA) in the myelination of the peripheral nervous system (PNS). Myelination is the process of forming a myelin sheath that surrounds the axon of various nerve cells to generate an electrically insulating layer. The loss of myelin can cause demyelination diseases such as Guillain-Barre Syndrome in the PNS.

In mammalian cells, PI4KA regulates the metabolism of phosphatidylserine (PS). The PS together with phosphatidylethanolamine (PE) are major components of the myelin sheath. To evaluate the function

of PI4KA during myelination, Alvarez-Prats and his colleagues in the laboratory of Dr. Tamas Balla created mice with PI4KA-deficient Schwann cells—glial cells of the PNS that participate in the formation of myelin sheath.

He studied mutant mice for three months in order to identify any visible motor defects. He was surprised to observe a subtle yet early gait abnormality in one-month-old homozygous mice. "These mice started showing dramatic impairment in using their hind legs by the age of two months," Alvarez-Prats explained.

In order to better understand the cellular and molecular aberrations, he examined the sciatic nerve by electron microscopy (EM) and immunohistochemistry. The sciatic

(continued on page 6)

Twelfth Annual Fellows Retreat Recap (continued from page 5)

nerve is one of the longest and widest nerves in the human and animal body. It provides the sensory connections to limbs and feet. Intuitively, any abnormality in the gait or impairment of the hind leg may have a direct connection with the sciatic nerve morphology. The EM results confirmed Alvarez-Prats's hunch. He saw a dramatic decrease in myelin thickness, onion bulb formations around the nerve, and decreased myelin-associated proteins, all hallmarks of demyelination disease.

Fueled by these exciting results, Alvarez-Prats performed a systems biological analysis to identify the downstream lipid molecules that are affected by the knockout of PI4KA. The lipidomic analysis of the sciatic nerves showed greatly reduced phospholipid content that disproportionately affected PS, PE, and sphingomyelin, which is another important component of myelin.

He is now looking forward to studying the role of PI4KA on actin polymerization within the context of migration and myelination processes. He added that he wants to compare genetic versus pharmacological inhibition of PI4KA.

"To our knowledge, this is the first study on the role of PI4KA in the PNS," Alvarez-Prats said. "Understanding the control of phospholipid synthesis and transport in myelin formation could help to identify new targets for the treatment of myelination disorders in the future."

(continued on page 7)

Twelfth Annual Fellows Retreat Recap

(continued from page 6)

INCREASED IMMUNE ACTIVITY WHEN LACKING La

By Uma Neelathi, PhD

In many neurodegenerative diseases, the central nervous immune system is turned on. Dr. Nathan Blewett, a postdoctoral fellow in Dr. Rich Marais's lab, is interested in understanding whether this phenomenon is good or bad for the progression of diseases. His work focuses on La, an endogenous protein with an ability to stimulate autoantibodies, an antibody against an organism's own tissues.

La is a conserved RNA binding protein that promotes tRNA maturation. It has implications in viral mRNA maturation and regulation of cellular mRNA. To study La's function, Blewett knocked down La protein in the excitatory neurons of the mouse brain. As expected, loss of La affected RNA maturation, specifically causing accumulation of pre 5.8S RNA. In the La mutants, he observed that the frontal cortex shrank by 16 weeks of birth, and the mice had progressive neurodegeneration by 12 to 14 weeks of age. He also showed that the reason for neuronal death was due to an up-regulation of cleaved Caspase-3 and increased apoptosis.

To further understand why La mutant brains shrank, he completed next generation RNA sequencing of the frontal cortex of the mutant mice and control mice and observed a strong up-regulation of immune and inflammatory pathways by 13 weeks, becoming more severe by 20 weeks of age. Blewett demonstrated with beautiful confocal images that astrocytes, which are involved in electrical impulse transmission, infiltrate the mutant cortex by 13 weeks. Microglia, the first line of immune defense in the nervous system, exhibit activated morphology. He also saw a dramatic loss of the blood-brain barrier in the mutant mice.

His combined data suggests that La mutant mice have strong activation of the immune system resulting in neurodegeneration. In his future studies, he is interested in understanding why La perturbs 5.8S processing and what's happening in the brains of La mutant mice before the onset of immune activation during the early stages of neurodegeneration.



Dr. Nathan Blewett

(continued on page 8)

Twelfth Annual Fellows Retreat Recap (continued from page 7)

COPPER HOMEOSTASIS AND NEURON DISEASE By Smriti Aryal A C, BDS, PhD

Dr. Diego Martinelli, visiting fellow in the Section on Translational Neuroscience, believes that the zebrafish is an important model for studying human disease. Scientists can easily manipulate and investigate the tiny fish at the genetic level, an important trait for Martinelli's research. He studies a type of ATPase protein, called ATP7A, which resides in the cell's golgi and regulates cellular copper homeostasis. It moves from the golgi to the plasma membrane in response to copper. In humans, ATP7A is associated with Menkes disease, an X-linked, severe pediatric neurocutaneous disorder that occurs in 1 out of every 50,000 to 100,000 infants.

Menkes disease is characterized by connective tissue abnormalities, severe neurodegeneration, and early lethality. The current treatment for Menkes disease is based on daily subcutaneous copper injection, but 75 percent of patients do not respond.

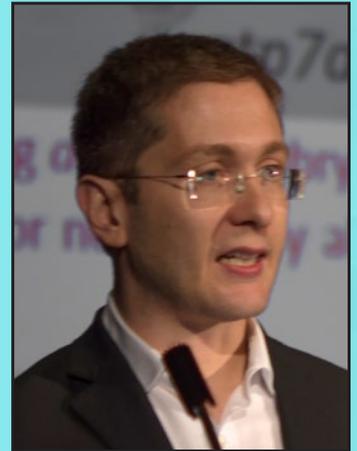
To understand the functional role of ATP7A in the peripheral nervous system, Martinelli used an existing

zebrafish model of Menkes disease known as *calamity*. The *calamity* mutant harbors a mutation in the zebrafish homolog of ATP7A and shows defects in pigmentation and hindbrain development along with early mortality.

To study mutations in human ATP7A linked to motor neuropathy, Martinelli and his colleagues generated homozygous *calamity* mutants that selectively expressed GFP in motor neurons. Using time-lapse confocal imaging, they evaluated motor neuron development in the living *calamity* embryos from 24 to 72 hours after fertilization.

Starting 48 hours after fertilization, they observed motor neuron degeneration, including disorganized motor neuron cell bodies and axonal distribution with reduced GFP signal. The embryos also had progressive paralysis, as assessed by neuro-behavioral testing. In an important experiment, microinjection of human ATP7A mRNA prevented the neuron and motor abnormalities.

(continued on page 9)



Dr. Diego Martinelli

Twelfth Annual Fellows Retreat Recap (continued from page 8)

Martinelli is now studying the effect of two unique missense mutations in *ATP7A* gene that are associated with an adult onset distal motor neuropathy without copper abnormalities. They are generating knock-in models for the two mutations via Crispr/cas9 targeted mutagenesis. Overall, Martinelli's findings highlight the essential role of *ATP7A* in the maintenance and function of motor neurons.

LANGUAGE DELAY LINKED TO BRAIN BLOOD FLOW By *Smriti Aryal A C, BDS, PhD*

When screening for autism, a common neurodevelopmental disorder marked by impairments in social communication and repetitive behaviors, identifying infants at high risk is a major challenge. Dr. Elizabeth Smith, postdoctoral fellow in the Section on Functional Biophotonics, discussed the importance of finding a way to predict autism risk in infants.

Autism researchers have several obstacles to overcome. First, genetic studies have shown that most cases of autism cannot be explained by genetic findings alone, with hundreds of genes associated with increased risk for autism. Second, metabolic biomarkers have been unsuccessful at predicting autism with high confidence. Brain imaging is a potential way to measure biological variation in autism, but delays in communication and social interaction typically emerge at an age when brain imaging is challenging.

In her study, Dr. Smith evaluated the efficacy and feasibility of using functional near infrared spectroscopy (fNIRS), an imaging method that captures changes in blood flow, on the brains of toddlers with emerging delays in language and social communication prior to autism diagnosis. Using this imaging technique, she measured the neuron function and oxygen levels in the prefrontal cortex in toddlers at 18 to 36 months, including toddlers with typical development and those diagnosed with language delays. She found that toddlers with language delays had lower oxygen levels across both hemispheres, indicative of the differences in the movement of oxygenated blood in the cerebrum.



Dr. Elizabeth Smith

(continued on page 10)

Twelfth Annual Fellows Retreat Recap (continued from page 9)

Smith's findings suggest that using neuroimaging to studying the blood flow properties of the resting brain might be useful in predicting and understanding early language delays. Her findings add to a growing body of knowledge concerning language development, which could one day aid in the diagnosis of toddlers with autism.

FACTORS IN FUSION: MAKING OSTEOCLASTS FROM MACROPHAGES

By *Smriti Aryal A C, BDS, PhD*

Osteoclasts (multinucleated cells of the bone) are the major cells to resorb bone during the bone remodeling process. They play an essential role in a number of diseases, such as osteoporosis and osteopetrosis. However, there has been little focus on studying the mechanism of cell fusion that generates osteoclasts. Dr. Santosh Verma from Dr. Leonid Chernomordik's laboratory has focused his research on isolating the fusion stage of osteoclast formation and identifying the proteins involved in this process.

Two macrophages fuse to form osteoclasts. Fusion is very slow, unsynchronized, and includes a number of intermediate steps, making it difficult to identify the protein's players. To address the fusion mechanism in osteoclast formation, Verma applied a reversible hemi-fusion inhibitor to human monocytes (a type of white blood cell that can differentiate into a macrophage) and murine established cell lines. The inhibitor blocked macrophage fusion and synchronized the cells, which allowed him to concentrate 16 hours of cell fusion events into one hour.

Verma found that at the time of fusion, macrophages exposed phosphatidylserine (PS), a lipid normally found in the inner leaflet of the plasma membrane, on their surface in a non-apoptotic manner. He also found that peptide inhibitors and antibodies to PS-binding proteins known as annexins suppressed fusion.

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Dr. Santosh Verma

Twelfth Annual Fellows Retreat Recap (continued from page 10)

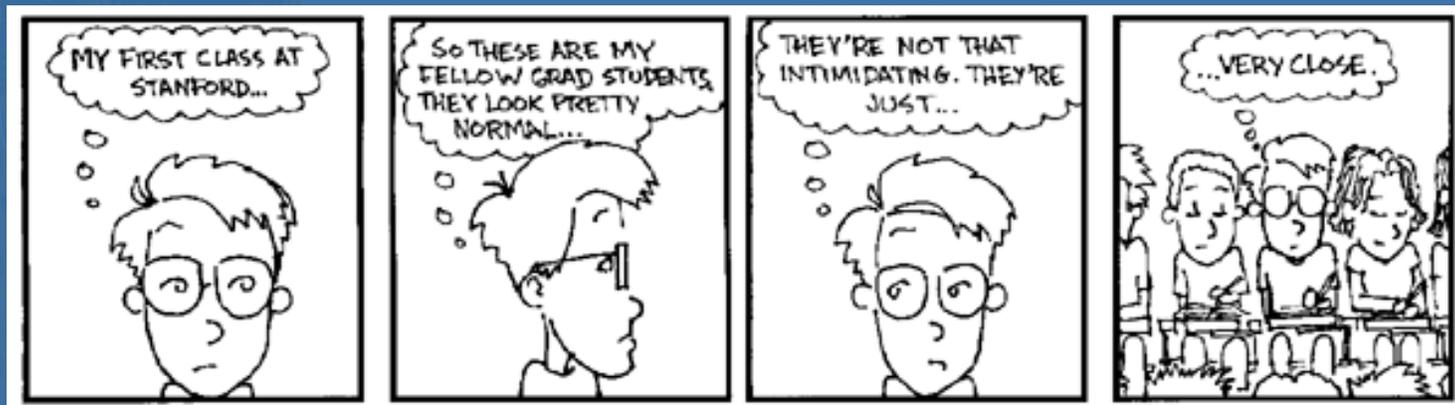
The fusion stage also involved endogenous retroviral human Syncytin-1 protein, which is known to fuse cells that turn into the placenta. Verma found that Syn peptide inhibited synchronized human monocyte fusion. Utilizing his synchronized fusion technique, he developed a membrane-mixing assay, which allowed him to decipher when proteins act during the cell fusion process. He showed that both annexins and Syncytin-1 act during the early membrane merger stage to initiate the macrophage cell fusion process.

Dr. Verma believes his novel approach to study macrophage cell fusion could also work to investigate other unexplored cell fusion processes during development. He hopes future experiments in a knockout mouse model and clinical disease samples will further characterize these protein players during fusion.

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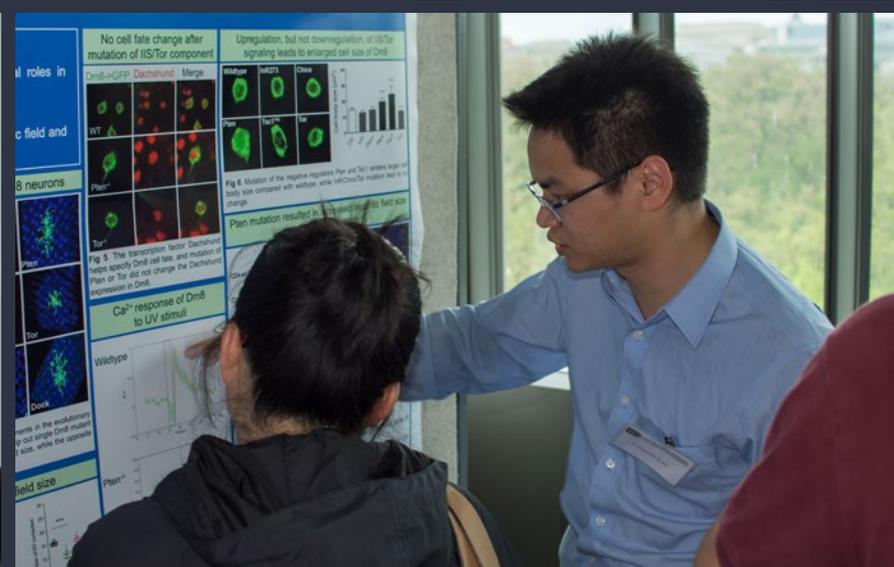
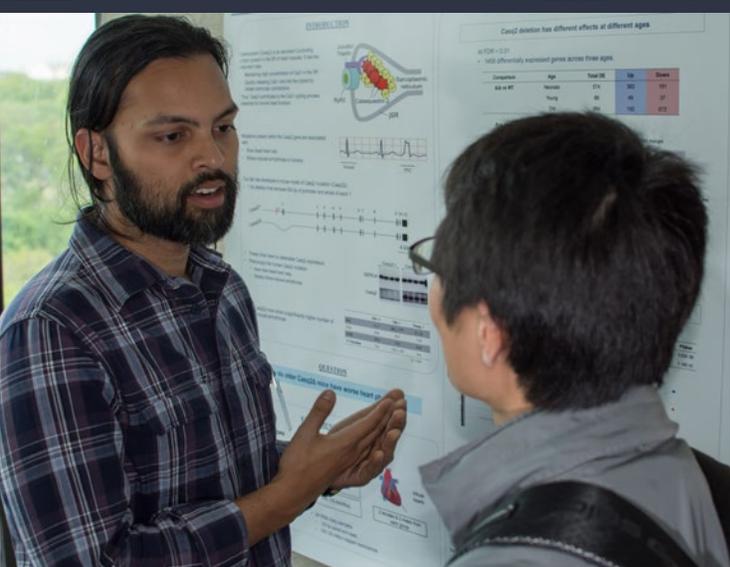
Twelfth Annual Fellows Retreat Recap (continued from page 11)

In honor of our afternoon guest speaker, Dr. Jorge Cham, the creator of *PhD Comics*, we bring you the strip that started it all. Presenting the first, ever, *PhD Comic*, originally published October 27, 1997.



Life Outside Lab: 2016 Fellows Retreat

See more photos online at newsletter.nichd.nih.gov



Have you been to a national park recently? We are seeking pictures of NICHD fellows at national parks. Please send your submissions to Shana.Spindler@gmail.com, including your name, park, and date the photo was taken. Thanks!

Meet Our New Fellow

Please join *The NICHD Connection* in welcoming the following fellow to the NICHD family:



FARDIN GHOBAKHLOU

Postdoctoral fellow

Home City: Montreal, Canada

Degree institution: Ph.D. Molecular Biology Microbiology,
University of Laval, Quebec City, Canada

NICHD mentor: Dr. Alan G. Hinnebusch

Area of research: I study the molecular mechanisms of
gene translation and the roles of particular proteins in
translation reinitiation and ribosome recycling using
yeast as a model for eukaryotic cells.



June Announcements

PRESENTING THE 2016 THREE-MINUTE-TALK (TmT) FINALISTS

The **Three-minute-Talk (TmT)** competition is a science communication training and awards program that teaches fellows how to explain their research, in three minutes or less, in a way that's meaningful to a broad scientific audience. This year, six NICHD fellows are finalists and will compete in the competition on **Tuesday, June 14**, with fellows from NHGRI and NIDCR. Congratulations to all on your hard work.

The NICHD TmT finalists and semifinalists (in no particular order):

FINALISTS

Alex Szatmary
Afrouz Anderson
Jeremy Weaver
Parmit Singh
Miranda Broadney
Courtney Kurtyka

SEMIFINALISTS

Edra London
Pushpanathan Muthuirulan
Santosh Verma
Gulcan Akgul
Neda Sadeghi
Vasilisa Aksenova
Saroj Regmi
Uma Neelathi



(continued on page 16)

June Announcements

(continued from page 15)

CONGRATS TO THE NICHD 2016 BEST POSTBAC POSTER WINNERS

During the 2016 Postbac Poster Day on April 20, 2016, NICHD postdocs selected the three top postbac poster presentations from our institute. The judging criteria were based on the fellow's knowledge of the lab's research, the fellow's ability to describe the project clearly, and the design and layout of the poster, highlighting hypothesis-driven questions.

Congratulations to the NICHD 2016 "best" postbac poster winners!

- » Alessandro Albano (Delaney lab)
- » Marci Rosenberg (Chitnis lab)
- » Lauren Wooddell (Suomi lab)

POSTBAC POSTER DAY 2016 AWARDS, NIH-WIDE

Congratulations to our postbacs who received an Outstanding Poster Award during the NIH-wide competition, hosted by the Office of Intramural Training and Education, during the 2016 Postbac Poster Day. Teams of graduate students, postdoctoral fellows, clinical fellows, and staff scientists/staff clinicians collaborated to judge the posters. The following NICHD postbacs authored posters that scored in the top 20 percent of all posters presented. Congratulations to all!

OUTSTANDING POSTER AWARD RECIPIENTS:

Michal Ad	Alexander Haddad
Yared Bayleyen	Nicolas Johnson
Nikhita Chahal	Katrina Koon
Vy Duong	Nabil Saleem
Daniel Flores	Katherine Thompson
Elena Ghanaim	

To see award recipients from other institutes, please visit www.training.nih.gov/postbac_poster_day_awards_no_year.

(continued on page 17)

June Announcements

(continued from page 16)

NICHD POSTBACS ACCEPTED INTO PRESTIGIOUS PROFESSIONAL SCHOOLS

We are pleased to announce that our postbacs will attend a large collection of professional schools this coming school year. Big congratulations to our postbacs on a job well done. Check out where they will be heading:

MD PROGRAMS

University of Pittsburgh
 Pennsylvania State
 University
 Georgetown University
 Yale School of Medicine
 Emory University
 Florida International
 University
 Tufts University

PHD PROGRAMS

University of California,
 Berkeley
 Oregon Health and
 Science University
 Johns Hopkins University
 Vanderbilt University

MD/PHD PROGRAM

University of California,
 San Francisco

STOP BY FOR “RESUME HOUR” WITH YVETTE PITTMAN!

Every Wednesday, 1 – 2 p.m.
 Building 3I, Room 1B44

Are you flustered when you have to describe yourself on paper? Learn how to perfect your resume or CV during Yvette Pittman’s weekly office hours, every Wednesday from 1–2 p.m. Don’t miss this unique opportunity to review your resume or CV in great detail with Yvette.

You will:

- » Learn about keywords
- » Review formatting
- » Establish what to include—and not to include
- » Identify your target audience
- » And much more!

(continued on page 18)

June Announcements (continued from page 17)

CHECK OUT THE NICHD ADVISORY COMMITTEE—A GREAT OPPORTUNITY TO SERVE

The advisory committee works with the NICHD Office of Education to develop and initiate academic support programs for the institute. The committee meets the last Thursday of every month, from 3:30-4:30 p.m. Both domestic and visiting fellows are needed. We want to achieve broad representation, culturally and academically, so we can address the needs of all our trainees at NICHD.

Some potential topics for our committee are how to:

- » Increase the participation for training activities
- » Expose fellows to various careers in science
- » Identify teaching opportunities, and internal and external research funding mechanisms
- » Establish a structure for sharing scientific and career resources within the institute

Please contact Yvette Pittman at yvette.pittman@nih.gov if you are interested in joining the committee.

NIH RESEARCH FESTIVAL POSTER ABSTRACTS DUE JUNE 15, MIDNIGHT

This is a call for poster abstracts for the 2016 NIH Research Festival, to be held from **September 14 to 16**, Wednesday through Friday. The Research Festival is the annual showcase of NIH intramural research. All members of the NIH Intramural Research Program are encouraged to participate, but each individual is limited to a single poster as a first author in order to allow as many people as possible to present their work.

To submit a poster abstract:

1. Visit <http://researchfestival.nih.gov/forms/poster.cgi>
2. Enter poster information.
3. Submit form by **11:59 p.m., June 15, 2016.**

June Events

(continued from page 18)

TUESDAY, JUNE 14, 1:00 PM

Three-minute-Talk (TmT) Final Competition
Lipsett Amphitheater



Join the NICHD cheering section, as our fellows compete with others from NHGRI and NIDCR in the 2016 TmT Finals Competition (see list of fellows in [announcements](#)) to win the NIH-wide grand prize for best three-minute talk about their research. Imagine describing your research in less than three minutes: come and see how it's done!

FRIDAY, JUNE 24, 10–11 AM

NICHD Postdoc and Graduate Student Orientation
(For new postdocs and graduate students)
Building 31, room 2A48

Led by the NICHD Office of Education, the orientation will highlight both NICHD and NIH-wide intramural resources for postdoc fellows and graduate students. Topics will include:

- » Career-planning tools
- » Grant opportunities for fellows
- » Ideas for presenting your science locally
- » Scientific resources and core facilities available to you

We will share information about key programs to support your professional development, complementing the mentored experience you will have at the bench. Don't miss this opportunity to meet fellows from other research areas and different buildings on campus.

To register, please contact Yvette Pittman (pittmanyv@mail.nih.gov).