

The NICHD Connection

December 2013

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EDITOR IN CHIEF

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

LAYOUT & DESIGN

Nichole Swan

CONTRIBUTORS

Libby Barksdale, PhD
Ricardo Correa, MD, Es.D.
Aniket Gore, PhD
Monica Gupta, PhD
Alison Heffer, PhD
Robert Mitchell, PhD
Saravana Murthy, PhD
Payal Ray, PhD
Alex Ritter
Megan Sampley, PhD
Naoyuki Sarai, PhD
Parmit Kumar Singh, PhD

PHOTOGRAPHY

Jeremy Swan
Stock.XCHNG
Morguefile

PGD Fellows Coordinate Invited Speaker Activity By Aniket Gore, PhD, and Alison Heffer, PhD

Fellows in the NICHD Program on Genomics of Differentiation (PGD) initiated the invited seminar speaker activity with support from NICHD Scientific Director Dr. Constantine Stratakis, PGD Director Dr. Brant Weinstein, PGD faculty, and following a suggestion from the site visit committee. Through this activity, PGD fellows (postdoctoral, graduate student, and postbaccalaureate) formed a committee to organize a visit by outside speakers.

During the first year, the committee members invited six seminar speakers, both local and domestic, to visit PGD, present a seminar on topics of broad interest to PGD groups, and meet with fellows to discuss the fellows' work and guidance about the future. Last year, these speakers included Dr. Todd Macfarlan (NICHD), Dr. Warren Leonard (NHLBI), Dr. Mahendra Rao (NIH), Dr. Tom Mistelli (NCI), Dr. Antonio Giraldez (Yale), and Dr. Elaine Fuchs (Rockefeller).

Committee members and participants for 2012-2013 were Aniket V. Gore (Chair), Robert Crouch (Faculty Advisor), Christelle de Renty, Kathryn Monzo, Kevin Francis, Aneeshkumar Arimbasseri, Mithun Mitra, Minho Won, Mariya London, Yoko Ogawa, Uma Neelathi, Payal Ray, Eric Sauce, Sandip De, Nate Parker, Brett Athans, Alison Heffer, Valeria Zarelli, and Sheran Law. For August 2013- July 2014, Alison Heffer is the chair of the committee, and Damian Dalle Nogare, Sandy Mattijssen, Naushaba Hasin, Amy Ton, and Valerie Virta joined as additional committee members.

We had a very successful PGD fellows seminar this past September. Dr. Denise Montell visited from UC Santa Barbara and presented a seminar about her lab's work on cell adhesion and migration and a new phenomenon her lab recently found, called anastasis, in which apoptotic cells change their fate. While she was visiting, many postdocs in PGD were able to meet and discuss their research with Dr. Montell.

(continued on page 3)

Letter from the Editor

Pour yourself a big mug of hot chocolate, grab your favorite blanket, and cozy up by a warm fire—or space heater (let's face it, we're on a trainee's budget). This issue is sure to keep you entertained on a cold December night. You'll find [event recaps](#), [interesting opportunities](#), [award-winning research summaries](#), [the year in review](#), and [December announcements](#) and [events](#). With so much to read, I'll let you get to it.

May the rest of your year be filled with cheer... and data.

Your Editor in Chief,
Shana R. Spindler, PhD

Please send your letters, questions, and ideas to Shana.Spindler@gmail.com.

PGD Fellows Coordinate Invited Speaker Activity (continued from page 1)

We are excited about our upcoming tentative speaker schedule:

SPEAKER	TOPIC	VISIT DATE
Dr. Michael Gottesman NIH	Drug resistance in cancer	Jan 31, 2014
Dr. Charles Kimmel University of Oregon	Evolution of craniofacial development	Apr 25, 2014
Dr. Marianne Bronner California Institute of Technology	Regulation of neural crest development	May 30, 2014
Dr. Claire Waterman NHLBI, NIH	Cellular movement	Summer 2014
Dr. Andy Baxevanis NHGRI, NIH	Phylogenetic diversity and protein evolution	TBA

These seminars have been a large success and are highly appreciated by PGD and non-PGD members. In particular, attendees enjoy the organization of the talks and selection of speakers. The seminars are open to all NIH researchers and are advertised on different NIH list-servs as well as the NIH calendar. The committee receives help from PGD administration in booking the seminar hall, posting seminar notices, and handling travel-related paperwork.

Fellows from PGD particularly benefited from this activity. They had a chance to meet with the visitors either in individual meetings or in a group at lunch to discuss their unpublished results with these scientists from outside their research area and receive feedback on their work. The activity also helped fellows become familiar with the speaker's work and created an opportunity for them to form a personal connection with the speaker. This seminar activity received positive response and enthusiastic support from PGD members to continue the activity into the next fiscal year.

Interesting Opportunity: Serving as a Medical Editor

By Ricardo Correa, MD, Es.D.

In this newsletter, I will tell you about my experience as a young physician engaged in medical editing, which involves working as an editorial board member on a medical journal. I hope that you will consider exploring this topic once you learn from my experiences thus far. As a new physician, this has been a fun and intellectually challenging opportunity for me.

Six years ago, I was invited by Latin America Medical Index (LATINDEX) to participate in a course titled "How to be an Editor." At first, I was reluctant to participate because I was busy working as a recently graduated clinician, and I was also doing a research fellowship in public health at Gorgas Memorial Institute (a Panamanian national public health institute). My mentor told me, however, that if I wanted to improve my country's scientific publications, I should get involved in the editorial process. This recommendation led me to participate in an editing course, which spanned several months.

The course ended up being a life-changing event. I have a new perspective on the entire scientific process. Prior to the course, when a journal rejected one of my articles, I usually criticized the editors. I thought that because I was not an established scientist, they did not read my work. Now I realize that an editorial board is made up of people who work because they have passion for science and want to contribute to it.

It was at this point I decided to become a medical editor. I can personally tell you that this pathway has been very difficult. At the beginning, nobody was paying attention to me, probably because I was a young physician with some training but not a lot of real publications.

However, I kept at it.

My first experience was with the Spanish journal, *Archivos de Medicina*. This journal gave me the opportunity to start applying what I had learned in the initial course I took. I started as a junior editor, which was frustrating because I felt I was doing basically secretarial work. Then after a couple of months, I was promoted to full editor and the real work started. I worked at least three hours per night. Given my other responsibilities, there were some points that, had it not been for my love of publications, I would have quit due to exhaustion. From my tenure with *Archivos de Medicina*, rather than quit, I decided to get more involved.

I had a meeting with senior editors of Panamanian journals and presented a project to make their journals more effective. That was one of the most frightening moments of my life. Can you imagine being in front of your professors and telling them what they need to do? At the end of that meeting, they agreed with my recommendations. As a result, we founded the Panamanian Association of Medical Editors (PAME), an organization that to this day has helped save seven journals in the region.

As a result of PAME, I was invited to work on a project to create the Central American Association of Medical Editors (CAME). Finally in 2009, the CAME project became a reality and we had our first meeting in Honduras. Thanks to CAME, regional journals have the opportunity to become indexed in prominent biomedical literature databases, such as Medline and Embase.

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Interesting Opportunity: Serving as a Medical Editor (continued from page 4)

Following these experiences, a couple of my mentors liked my enthusiasm and offered me training positions on their editorial boards. I had my first non-Spanish editorial positions with *The Journal of Infectious Disease in Developing Countries* and *International Archives of Medicine*. From these experiences, I learned the methodology of approaching different papers, how to evaluate double publication, how to use certain editorial computer programs, and how to analyze and integrate the peer reviewers' commentaries and recommendations.

I am now using my editing experience to tackle a personal goal: making science available to everyone. In 2011, I trained on the Open Journal System (OJS). OJS is a journal management and publishing system developed by the Public Knowledge Project in an effort to expand and improve access to research. Using this training, some of my residency friends and I decided to create the first open access resident journal in the United States. The main objective is to stimulate publication among our peers. This dream is becoming a reality, as the journal will be launched at the beginning of next year (2014).

Every day I am learning more about the fascinating area of medical editing. I can tell all of you that even though this journey has had its difficult and busy periods, I don't regret it. Being a junior medical editor has allowed me behind the scenes in scientific publication and clarified why some articles get published and others do not.

The role and make-up of medical editors is changing. In the past, most editors were scientists with many publications. Nowadays, in addition to publications, you need training in the editorial process. I remember that the Editor in Chief of *The Journal of the American Medical Association (JAMA)* once told me that if he knew he would become the senior editor of JAMA, he would have started his training when he was a resident, 30 years ago. These are true words of wisdom.

If you have an interest in a medical editing career, don't be afraid to start. The path may seem difficult, but you will learn a lot while making an important contribution to the scientific world.

For those who have interest in this area, I would like to establish an interest group to develop new projects. My contact information is below.

ricardo.correa@nih.gov

Clinical Fellow, Endocrinology NICHD/
NIH

Associate Editor of *International
Archives of Medicine*

Junior Editor of *International Journal of
Case Report and Imaging*

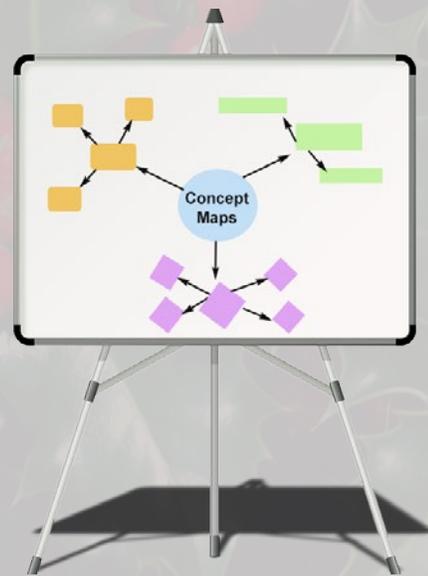
Internet Medical Publishing, Editorial
Board

Editor's Note: In 2010, Dr. Correa published a book in Spanish titled *Casos Clinicos: Semiologia y Publicacion*, a guide for medical students on publishing clinical articles.

Dr. Quimby's Teaching Workshop Recap

By Parmit Kumar Singh, PhD

The NICHD Office of Education has arranged a teaching workshop for postdoctoral fellows since 2012. Dr. Boots Quimby, an associate director of the Integrated Life Science Honors program, University of Maryland, conducts this six-week workshop, which takes place in the summer. During the workshop, she introduces students to new concepts in college teaching, like concept maps, backward design, the 5E model of teaching, and Bloom's taxonomy. Her lectures span three main areas involving student learning: how to plan for learning, how to make learning more effective, and how to assess learning.



PLANNING FOR LEARNING

Concept maps, backward design methodology, and Bloom's taxonomy are three planning tools that maximize student learning. During the first class, Dr. Quimby taught us how to make a concept map when organizing lecture content. A concept map is a graphical way of representing information. It has two components: 1) concepts that are enclosed in circles or boxes, and 2) the relationships between concepts, represented by connecting lines. Such representation visualizes how one piece of knowledge is related to another. Concept maps help organize lectures and learning activity based on the concepts' relationships. At the course level, the concept map helps link each lecture.

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The best learning outcome focuses on what the learner has achieved and not what the instructor has taught.

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Next, Dr. Quimby introduced the concept of backward design. In this planning method, we identify the target learning outcomes—what we want students to learn. We then determine the criteria for the evaluation of student learning, i.e., their assessment. Only after we establish the learning outcomes and criteria for evaluation do we plan the learning exercises and instruction. Classroom exercises should ensure that everyone acquires the new information and can use the information for their target goal.

To meet those target goal requirements, Dr. Quimby taught us how to generate learning outcomes when planning our instruction. The rule of thumb is to focus on what we want students to know or know how to do. For example, is it a skill development or a knowledge increment? What does the student need to know in order to do such an activity? The best learning outcome focuses on what the learner has achieved and not what the instructor has taught.

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Dr. Quimby's Teaching Workshop Recap (continued from page 6)

So how do we establish learning outcomes? Dr. Quimby suggests that we use Bloom's taxonomy, a categorization of the types of learning. It has six components:

1. Knowledge (remembering or recalling the information)
2. Comprehension (understanding the concepts behind the information)
3. Application (using the knowledge in a new situation)
4. Analysis (breaking big or complex data into smaller parts to draw a conclusion)
5. Synthesis (putting the conclusions from analysis together to make a big picture)
6. Evaluation (making a judgment or final conclusion)

Bloom's taxonomy helps remind us that recalling information and applying information are two different learning outcomes that may require different types of instruction. In general, each step along Bloom's taxonomy requires a greater depth of learning.

MAKING LEARNING MORE EFFECTIVE

Next, we learned how to make our learning activities more effective. She explained that it's important to use a method of learning that supports interaction. She introduced us to the 5E model of teaching:

1. Engagement: activities promote curiosity among students.
2. Exploration: students attempt to solve problems to show they understand the concept.
3. Explanation: students discuss confusion that arises during exploration.
4. Elaboration: students apply concepts to solve novel problems.
5. Evaluation: students assess their understanding of the new concept.

To increase student interaction, Dr. Quimby suggests ending the class with a short discussion using words like "what," "so what," and "now what." Such small pauses will help get feedback from the students.

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Dr. Quimby's Teaching Workshop Recap (continued from page 7)

ASSESSING LEARNING

In Dr. Quimby's last lecture, we learned the method of assessment. The key point is to make sure assessment matches learning outcomes. Assessment should include both formative assessment and summative assessment. Formative assessment is the evaluation of student progress and occurs throughout the course of learning. It creates a feedback loop between students and teachers. Such feedback helps to modify the teaching and learning activities to maximize student progress. Summative assessment is at the end of a full course, and includes activities like final exams, presentations, and final projects. Assessment should cover all of the learning outcomes and should balance between summative and formative assessment.

Teaching college students is an exciting career option for many postdoctoral fellows. This workshop helped me understand that teaching is a continuously evolving process and that feedback and interaction with students is very important. Having knowledge of concept maps, learning outcomes, Blooms taxonomy, and the 5E model of teaching will help me design more effective and interactive lectures. I am confident that by utilizing these concepts, anyone can motivate more and more young minds in college and enjoy being a science educator.



NICHD Award-Winning Research: FARE Competition

The Fellows Award for Research Excellence (FARE) is an annual competition that recognizes fellows' research contributions to intramural programs. Winners are announced during a FARE Award Ceremony at the NIH Research Festival each autumn. FARE recipients receive a \$1000 travel stipend to present their work at an upcoming scientific meeting, the chance to display a poster at the FARE awards presentation ceremony, and the opportunity to serve as a judge for the following year's FARE competition. Twenty-six NICHD fellows received the award this year, and many of those fellows volunteered to share their work with *The NICHD Connection*. See what your fellow fellows are up to!

When Too Much Excitement Isn't a Good Thing

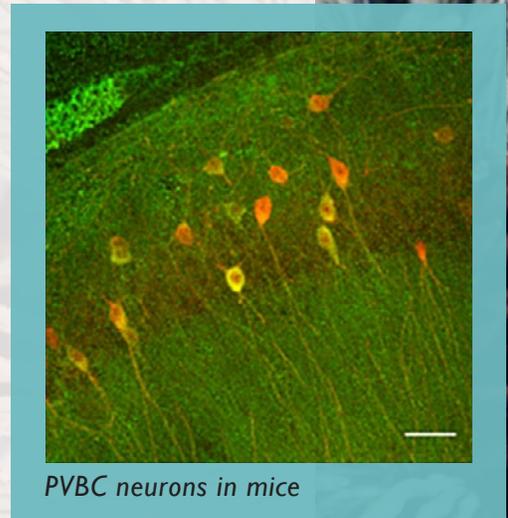
By Libby Barksdale, PhD (postdoctoral fellow, McBain lab)

A growing number of neurological disorders, from autism to epilepsy to schizophrenia, are hypothesized to result from an imbalance of excitation and inhibition in the brain. Mechanisms behind this imbalance vary widely, however, and are still not fully understood. One area of intense scrutiny is the role of gamma oscillations, a type of measurable brain wave, in the process of disease. Gamma oscillations have been implicated in higher cognitive functions, including storing and recalling information and combining sensory inputs into unified percepts.

Generation of gamma oscillations is driven by the activity of parvalbumin-expressing basket cells or PVBCs, a type of neuron whose activity inhibits other neurons. Factors affecting PVBCs' ability to regulate excitation/inhibition dynamics could provide potential therapeutic targets for associated disorders. A family of carbohydrate-binding proteins, called Neuronal Pentraxins (NPs), present an intriguing possibility in this capacity.

NPs localize to excitatory synapses on PVBC dendrites to promote clustering of specific proteins used in neural interactions. When we deleted NPs in mice, those specific proteins used in excitatory interactions failed to show up. By eliminating two different NP proteins, we found physiological changes in the strength and frequency of spontaneous neural firing of PVBCs. This in turn significantly decreased the power of gamma oscillations in these mice as compared to typical mice.

Altogether these results demonstrate the importance of NPs in controlling the integration of excitatory synapses on PVBCs within neural circuits and the role this has in gamma wave generation.



PVBC neurons in mice

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FARE Competition (continued from page 9)

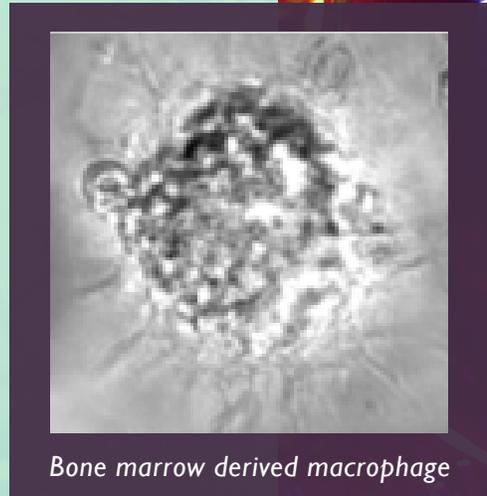
Bacteria's Fate Rests with IRF8 By Monica Gupta, PhD (visiting fellow, Ozato lab)

The innate immune system protects us from the constant onslaught of bacteria, viruses, and other pathogens. Without it, our bodies would be susceptible to a wide range of infection. But once cells of the innate immune system, such as macrophages, engulf invading pathogens, they must eliminate these unwanted visitors. To do so, they use a process called autophagy, a regulated, catabolic pathway for the turnover of proteins, damaged organelles, and in the case of immune system cells, pathogens. More than 20 genes are involved in the execution of autophagy; however, regulation of autophagy in immune cells is not well understood.

In our study, we found that a protein known as Interferon Regulatory Factor 8 (IRF8), which controls expression of immune system related genes, is an important regulator of pathogen-induced autophagy in macrophages. When a macrophage cell is called to action in response to pathogen infection, Toll like receptors emit signals within the cell. We found that in response to these signals, IRF8 stimulates expression of at least 17 autophagy genes in macrophages.

We infected macrophages with the bacterial pathogen *Listeria monocytogenes* and found increased autophagy with most of the bacteria localized within autophagosomes—a unique and important structure formed during autophagy. But without IRF8, autophagosome formation was impaired. The IRF8-deficient macrophages failed to induce autophagy, resulting in poor clearance of bacteria upon infection. Taken together, our results provide new insights into the mechanism of pathogen elimination in the innate immune system.

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Bone marrow derived macrophage

FARE Competition

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Finding a Novel Target for Treating Neuropsychiatric Disorders

By Robert Mitchell, PhD (postdoctoral fellow, Buonanno lab)

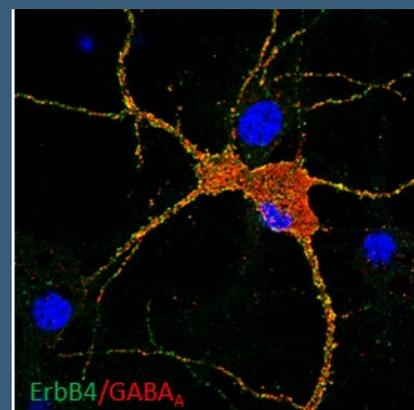
corrected from previous version, which listed Dr. Mitchell in the Huang lab

In higher organisms, networks of excitatory and inhibitory neurons sculpt patterns of neurotransmission to orchestrate complex behaviors. Some of the genes involved in “fine tuning” these patterns of neuronal activity are linked to complex psychiatric and neurological disorders like schizophrenia and epilepsy. Two such genes encode proteins for the neuronal receptor ErbB4 (a tyrosine kinase, which is an enzyme that phosphorylates other proteins, altering their activity) and its binding partner Neuregulin (NRG).

ErbB4 is present in inhibitory, but not excitatory neurons, and likely regulates neuronal networks by altering the activity of these neurons. However, little is known about how ErbB4 performs this task. Using advanced approaches (i.e., proteomics, electrophysiology, transgenic animals, and quantitative microscopy) we showed that ErbB4 interacts with the GABA_A receptor, a protein channel whose activity makes neurons less likely to fire an action potential. We found that this interaction is augmented by NRG binding, and activation by NRG results in the removal of GABA_A receptors from the inhibitory neuron’s surface, likely increasing the activity of these neurons. Importantly, using a kinase-inactive mutant version of ErbB4, we showed that this process does not require ErbB4’s kinase activity.

These results are exciting because they reveal a novel potential pharmacological target that bypasses ErbB4’s kinase activity; inhibitors of which tend to have off target side effects that limit clinical efficacy. Selectively manipulating this pathway could pave the way to better drugs for neuropsychiatric disorders and epilepsy. Our results have recently been published in the *Proceedings of the National Academy of Sciences*.

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Surface labeled GABA_A receptors (red) on ErbB4 (green) expressing cells. Nuclei labeled blue.

FARE Competition

(continued from page 11)

This Is Your Brain on Stress

By Saravana Murthy, PhD (research fellow, Loh lab)

We've all been there: we crammed like crazy for our school exams, but we still blanked out while taking the actual test. And we wondered, "Why?" While the stress of preparing for our exams motivated us to study, it impaired our recall. If the stress persists, it could lead to the development of neurological diseases and even accelerate cancer progression. There is a very fine line between beneficial stress and harmful stress. This fine line is called allostasis, the body's way of responding to stress by maintaining normalcy in our brain.

Researchers have struggled for a long time to unravel the role of allostasis in protecting against neuronal damage. Our lab is studying carboxypeptidase E (CPE), an enzyme that functions as a signaling molecule to combat oxygen deficiency in brain cells. In mice lacking CPE (CPE-KO), we found that even mild stress such as weaning caused damage in the hippocampus, a region that plays an important role in encoding memory.

To follow up on these observations, we stressed normal and CPE-KO mice by immobilizing them for one hour each day for seven days. In normal mice, stress hormones increased CPE protein levels in the hippocampus. Other proteins such as BCL2 and p-AKT, which indicate healthy neurons, were also increased, while the protein BAX, a marker of dying neurons, was reduced. In CPE-KO mice, the healthy neuron markers were decreased and the dying neuron markers increased. Based on these results, we think that CPE protects normal mice from brain damage and maintains allostasis during stress. We hope that our research on CPE could one day inspire new treatments for chronic stress.



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FARE Competition

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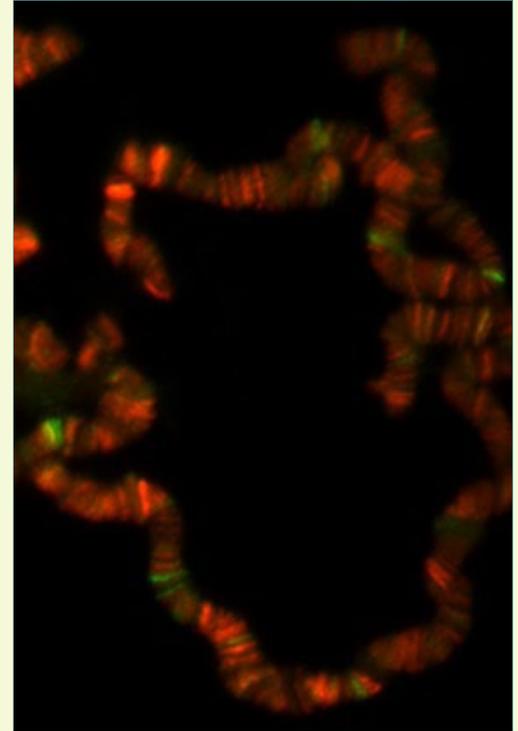
Silence of the Gene

By Payal Ray, PhD (visiting fellow, Kassis lab)

Animal development requires a fine-tuned, well-orchestrated series of gene expression changes. This developmental programming involves the activation or silencing of various sets of genes at different time-points. I aim to identify how certain developmentally regulated genes are turned off (repressed) in animals. A group of factors known as Polycomb Group (PcG) proteins are known to regulate gene repression. Members of the PcG protein family are conserved from the common fruit fly to humans, and mutations within PcG proteins have been linked to cancer.

Within the nucleus, the DNA is tightly wrapped around an octamer unit that is composed of a class of proteins called histones. Addition of methyl (-CH₃) groups to histone protein by PcG protein complexes spatially compacts the DNA so that the machinery for gene expression cannot access the code. PcG proteins interact with elements in the DNA known as PcG Response Elements (PREs). The PRE region has several binding sites for DNA binding proteins that recruit PcG proteins to methylate the histone subunits, thus repressing gene expression in that area.

The exact mechanism of PcG protein recruitment and the factors involved are unknown. Identification of the factors that bind to PREs is important to completely understand how PcG proteins silence gene expression. Using a mass spectrometry-based approach, I identified a protein, Combgap, which binds to a set of PcG target genes in the common fruit fly, *Drosophila melanogaster*. My future work will address the role of Combgap in this interaction.



Drosophila melanogaster salivary glands showing colocalization of Combgap (red) and well-known PcG member, Spps (green)

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FARE Competition

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How Single-Shot Cytotoxic T-Cells Stay Fully Loaded

By Alex Ritter (graduate student, Lippincott-Schwartz lab)

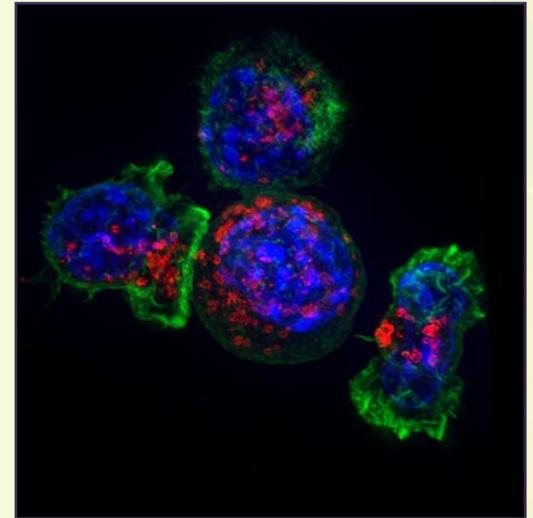
I study the cells of the immune system that are responsible for hunting down and killing cancerous and virus-infected cells in the body. These cells have been aptly named cytotoxic T cells (CTL). CTL kill their targets through the polarized secretion of organelles called lytic granules, which contain an array of cell-damaging proteins.

CTL are serial killers, meaning that they can kill multiple cells, one after another. The lytic granules within CTL are the ammunition that these cells use to destroy their targets. This ammunition is very potent. It takes only one or two lytic granules to destroy a target cell. In order to kill multiple cells, CTL must regulate the number of granules they secrete, saving ammunition for the next target. But how CTL regulate their secretion is unknown.

The cortex of mammalian cells consists of a thick layer of branched actin, which has been proposed to act as a barrier to secretion. I hypothesized that CTL manipulate the density of this actin barrier to control the number of lytic granules released toward a target cell.

Using high-resolution, live-cell imaging techniques on fluorescently labeled actin and lytic granules in CTL, I observed that the density of actin at the interface between the CTL and a target cell is greatly diminished. I also observed that just after granule secretion, a wave of actin polymerization emanates from the secretion site, increasing the density of actin covering the interface. This actin “wall” appears to prevent further granule secretion, as lytic granules that are visible close to the plasma membrane do not fuse for release.

When we treated cells with latrunculin A, a small molecule which destabilizes actin polymers, the actin “wall” disappeared, and granules immediately and continuously fused with the plasma membrane. This data suggests that secretion of a few lytic granules induces actin polymerization at the CTL-target interface, which acts as a barrier to prevent further secretion. In this way, CTL preserve “ammunition” to use on other virally infected or tumorigenic cells.



Immunofluorescence image of cytotoxic T cell (left) engaging a cancer cell (round cell, center). Labels: actin (green), lysosomes (red), nuclei (blue)

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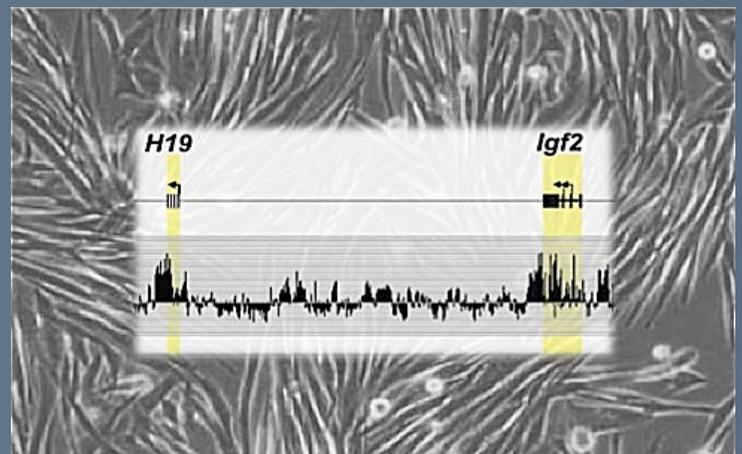
FARE Competition (continued from page 14)

Simon Says Stop

By Megan Sampley, PhD (postdoctoral fellow, Pfeifer lab)

Cell growth and division are like the children's game Simon Says. Sometimes Simon says grow, and the cell activates genes that promote cell division. Sometimes Simon says stop, and the cell summons genes that suppress division. But sometimes, a cell makes a mistake and grows when Simon never said to grow. When certain genes are mistakenly activated or deactivated at the wrong time, tumors can form.

I study two genes: one called H19, which is presumed to be a tumor suppressor gene, and another gene called Igf2, conversely shown to stimulate growth. The dysfunction of these genes is implicated in multiple cancers, though the exact nature of their contribution lacks clarity.



ChIP-chip data overlaid on primary mouse monocytes

H19 and Igf2 reside on the distal end of mouse chromosome seven, giving them a fascinating DNA structure and organization. They are interesting because they have directly opposing functions; yet, they sit right next to each other in the genome and are controlled by the same short stretch of regulatory DNA.

In the case of muscle cells, this short stretch of DNA is an enhancer that binds specific machinery that helps make gene products. I use genomics approaches to study how these gene-activating molecular machines are recruited to H19 and Igf2. I found that the enhancer has much broader effects. The enhancer appears to set up the architecture of the entire genomic region that contains the H19 and Igf2 genes, not just those two. This work provides new insight into how many genes are available for activation in that genomic region. From this, we stand to gain new information on just what goes wrong in a tumor cell when it stops playing by the rules.

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FARE Competition

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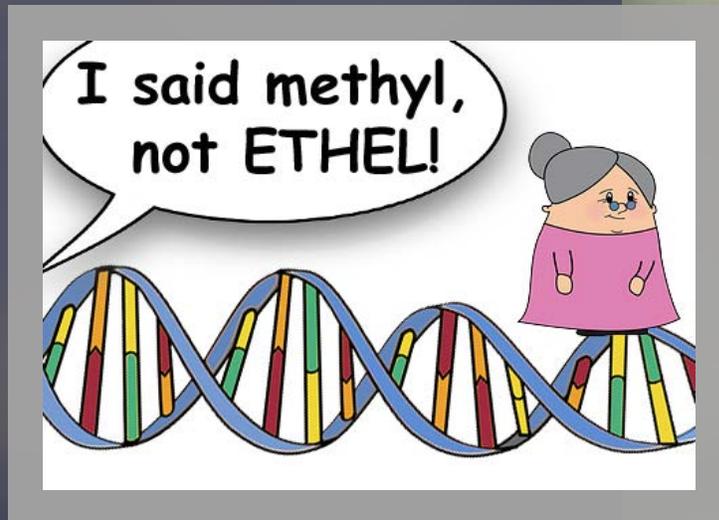
Why'd You Put a Methyl on My K36?

By Naoyuki Sarai, PhD (visiting fellow, Ozato lab)

There has been a flurry of publications demonstrating the link between pediatric glioblastoma, a cancer of the supportive tissue in the brain, and mutations in a protein called Histone H3.3. These studies provide compelling evidence for the role of H3.3 in normal brain development, and more importantly its potential role in cancer.

Histone H3.3 is a variant of Histone H3, a protein that helps pack DNA into tight structures called nucleosomes. H3.3 is expressed throughout the cell cycle and deposits along DNA when cellular machinery destabilizes the nucleosomes to access genes during transcription. In H3.3, the thirty-sixth amino acid (K36) receives a methyl group modification. However, it is unclear what H3.3 is doing in the normal brain, what the role of K36 methylation is, and how this variation affects cancer.

To gain further insight, we investigated the role of the protein that adds the methyl group to K36, called WHSCI. WHSCI is associated with diseases affecting growth and development, and is essential for embryonic development. We found that WHSCI and trimethylation of K36 is essential for H3.3 placement onto DNA in response to a certain signaling molecule, called interferon (IFN). Our analysis revealed that WHSCI facilitates the assembly of gene products from IFN-stimulated genes. This study sheds a new light on a previously unrecognized role of WHSCI, which links gene product assembly to H3.3 placement in activated genes.



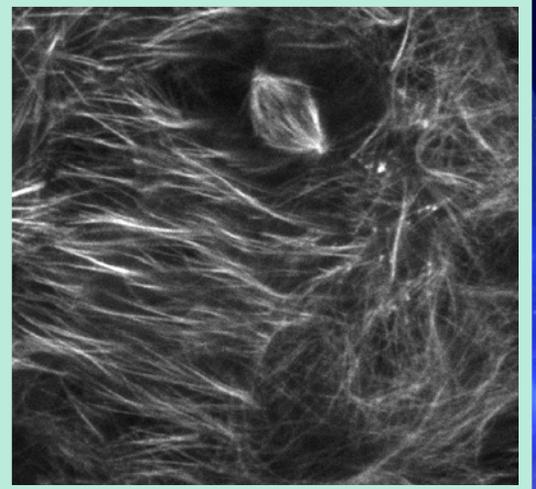
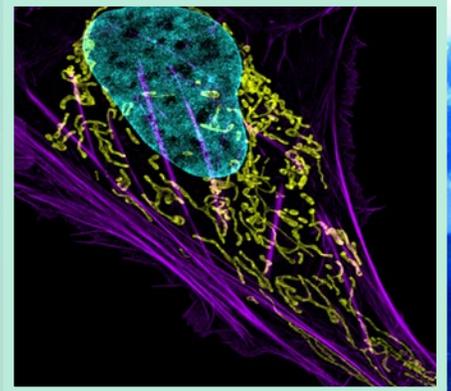
NICHD DIR Year in Review

We're near the end of another successful year in the NICHD Division of Intramural Research (DIR). Check out a few highlights below!

During 2013...

- » NICHD DIR researchers authored over 185 publications
- » Twenty-six NICHD fellows received the Fellows Award for Research Excellence
- » Dr. Schuyler van Engelenberg received NICHD fellow Mentor of the Year
- » Dr. Mary Dasso received NICHD investigator Mentor of the Year
- » Dr. Dylan Burnette of the Lippincott-Schwartz lab placed third in the Nikon 2012 Photomicrography Competition with his unique image of mitochondria (yellow), actin (purple), and DNA (blue) in a human bone cancer cell (osteosarcoma). He leaves in early 2014 for an academic position at Vanderbilt University. 
- » Eighteen NICHD graduate students presented posters at the NIH 9th annual Graduate Student Research Symposium. Students Chris Wassif of the Porter lab and Sisi Liu of the Stratakis lab received two of the 12 best poster awards, and NICHD graduate student Xuefeng Yin of the Storz lab was one of only four student oral presenters
- » Drs. Tatiana (Tanya) Rostovtseva and Owen Rennert received two of the three mentoring awards at NIH 9th annual Graduate Student Research Symposium
- » The NICHD DIR held the Ninth Annual Meeting of Postdoctoral, Clinical, and Visiting Fellows and Graduate Students at the beautiful National Museum of the American Indian
- » Dr. Valerie Virta won the 2013 Annual Meeting Image Competition with her image of microtubules in a dividing cell in a living embryo 

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NICHD DIR Year in Review

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- » Drs. Micah Hill and Gary Levy won first and second place awards, respectively, in the annual Bailey K. Ashford Walter Reed clinical and basic research competition for 2013—the first win for NICHD fellows since 1986!
- » Postbacs Brett Athans, Ankur Narain, Ankita Prasad, and Amy Ton received OITE Poster Awards at the 2013 NIH Postbac Poster Day. The three NICHD-selected "best postbac poster" winners for 2013 were Oghomwen Igiesuorobo, Joshua Lee, and Amy Ton
- » Dr. Kevin Francis won first place for his poster at the NIH Center for Regenerative Medicine (CRM)/Stem Cell Interest Group (SCIG) Stem Cell Research Symposium
- » Dr. Katherine Donigan received the American Society of Human Genetics (ASHG)/National Human Genome Research Institute (NHGRI) 2013 genetics and public policy fellowship award
- » Women Scientists Advisors (WSA) selected Dr. Sarah Cohen as one of the two WSA Scholars for this year

Please submit your accomplishments for publication in the newsletter throughout the year to Shana.Spindler@gmail.com.

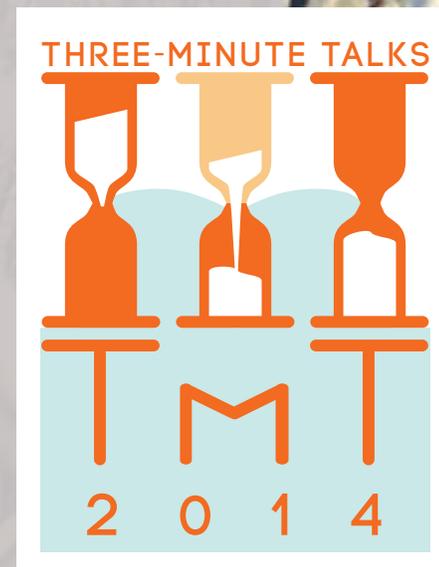
NICHD Science Communication Awards

THREE-MINUTE TALKS “TMT” COMPETITION

NICHD DIR is launching a competition among intramural trainees at the graduate and postdoctoral level, to communicate in three minutes or less the substance of their research and its significance to improving human health—in such a way that can be understood by a broad scientific audience.

Objectives:

- » Develop scientific communication skills among our trainees
- » Showcase intramural science to the general public, on the NICHD web site and in social media
- » Enhance awareness of the breadth and significance of NICHD research



Eligibility: For entry into the competition, fellows must electronically submit a 200-word (maximum) narrative about their three-minute research story and a submission form, no later than **January 10, 2014**, to yvette.pittman@nih.gov. Up to 25 DIR predoctoral and postdoctoral, visiting, and clinical fellows will be invited as semifinalists to compete for these science communication honors.

Awards: The competition winners will each receive recognition, the specifics of which still need to be finalized, for: 1st place, 2nd place, and people’s choice. For the people’s choice award, online ballots will be provided to the DIR community. In addition, the winning recorded presentations may be played at the annual NICHD BSC and/or Advisory Council meetings and used on other occasions, which will promote the research of our trainees.

Timeline: All accepted fellows must participate in the two “science communication” preparatory workshops, receiving professional oral presentation coaching. Led by public speaking coach, Scott Morgan, the interactive workshops will help fellows explain their work to a nonspecialized audience in a short timeframe.

The first workshop on January 30th will offer fellows tips on how to convey their science clearly and quickly, telling an interesting story with only one slide, using accessible language. On February 24th, fellows will practice their delivery, receiving individual feedback from Scott Morgan.

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NICHD Science Communication Awards (continued from page 19)

The three-minute talks of all accepted fellows will be judged by a distinguished NIH panel on March 11th, and 10 finalists will be selected to advance to the next stage of the competition. To improve the finalists' stage presence, eye contact, vocal range, and maintenance of a steady pace, they will participate in a video training workshop on March 28th led by Scott Morgan. Subsequently, the taping of all finalists will occur in mid-April, and online voting by an outside panel of judges will take place in May.

Rules: Fellows can use only a single PowerPoint slide with animations. However, no sound, props, or video files are permitted. Presentations cannot exceed three minutes, and must use only spoken words.

Judging Criteria:

- » Ability to clearly and succinctly describe a research project in language appropriate to a general scientific audience
- » Have a logical flow that captures and maintains the audience's attention
- » Spend adequate time on each element of the presentation, from description of the background to key results, including its relevance to human health
- » Enhance the presentation with a clear and concise PowerPoint slide

Questions should be directed to Yvette Pittman (Yvette.Pittman@nih.gov) in the Office of Education, DIR, NICHD.

December Announcements

SPECIAL CONGRATULATIONS TO DR. SARAH COHEN

At the recent FARE 2014 Awards Ceremony, Dr. Cohen was announced as one of the two WSA (Women Scientists Advisors) Scholars for this year. A panel of WSA institute representatives selects WSA Scholars from all female FARE winners. Congrats to Dr. Cohen on this wonderful recognition.

GRANT WRITERS: ERA UPDATE: THE ALL NEW ERA SITE NOW AVAILABLE

As of November 21, the new Electronic Research Administration (eRA) web site is available. You can access the new pages by going to <http://era.nih.gov>.

The new design is more graphical, focusing on processes. The less text heavy approach is designed to make the pages easier to navigate, and less overwhelming.

With the idea of making the site as useful as possible, applicant steps are broken down into major chunks and each chunk includes:

- » Links to the specific section of the online help, with step-by-step information on basic tasks
- » Policy links, where appropriate.
- » Communication resources, from fliers to presentations to FAQs.

Please send feedback on the new design to eracommunications@mail.nih.gov.

DEADLINE FOR NIH SUMMER MENTOR AWARD QUICKLY APPROACHING

The Office of Intramural Training & Education is looking for graduate students and fellows with a passion for mentoring/training students, a level of creativity for experimental design, and the patience and commitment to invest in a summer student's first NIH research laboratory experience! Application deadline is December 9 at 5 pm. For more information, please contact Yvette Pittman at Yvette.Pittman@nih.gov.

UPCOMING: THE NEW ENGLAND SCIENCE SYMPOSIUM

The New England Science Symposium, established in 2002, provides a forum for postdoctoral fellows; medical, dental and graduate students; post-baccalaureates; college and community college students (particularly for African-American, Hispanic/Latino and American Indian/Alaska Native individuals) to share their biomedical and health-related research activities through oral or poster presentations, to engage in discussions related to career development in the sciences, to exchange ideas and to expand their professional networks. It will take place on Sunday, April 6, 2014 at Harvard Medical School, and the online registration and abstract submission deadline is January 9, 2014. For more information, here is the web link: <http://www.NewEnglandScienceSymposium.org>

The NICHD-Office of Education would like to sponsor an intramural fellow to attend the New England Science Symposium. If you are interested and plan to submit an abstract, please send Yvette Pittman (yvette.pittman@nih.gov) an email by Friday, December 20.

December Events

TUESDAY, DECEMBER 3, 5:30 – 7 PM

NICHD Fellows Fall Social Event
Bethesda Naval Bowling Center (directly across from the
NIH campus)

FRIDAY, DECEMBER 6, 2 – 4 PM

OITE Holiday Party for Trainees

The staff of the NIH Office of Intramural Training & Education would like to invite you to attend our annual holiday gathering and celebration of the New Year. Enjoy light refreshments, network with other trainees, and talk with OITE staff members.

Building 10, FAES Terrace

Please [register](#) to attend by Wednesday, Dec 4.