



## GREETINGS FROM THE HEAD

Jie Chen

Dear CDB friends,

Welcome to the 2016 edition of the CDB newsletter! In these pages we celebrate the achievements of our faculty and students in 2015. You will also read the stories of two highly accomplished alumni.

A year ago we vowed to maintain our competitive research programs to withstand fiscal difficulties at both the state and federal levels. As drastic state budget cuts continue to loom, I am proud to report that 2015 was a highly successful year for CDB faculty to secure federal research funding. From the National Institutes of Health (NIH) came the first R01 grant for Assistant Professor Rachel Smith-Bolton, competitive renewals as well as new grants for several senior faculty members, and a center grant to recognize Professor Andy Belmont's cutting edge research program and leadership in the emerging field of the "4D Nucleome" (see story on page 2). Our faculty continue to make significant impact on collaborative research efforts across campus, as exemplified by Professor Martha Gillette's seminal contribution to several interdisciplinary initiatives on campus (see story on page 8).

Students in the department also continue to thrive. In a school-wide ceremony in May we recognized outstanding graduate and undergraduate students for their achievements in research, academic performance, and teaching (see page 7). Particularly noteworthy is the announcement of the inaugural Tunji Toogun Memorial Award to honor two CDB graduate students, Frank Echtenkamp and Nimish Khanna, for their outstanding research achievements. This award is created in memory of Tunji who was a PhD student in CDB at his untimely passing in 2007. Marking the opening of the Tunji Toogun Memorial Graduate Fellowship Fund, Tunji's friends in graduate school shared with us their memories of Tunji with photos and stories ([mcb.illinois.edu/departments/cdb/toogun](http://mcb.illinois.edu/departments/cdb/toogun)).

As President Obama's proposed NIH funding increase for FY2016 offers a long-overdue partial relief from the devastating NIH budget cuts in the past decade, we have reasons to be optimistic about building and maintaining excellence in biomedical research and innovation. At the same time, the CDB faculty are committed to our mission of delivering the highest caliber undergraduate education at Illinois. •

## SPRING 2016 NEWSLETTER

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### ABOUT THE NEWSLETTER

*The Cell and Developmental Biology Newsletter* is an annual publication of the Department of Cell and Developmental Biology in the School of Molecular and Cellular Biology at the University of Illinois, Urbana-Champaign. The newsletter is written by CDB faculty and friends, and is designed by MCB Communications.

Our alumni are important to us. We want to hear from you. Send us your latest news, and we'll include it in the next newsletter's CDB Family News. We also welcome articles and suggestions for future newsletters. Here's how to reach us:

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## BELMONT'S SIX YEARS AS HEAD STRENGTHENED THE WHOLE DEPARTMENT

Andy Belmont is a tall, craggy, and solitary figure striding across campus. If you don't know him (and sometimes even if you do) he can be intimidating. So it might be hard to imagine him as department head, which requires so much interaction and negotiation with others. Still, when the position of department head came open eight years ago, he was the choice of the faculty and, because he was very concerned about the department's future, he felt he had to take it.

"I wanted the department to be a place people would want to stay," he said.

He had strong opinions of what he thought should happen and told his wife, Chifan Cheng, so often.

"Then it's time to put your money where your mouth is," Cheng told him.

For example, Belmont was concerned that the graduate program would suffer.

"I wanted to protect and strengthen the graduate program," he says. "People stay here because of the quality of our graduate program."

In addition, there was a very large percentage of the faculty coming up for tenure in the next year or two. Five (out of a total of fewer than 20 total) faculty in CSB were going up for tenure the year he became head. Belmont knew how critical it is for departmental morale that tenure decisions go smoothly.

So, in true scientist mode, he researched the history of tenure in the department by reviewing all cases. He determined what made a good or a bad tenure case. Then he presented all his findings to the junior faculty.

"No one had ever done that," says Jie Chen, current department head.

Belmont also created a grant mentoring committee to coach junior faculty to write grants.

"It was hugely successful," says Chen, who served on the committee the first few years.

That committee is still going strong. Faculty members bring ideas and specific aims to the group and they have brainstorming sessions. All the members of the committee have been on NIH study sections, so they share their expertise and experience.

Chen credits Belmont's six years of leadership with really helping the department remain strong and get stronger.

"He personally mentored each of the junior faculty and we are all very, very grateful," she says. •

## "OUT IN LEFT FIELD" NO LONGER

### CORRELATION BETWEEN NUCLEAR ORGANIZATION AND GENOMIC FUNCTION

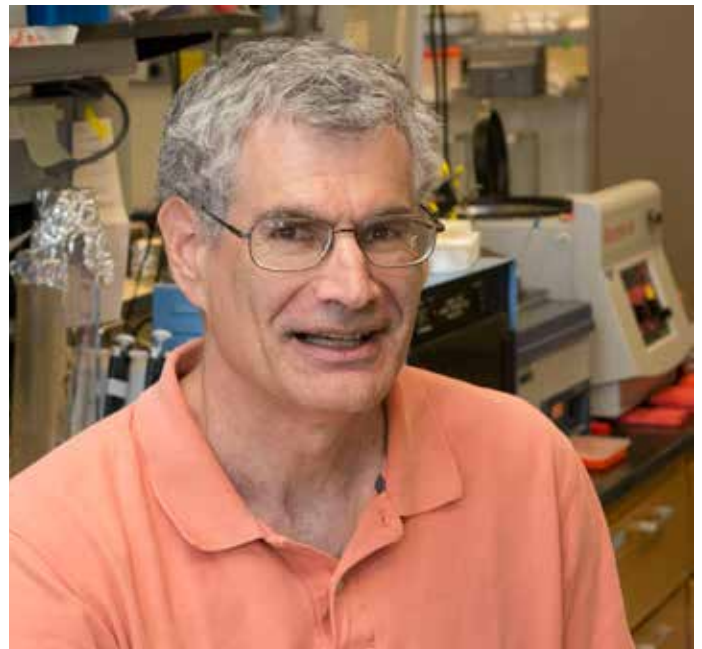
by Deb Aronson

Andy Belmont has spent the last 20 years "out in left field," studying an area—the highest levels of chromatin folding—not many people were interested in. But in a funny twist of fate, changes in the field and the route of his own research have intersected to put Belmont smack dab in the middle of the latest exciting obsession in biology.

Belmont and his team were recently awarded an \$8 million grant from NIH over five years to map the structure and function of nuclear chromosome organization. The grant is part of NIH's 4D Nucleome Program, which seeks to develop new techniques for mapping and imaging chromosome folding within the nucleus, studying the structure and function of nuclear bodies and processing the massive quantities of data the researchers will gather. Belmont's grant is one of six Center grants.

There are several new developments propelling interest in the field of chromosome and nuclear structure now, says Belmont. One is the realization that there are long-range interactions between regulatory sequences. The big surprise is that many mutations are not in protein coding parts of genes but are actually in regulatory regions. And many regulatory regions are not near genes they regulate but can be far away.

"You can have regulatory regions mega base pairs away from any known gene," says Belmont. "Who does that regulatory sequence regulate, how does that work? If you do have long-distance interactions, why does it interact with one gene and not another?"



The thinking now is perhaps the answer lies in exactly how chromatin folds into interphase chromosomes and the folding of these chromosomes within the cell nucleus.

"We were working for many years in left field because there was no obvious function to this level of organization," says Belmont. "Now the biology came to the idea that this level of organization might be important."

Belmont, a self-described "very visual person," finds great satisfaction looking at structures. "I like looking in the microscope and seeing a real structure directly—rather than guessing at the structure using indirect molecular methods," he says. "I was happy just to look at structures."

While looking through the microscope as a post-doc in the 1980s, Belmont observed a very thin loop sticking out of a DNA ultrastructure. He thought that structure might be interesting, but needed a way to tag it that didn't mangle the DNA ultrastructure. So, upon arriving at Illinois, his lab developed a technique that, when combined with a fluorescent protein, enabled him to see the locus of interest under a light microscope.

As Belmont used this technique to look at chromatin structure, he made a startling

discovery: the structure of the chromosomes they looked at was highly correlated somehow to the location of that chromosome within the nucleus, whether at the periphery, or close to a particular type of nuclear body, for example.

“Initially we were not interested in that finding, but we kept repeatedly getting hit over the head with it,” says Belmont.

“We started to see different locations that correlated with different structural and functional chromosome properties. It turns out that the chromosome tagging method we developed to preserve structure was also a method we could use with live cells. The major novelty has been the ability to watch chromosome behavior in live cells.”

Once Belmont realized that not only did location correlate in some cases with transcriptional activation, but that he could watch specific chromosome regions moving from one region of the nucleus to another during this activation process, he became very interested in understanding what functional role chromosome movement and location played in transcriptional regulation.

At first, however, Belmont’s results — that chromatin moved unidirectionally over significant distances to different nuclear regions during the transcriptional activation process — were met with skepticism.

One reviewer, in rejecting a paper, wanted to know if all genes move. Belmont, in his dry manner, says he wrote back, “If I saw an oak tree pull itself up by its roots and run down Lincoln Ave., I would think that was newsworthy. I wouldn’t be asking whether every oak tree did this, and, for that matter, whether every maple or ash tree also did it. I would say it was pretty amazing to see a significant fraction of oak trees run down the street and that it would be a safe bet that other people would be interested in this as well.”

Next, Belmont became interested in finding a method through which he could map all chromosome locations throughout the nucleus at once. That way he could look at cells differentiating or responding to a physiological stimulus to ask how many genes within the entire genome changed their location within the nucleus as they turned on or off. Existing techniques could tell when something was touching a specific protein that was enriched in a particular nuclear compartment, but not how far away this chromosome region was from this compartment as you could observe in a microscope.

Belmont dreamed of developing a genomic method to measure chromosome location within the nucleus. Could they then go back to the microscope and directly image those chromosome regions that were predicted to move during some type of biological transition as genes in these regions turned on or off?

“We were looking for a way to translate what we were seeing in the microscope to a genomic signal, so instead of studying one gene at a time we could study all the genes,” says Belmont.

Happily for Belmont, graduate student Yu Chen took on this project, working for five years before her first positive result. The method, “tyramide signal amplification,” depends on a labeled tyramide free radical generated by an immunostaining procedure. This free radical diffuses and crosslinks to DNA. The amount of DNA labeling falls off with distance from the source of the free radical, providing information about how far away the labeled DNA is from the target protein that is immunolabeled. Chen was able to tune the method to control how far the label diffuses and also figured out how to translate the observed genome signal into actual distances.

Just about then, NIH announced their 4D Nucleome project, which aligned perfectly with Belmont’s interest. He went from “working in left field” to being in the center of the latest big thing.

Belmont frequently acknowledges that he could not have done this work without the numerous collaborations, particularly on campus. Those collaborations, at IGB and elsewhere, played a strong role in keeping Belmont here when Northwestern tried to hire Belmont two years ago. Northwestern had a stronger microscopy group, better imaging facilities and nuclear structure group. They wanted Belmont to join their cohort. But Belmont felt like his collaborations here with both biologists but also non-biologists-physicists and engineers- were of more value. For example, his collaboration with Dr. Jian Ma’s group in the Department of Bioengineering enabled Yu Chen and he to properly interpret and analyze the results of the TSA-Seq experiments. Plus, the way the finances were structured at Northwestern would have required him to have a much smaller group with fewer graduate students and to do more conservative, “safer” work.

“I like the idea of taking chances,” says Belmont. “Having a larger group allows you to roll the dice more times, and graduate students are more willing to roll the dice and

take that chance. Plus there is a safety net here that lets you do riskier things.”

“It was a big coup, keeping Andy here,” says current head Jie Chen. “It says a lot about his loyalty and also how much he values his colleagues.”

And Chen made sure that, as part of his retention packet, Belmont was able to get some of the facilities, like an upgraded microscope and high pressure freeze system that he admired at Northwestern. •

## PICTURE THIS

### ANNE CARPENTER’S POPULAR IMAGING TOOL MEASURES A MULTITUDE OF CELL FEATURES

by Doug Peterson



Anne Carpenter’s task was not as straightforward as it sounded. She was trying to measure the size of *Drosophila* fruit fly cells, but she was continually frustrated by the bottleneck in the processing of cell images.

It was the mid 2000s, and the problem was that commercial software for doing high-throughput image processing “was choking on the type of images we had created and doing a terrible job of quantifying cellular features,” says Carpenter, who received her PhD in cell and developmental biology from Illinois in 2003 (under the name Anne Nye). “The *Drosophila* cells are not as nicely shaped or as uniform as mammalian cells.”

So she took the matter into her own hands. Carpenter decided to write her own software code to solve the imaging problems she faced at the time while doing postdoctoral work at the Whitehead Institute for Biomedical Research in Massachusetts. The result eventually became CellProfiler, an extremely popular and successful open-source software program that biologists around the world now use.



CellProfiler is launched more than 125,000 times each year, and it has been cited in more than 3,000 scientific papers published since the software was released in 2005. The tool can be used for all types of imaging work, such as correcting illumination patterns; identifying cells, yeast colonies, and other biological objects; or measuring features such as the size and quantity of specific cells.

Since 2007, Carpenter has been director of the Imaging Platform at the Broad Institute, a joint venture between Harvard and MIT. In addition to offering the use of CellProfiler to biologists all over the world, her lab collaborates directly with many researchers using the software.

For instance, Carpenter's lab collaborated with John Crispino from Northwestern University, who recently found a promising drug treatment for AMKL leukemia—a cancer that primarily affects children. Carpenter used CellProfiler to measure the DNA content of individual cells, which was key to the discovery.

Crispino's lab identified a drug that causes the leukemic cells to become polyploid. This results in the cells having excess DNA, which the body recognizes as abnormal. The body then uses its own mechanisms to destroy the cancer cells.

A clinical trial opened last October to test the drug in AMKL patients.

Carpenter's venture into imaging technology actually began when she was a PhD student in CDB professor Andrew Belmont's laboratory at Illinois. The lab was interested in figuring out whether particular transcription factors caused chromatin to unfold or not, but the technology available back then made the work "incredibly tedious," she says.

Belmont had just acquired a new robotic microscope, so Carpenter spent a summer programming the equipment to do the image analysis automatically, cutting out the tedium and speeding the process.

"My experience in Andy Belmont's lab was a turning point for me," Carpenter says, "because that's when I became interested in developing systems to automate biology.

"If you had told me in high school or even college that eventually I would be leading a group of software engineers and

computer scientists at Harvard and MIT, I would be utterly confused and surprised," Carpenter adds. She didn't even know she would be going into a scientific field until her first year at Wheaton College in the Chicago area. Growing up on a farm in northwestern Indiana, she was a bookworm with little interest in science. In fact, when her grandfather gave her a science kit, she never used it once, and by deferring to lab partners, she went through high school and college avoiding the dissection of anything larger than a fruit fly. But during her first year at Wheaton College, she enjoyed her science classes so much that she transferred to Purdue to get a bachelor's degree in biology.

Imaging and microscopy have come a long way since those days. Before the 1990s, she says biologists didn't often capture and measure images; they looked through their

microscopes and judged the characteristics of cells subjectively by eye.

In the 90s, things began to change, as the digital capture of cell images emerged. Then, in the 2000s, during her PhD and postdoc years, automated image analysis tools exploded on the scene. So she has ridden the wave of technological change in image analysis from the beginning, and CellProfiler has been part of that transformation.

CellProfiler has been used in research on all sorts of diseases, including Ebola, tuberculosis, and cancer. In another leukemia research project, Carpenter's laboratory is working with Todd Golub, also with the Broad Institute, in targeting cancer stem cells.

"In many cases of cancer, it's possible to kill the tumor cells, but it's very hard to kill the last tiny percentage of the cells—the cancer stem cells," she says. These stem cells often trigger the recurrence of cancer in patients.

In this project, researchers succeeded in finding a drug candidate that could kill the leukemic stem cells but leave the bone marrow stem cells safely intact. Carpenter's lab used CellProfiler to distinguish between leukemic stem cells, which have a cobblestone appearance, and bone marrow cells, which are more rounded.

Over the past 10 years, her lab has amassed a huge collection of images, but she says there remains a lot of information that can be extracted from them. Every single cell has thousands of features that can be measured. Therefore, one branch of her lab is harvesting this information from the cell images in their collection.

She says there are a myriad of uses for this kind of information. For instance, one possibility is to create a profile, or fingerprint, of cells' responses to treatments with various compounds.

Researchers can then use this profile to screen compounds for toxicity to cells in the liver, heart, or other organs.

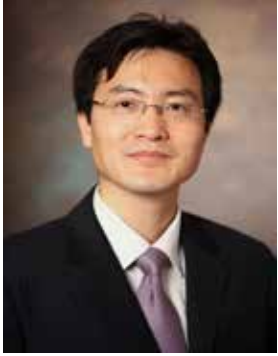
Since the time that Carpenter first created the CellProfiler software in the mid-2000s, the program has been rewritten and improved by her laboratory's professional

software engineers. But one reason CellProfiler has been so successful, she says, is that it "was originally designed from the ground up by a cell biologist who was trying to accomplish something in a real project. It started out as a bit of code that I needed for my project, but grew into a multifunctional toolbox that is changing the world."

Carpenter received the NSF CAREER award in 2012, and has received recognition and research funding from numerous other groups including the NIH, the Human Frontiers in Science program, and the Howard Hughes Medical Institute. She was named Young Leader of the French-American Foundation, and elected fellow of the Massachusetts Academy of Sciences. She was featured in a public television special, "Bold Visions: Women in Science & Technology," and named a "Rising Young Investigator" by Genome Technology magazine. CellProfiler was awarded the Best Practices Award by Bio-IT World in 2009. •



## CHANGING THE CULTURE OF DISCOVERY



### IN-HYUN PARK GETS AT THE GENETIC ROOTS OF RETT SYNDROME USING IPS CELLS

by Doug Peterson

Children with Rett Syndrome develop normally for about 18 months, but then the signs begin: abnormal breathing, a regression in speech, and some loss of the use of their hands. The growth of

the head slows down, and the children begin to show autistic-like symptoms.

Rett Syndrome is a disorder that almost exclusively strikes female children, says cell and developmental biology alumnus In-Hyun Park, associate professor of genetics at Yale Medical School. Rett Syndrome is triggered by a mutation in the MeCP2 gene, found on the long arm of the X chromosome, and it is the second most common cause of mental retardation in females, he says, affecting about 1 in 10,000.

Park runs a laboratory in the Yale Stem Cell Center, and he is helping to lead the genetic charge against Rett Syndrome, developing an effective in vitro model of the syndrome using “induced pluripotent stem cells,” or iPS cells, which have revolutionized stem cell research.

In fact, his lab was among the earliest to reprogram human somatic cells to create the iPS cells so important for research on all kinds of diseases. Like embryonic stem cells, iPS cells have the ability to differentiate into many types of cells—muscle, nerve, blood cells, and more. But they don’t carry with them the controversy of embryonic stem cells.

When Park finished his PhD at Illinois in 2005 and went to Harvard Medical School for his postdoctoral work in the lab of George Q. Daley, the debate over embryonic stem cell research was grabbing headlines across the country.

“Deriving new embryonic stem cells using federal funding was banned because to obtain these stem cells, you have to destroy human embryos,” Park explains. “They only allowed using a few lines of human embryonic stem cells.”

He became part of the search to find ways to create “embryonic stem-like” cells. This meant hunting for techniques to reprogram somatic cells to behave like embryonic stem cells, which theoretically can generate any cell type within the human body.

Then, in 2006, Japanese researcher Shinya Yamanaka became the first to reprogram fibroblasts—cells of connective tissue—to create iPS cells. The expression of four transcription factors reset the fibroblasts to an embryonic stem-like state. For this discovery, Yamanaka shared the 2012 Nobel Prize in Physiology or Medicine.

Reprogramming a patient’s own somatic cells doesn’t just bypass the controversy over embryonic stem cells; there is also less risk that

the cells will be rejected by the body’s immune system. The ultimate goal is to use iPS cells in cell replacement therapy—replacing defective cells with healthy ones. But because the use of iPS cells in cell therapy still has biosafety issues that need to be resolved, the more immediate use for them is with in vitro modeling of diseases.

The idea is to create iPS cells that carry genetic diseases and can then be studied in vitro—the kind of work that Park has been doing with Rett Syndrome since establishing his lab at Yale in 2009.

“Cell culture has been the backbone of basic biomedical research for many decades,” he says. But iPS cells expand the cell culture possibilities. As he puts it, reprogramming technology “now provides an unprecedented approach to study Rett and indeed other diseases.”

In addition to Rett Syndrome, iPS technology has been used to model disorders such as Alzheimer’s disease, Parkinson’s disease, Becker muscular dystrophy, and amyotrophic lateral sclerosis, or ALS.

Park notes that human iPS cells are close to 99.9 percent equivalent to human embryonic stem cells. However, iPS cells retain some memory of the parental cell, which means that when they are developed from blood cells, they will retain some epigenetic memory of the parental blood cells. But as Park says, “I don’t think that will affect the future utility of human iPS cells.”

He points out that reprogramming somatic cells into iPS cells will also help researchers acquire cellular material that would normally be difficult to obtain—such as cortical brain neurons.

Park grew up in Suwon, South Korea, about 19 miles south of Seoul. He came to the United States for the first time when he arrived on the Illinois campus during the summer of 2000. Coming from an extremely crowded city in a geographically small country like South Korea, he says he was initially shocked by the open spaces of Central Illinois—especially on a large campus with few students present during the summer.

“It was a big school, and nobody was around,” he recalls. “It seemed very strange.”

But perceptions quickly change. Park says that after spending five years at Illinois, he went to Boston to do his postdoctoral work and “it was too many people.”

Park credits his PhD mentor at Illinois, Jie Chen, for laying the groundwork for his career, and he’s grateful to the CDB program for exposing him to many research areas, from cellular signaling pathways, epigenetics, and cytoskeletons to yeast genetics, neuroscience, and *Drosophila* genetics.

He also has fond memories of playing basketball regularly at the Activities and Recreation Center, and once he even squared off against Illinois basketball standouts Warren Carter and Deron Williams, who went on to become an NBA star. He says the one time he faced Williams on the court, no one could stop the Illini point guard, and Williams scored at will.

But Park likes challenges, especially those in the lab. For instance, when he was first trying to develop human iPS cells, it took about 24 trials to succeed. “But now, after seven or eight years, it’s become standard protocol,” he says.

“It can be difficult, but having to overcome challenges is all right when you like the research,” Park adds. “Most scientists do science because they like challenges.” •

## TUNJI TOOGUN MEMORIAL GRADUATE FELLOWSHIP FUND



The Department of Cell and Developmental Biology is pleased to announce the first recipients of the Tunji Toogun Award: Nimish Khanna, postdoctoral fellow in the Department of Biological Sciences, UCSD; and Frank Echtenkamp, postdoctoral fellow at Technische Universität München, Germany. Both graduated with PhDs in 2015.

This award was created in memory of a PhD student who tragically passed away in 2007. He was an energetic and dedicated graduate student, and this award will recognize students who reflect the spirit of Tunji Toogun.

Tunji Toogun, a Cell and Developmental Biology PhD student at the University of Illinois at Urbana-Champaign, died August 3, 2007 at the age of 26 after falling into Lake Shelbyville and drowning. Within his career, Tunji was best known for his hard work and boundless enthusiasm for research and learning.

Born in Nigeria on October 15, 1981, Toogun studied at the University of Illinois at Urbana-Champaign receiving his B.S. in 2001 and a posthumous PhD in 2007. Tunji first arrived to UIUC as an incoming freshman in 1997 at the age of sixteen to begin a long and fruitful academic career. He left an immediate impact on those around him with his strong Nigerian accent and his always friendly and persistent demeanor.

Friends and teachers of Tunji characterized him as a bright, kind, and enthusiastic individual who was also a great friend.

A year after Tunji's death, a fund was established in his memory, with contributions from Tunji's friends, classmates, and faculty. The Department of Cell and Developmental Biology has decided to use this fund to recognize and support outstanding graduate students in our program in the form of awards and potential fellowships. The Tunji Toogun Memorial Graduate Award will be offered annually to a CDB graduate student at any stage of the graduate program for his or her outstanding research accomplishments. •

## CDB FACULTY NEWS 2015

### Extramural grants newly awarded to CDB faculty in 2015

#### Andrew Belmont

National Institutes of Health, U54 4D Nucleome Center grant  
"Combined cytological, genomic, and functional mapping of nuclear genome organization" (PI: Belmont)

#### Jie Chen

National Institutes of Health, R01 grant "mTOR signaling in myogenesis"

Keck Foundation grant

"Aminoacyl-tRNA synthetases: evolutionary scaffolds to novel biology and physiology" (PI: Martinis)

#### Brian Freeman

National Institutes of Health, R01 grant "The Hsp90 molecular chaperone system"

#### Martha Gillette

Abbot Center for Nutrition, Learning and Memory grant  
"Diet-modified neuron physiology assessments" (PI: Gillette)

National Science Foundation, Science and Technology Center grant  
"Emergent behaviors of integrated cellular systems" (PI: Kamm, MIT)

National Institutes of Health, U01 Brain Innovative grant  
"Integrated multimodal analysis of cell and circuit-specific processes in hippocampal function" (PI: Sweedler)

#### Jon Henry

National Institutes of Health, R01 grant  
"Cell and molecular biology of cornea epithelial stem cells"

#### Phil Newmark

National Institutes of Health, R21 grant "Hymenolepis diminuta as a model for studying stem cells in parasitic flatworms"

#### K. Prasanth

National Institutes of Health, supplement to R01 grant "Characterization of nuclear retained RNA-mediated gene regulatory mechanism"

#### Supriya Prasanth

National Institutes of Health, supplement to R01 grant "Role of ORCA in DNA replication"

#### Mary Schuler

National Science Foundation grant "Genetic parallelism and allele reuse in herbivore plant chemical phenotype matching" (PI: Berenbaum)

#### Rachel Smith-Bolton

National Institutes of Health, R01 grant "Regulation of cell fate and patterning during regenerative growth"

#### Lisa Stubbs

National Institutes of Health, R21 grant "A mouse model for AUTS2-linked neurodevelopmental disorders"



## STUDENT AND POSTDOC NEWS 2015

### Edwin Arauz awarded PhD, August 2015

Thesis title: "BIOCHEMICAL AND SINGLE-MOLECULE ANALYSIS OF SIGNALING MOLECULES"

Current position: Postdoctoral fellow with Professor Anthony Kossiakoff, University of Chicago

### Chase Bolt awarded PhD, May 2015

Thesis title: "REGULATORY DYNAMICS OF THE TBX 18 LOCUS IN MOUSE UROGENITAL DEVELOPMENT"

Current position: Postdoctoral Fellow with Prof. Denis Duboule, at the École Polytechnique Fédérale de Lausanne (EPFL) in Lausanne, Switzerland

### Yijie Geng awarded PhD, August 2015

Thesis title: "A CHEMICAL BIOLOGY STUDY OF HUMAN EMBRYONIC STEM CELL PLURIPOTENCY AND DIFFERENTIATION"

Current position: Postdoctoral fellow with Professor Randall Peterson, Harvard Medical School/Massachusetts General Hospital

### Lisa Moore awarded PhD, May 2015

Thesis title: "FGF SIGNALING IN XENOPUS LAEVIS LENS REGENERATION"

Current position: Medical student, College of Medicine, UIUC

### Min Zeng awarded PhD, May 2015

Thesis title: "INVESTIGATIONS INTO THE ROLES AND MOLECULAR MECHANISMS OF LANC-LIKE PROTEINS"

Current position: Adjunct Faculty, Ivy Tech Community College

**Mahdieh Jadaliha** was one of three inaugural awardees of the UIUC-Carle Cancer Scholars for Translational and Applied Research (C\*STAR) program, a graduate education program that fosters translational cancer research.

**Dr. Aamira Tariq**, a visiting scholar in the laboratory of K. Prasanth, received a Fulbright Scholar award.

**Dr. Vidisha Tripathi**, formerly postdoc in K. Prasanth laboratory, started her position as an assistant professor in January 2015 in the National Center for Cell Science, University of Pune, India.

**Dr. Bo Wang**, formerly a postdoc in the Newmark lab, started his position as an assistant professor in May 2015 in the Department of Bioengineering at Stanford University.

## HIGHLIGHTS OF RECENT RESEARCH BY GRADUATE STUDENTS

### Ambika Nadkarni (Briehner Lab)

Disassembly of cytoskeleton is required for cell motility and division. Cofilin, a vital severing protein necessary for actin disassembly, depolymerizes filaments most effectively at low cofilin to actin ratios. Ambika demonstrates that Aip1, a previously thought to be a cofilin-dependent actin capping factor, aids cofilin-mediated disassembly by binding to the sides of actin filaments to promote rapid depolymerization. Aip1 is able to depolymerize actin filaments even at saturating concentrations of cofilin such as in thymus. This led to a revision of the existing model and attributed novel functions to Aip1. [*Current Biology* 24:2749, 2014]

### Phil Kenny and Miri Kim (Ceman Lab)

Fragile X Syndrome (FXS) is the leading cause of inherited intellectual disability caused by loss of the Fragile X Mental Retardation Protein (FMRP). In the brain, FMRP regulates localized protein synthesis in neurons, necessary for maintaining synaptic plasticity and dynamics at dendritic spines. Phil, Miri, and collaborators have shown that FMRP is able to facilitate or suppress the translation of a subset of its target mRNAs through its association with the RNA helicase MOV10. This research has led to the identification of a functional partner for FMRP, a potential therapeutic target for FXS. [*Cell Reports* 9:1729, 2014]

### Christina Plaisier-Rosenberger (Chen Lab)

The mammalian target of rapamycin complex 1 (mTORC1) integrates a variety of intra- and extra-cellular signals to control cell growth. In collaboration with Dr. Mee-Sup Yoon and others, Christina has discovered that the endogenous inhibitor DEPTOR is rapidly and temporarily dissociated from mTORC1 upon mitogenic stimulation, and that this regulation is dependent on phospholipase and the signaling lipid phosphatidic acid. These findings reveal a novel mechanism of mTORC1 regulation in growth control that may be exploited in anti-cancer strategies. [*Molecular Cell* 58:549, 2015]

### Sumanprava Giri (S. Prasanth Lab)

Organizing eukaryotic genome into active euchromatin and inactive heterochromatin is crucial for maintaining cell identity and fate. Suman and collaborators identified the Origin Recognition Complex Associated (ORCA) protein as a scaffolding factor involved in assembling histone H3 lysine 9 methyltransferases (H3K9 KMTs), enzymes that tag repressive histone marks on heterochromatin. Depletion of ORCA resulted in alteration of chromatin architecture and loss of histone marks at specific chromatin sites. This study unveiled an extremely interesting crosstalk between DNA replication machinery and repressive H3K9 KMTs. [*eLife* 4:e06496, 2015]

### Keaton Schuster (Smith-Bolton Lab)

The Smith-Bolton laboratory utilizes a nonsurgical method for inducing tissue damage in *Drosophila* wing primordium to study tissue regeneration. A variety of signal transduction pathways are activated during tissue repair, and Keaton shows that one of the signaling pathways called Jun N-terminal Kinase (JNK) signaling can cause unwanted side effects by misregulating cell fate genes. Keaton identifies the gene *taranis* to play a critical role in suppressing these JNK-induced cell fate changes without interfering with JNK signaling activity, elucidating a mechanism by which tissue employs protective factors that aid in regeneration. [*Developmental Cell* 34:119, 2015]

## CDB STUDENT AWARDS 2015

### Outstanding Teaching Assistant Award

James Kemp

### Undergraduate Research Achievement Awards

Ralph Claveria

*Dr. Mary Schuler*

Muhammad Ilyas

*Dr. Lisa Stubbs*

Mabel Seto

*Dr. Rachel Smith-Bolton*

### Outstanding Undergraduate Research Achievement Award

Clara R. Stelman

*Dr. Phillip Newmark*

### Roderick MacLeod Award for Academic Excellence

Clara R. Stelman

*Dr. Phillip Newmark*

### Oyetunji A. Toogun Memorial Awards

Frank Echtenkamp

*Dr. Brian Freeman*

Nimish Khanna

*Dr. Andrew Belmont*

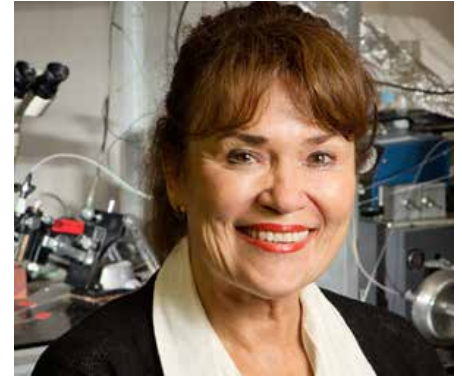
## MARTHA GILLETTE AND COLLABORATORS RECEIVE TWO GRANTS TO STUDY THE BRAIN

Martha Gillette, Professor of Cell and Developmental Biology at UIUC, and colleagues are collaborating on various projects involving brain imaging and the creation of biological machines in hopes of creating new diagnostic and therapeutic tools.

The work is facilitated by two grants that Gillette is a part of: the Emergent Behaviors of Integrated Cellular Systems (EBICS), which received \$25 million in National Science Foundation (NSF) renewal funding for the next five years and the National Institute of Health (NIH) BRAIN Initiative grant which has received more than \$2 million in funding over three years.

The goal of the EBICS project is to build living, multi-cellular machines to solve environmental, health, and security problems. These “biological machines” will serve as a basis to deliver drugs more effectively, function as internal diagnostic tools, or as contaminant sensors in the field. Gillette’s group focuses on developing neuronal circuits to provide sensing and processing for the cells.

The (NIH) Brain Research through Advancing Innovative Neurotechnologies (BRAIN) Initiative works towards developing tools to characterize and analyze the brain at the cell and even subcellular levels to show how individual cells and neural circuits interact with each other in time and space. Gillette currently works in Beckman’s NeuroTech Group and studies the brain’s plastic responses to experience, investigating signals that shape and wire the nervous system. •



<https://bioengineering.illinois.edu/news/new-life-ebics-bio-machines-project>  
<http://beckman.illinois.edu/news/2015/10/nih-brain-sweedler>

### OYETUNJI A. TOOGUN MEMORIAL GRADUATE FELLOWSHIP FUND

Our graduate program is critical to the research enterprise as well as the educational mission of the department, the success of which relies on the excellence and dedication of the faculty, and financial support of students on research assistantships. We hope that our alumni and friends will help us enhance our graduate program. Your donation of any size will be greatly appreciated.

#### I would like to make the following contribution to CDB

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