

## The Jane Coffin Childs

MEMORIAL FUND FOR  
MEDICAL RESEARCH

### JCC Research Highlights

*In this issue, we feature high profile research from four current fellows.*

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#### Friend or Foe?

*JCC fellow June Round uncovers how our immune system recognizes gut bacteria as harmless—or not.*

At first, nothing about the blue, green, and black microscopy image appears remarkable, but upon closer inspection, something emerges—something that hints at a much bigger story. “It wasn’t what we were looking for,” says JCC fellow June Round.

The image shows fluorescent labeled gut bacteria deep within the intestinal crypts of a mouse—not merely in the spacious lumen where most

microbes reside. “The closeness of this association highlights that an active communication is occurring between the bacteria and their host,” says Round. But what sort of communication?

It’s a question that has been part of Round’s research since 2007, when she joined Sarkis Mazmanian’s California Institute of Technology lab to pursue her interests in immunology and microbes.



*June Round*

CONTINUED ON PAGE 2 . . .

#### Full Potential

*JCC fellow Effie Apostolou identifies key gene cluster in stem cells.*



*Effie Apostolou*

Embryonic stem cells have the potential to become just about any cell in an organism—or, under the right conditions, a whole organism in and of itself. But what about cells that have already differentiated?

Some can be induced into an embryonic-like state, and they are considered a promising source for regenerative medicine, since their derivation is technically simple and ethically

uncontroversial. But in many experiments, their development potential is limited. These induced pluripotent (iPS) cells might express genes abnormally, and creating whole animals with them is much more difficult than using embryonic stem cells. Whether or not iPS cells are equivalent to embryonic stem cells has been a contentious question among scientists.

CONTINUED ON PAGE 2 . . .

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JUNE ROUND CONTINUED FROM PAGE 1

“Within the gut, there are tons of immune cell types waiting to attack pathogens,” she explains. But we live in harmony with many beneficial bugs. How does our immune system know the difference? She set out to answer this question by studying a common friendly gut bacterium, *Bacteroides fragilis*.

Round’s work hinges on a sugar molecule known as polysaccharide A (PSA), made by *B. fragilis* and found on its capsule surface. Early in her postdoc, Round found that PSA is integral to gut health. Without it, mice develop inflammatory bowel disease, often a debilitating precursor to colon cancer.

But replacing PSA cured the problem. She published the results in a 2008 *Nature* paper.

Next, she began a series of experiments to sort out the molecular mechanism behind this host-microbe interaction. She found that one set of immune cells recognized the sugar molecule and suppressed additional immune cells from mounting an attack against *B. fragilis*. If she removed the sugar molecule, the immune system attacked the friendly bacteria. If she removed the immune cell’s receptors that recognize the sugar molecule, the immune system attacked.

And, if she removed the first set of immune cells themselves, the immune system attacked.

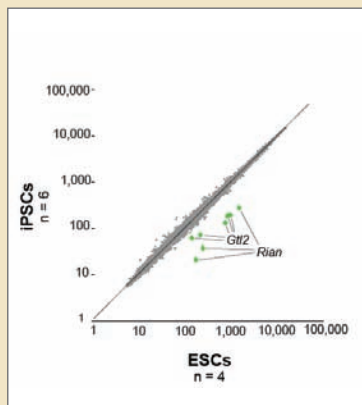
The bacteria had not changed, but the immune system could no longer recognize them as harmless.

For scientists in the field, the stunning result was in the details. The type of receptor that recognizes PSA is known as a toll-like receptor. Until Round published her research, toll-like receptors were only known to launch an attack against harmful bacteria. But her work showed that the receptors could also induce a regulatory response, essentially turning off an inflammatory reaction. The

results appear in a 2011 *Science* paper.

Round says that one of her biggest challenges throughout the work was not related to the research itself. Instead, she had to work especially hard to be successful while raising her two children, ages six and two. She’s grateful that the JCC offered a childcare stipend so that she could afford daycare and live close to lab, enabling her to frequently go back and forth between home and work. Her efforts have paid off. Now, she is moving on from her postdoctoral fellowship to join the faculty at the University of Utah. \*

EFFIE APOSTOLOU CONTINUED FROM PAGE 1



Scatterplot showing that the expression profile of iPS cells is extremely similar with the one of ES cells with the exception of two transcripts indicated in green.

JCC fellow Effie Apostolou began working on the question three years ago, when she joined Konrad Hochedlinger’s lab at Harvard Medical School. Her findings culminated in a 2010 *Nature* paper.

Back in 2009, she had just arrived in the US from Greece and didn’t know much about stem cells. Her colleague (and co-lead author on the 2010 paper) Matthias Stadtfeld had developed a system to create genetically identical embryonic induced pluripotent stem cells. Thorough analyses revealed that the two types of stem cells expressed genes almost identically in tests. Almost.

Apostolou, armed with her extensive knowledge of chromatin analysis, and Stadtfeld began to tackle the problem together. Two genes (and several microRNAs) on chromosome 12 were aberrantly silenced in the majority of iPS cells. Information about the genes was scarce in the literature, explains Apostolou, and it might have been convenient to ignore the

finding as some kind of artifact of the experimental techniques. “But the data were so clear, so beautiful, that we had to work with it,” she says.

In experiments, mouse embryonic stem cells could be coaxed into creating whole new animals, but only some iPS cells could do the same. The successful iPS cells expressed genes normally, including the two genes on chromosome 12. And, in one case, iPS cells with the two genes silenced could be forced into normal expression with the addition of a histone deacetylase inhibitor.

The research shows that the two genes play an important developmental role. Plus, the two types of stem cells are more similar than many people originally thought, opening the door for more uses of iPS cells—a boon to clinical applications, since iPS cells are easier to collect and they sidestep ethical issues surrounding embryonic cells. This is a big deal,



Whole mice generated by tetraploid complementation using iPS clones having normal expression of the *Gtl2* locus. The “all-iPS” pups express constitutive GFP.

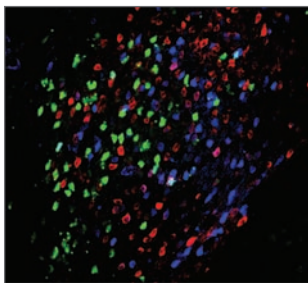
according to Apostolou, because now researchers can screen their iPS cells for normal gene expression and choose only those most suitable for further work.

Apostolou says that working on this project was a gift. She jumped into stem cell research from another field and feels that she has learned so many things in such a short amount of time that completing the work was like earning a second PhD. “The whole experience was just amazing,” she says. \*

## Fear in the Balance

*A delicate balance between two types of neurons controls fear, according to recent work by JCC fellow Prabhat Kunwar.*

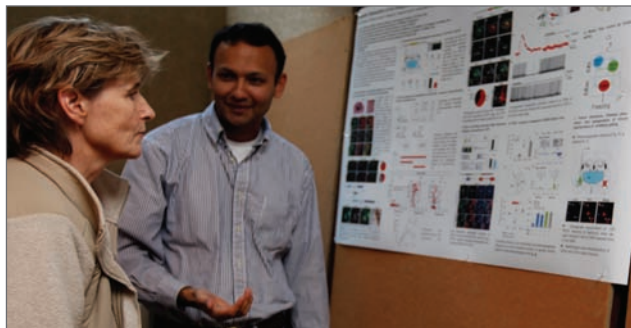
A pounding heartbeat, quick breathing, sweaty palms—we all know these common responses to fearful situations. The signal that initiates fear in our bodies begins in a structure of the brain known as the amygdala. But what exactly happens between individual neurons? Recent work by JCC fellow Prabhat Kunwar begins to untangle the details.



*This picture shows three largely non-overlapping inhibitory neuronal populations in the central amygdala. Activity in this local neuronal circuit gates learned fear.*

“It is very exciting that we are in a stage where we can explain complex behaviors such as fear at the level of neurons,” says Kunwar, a postdoc in David Anderson’s lab at the California Institute of Technology. “This work is just a beginning but a very important step. In our case, we identified a microcircuit of two opposing neuronal populations in the amygdala that act like a seesaw to control learned fear.”

The work hinges on genetic markers that help distinguish different cell types within the amygdala. Kunwar fluorescently marked different populations of neurons within the mouse brain, in order to map their connectivity and manipulate their activity. The research, published in a 2010 issue of *Nature*, was the result of a collaboration between



*Prabhat Kunwar discusses his poster with Dr. Ruth Lehman at the 2010 JCC Symposium.*

scientists at CalTech and the Friedrich Miescher Institute in Basel, Switzerland.

The work is part of Kunwar’s interest in the genetic dissection of neuronal circuitry underlying defensive and offensive behaviors in mice. Kunwar is originally from a small town in Nepal and moved to the United States to

advance his education. “I considered scientific research early on, as I realized its power both to explain the natural world and our existence, and to bring practical benefits to society,” Kunwar says. “Soon, I became captivated by the spectacular progress in genetics and biomedical sciences.” \*

## Thwarting Melanoma

*JCC fellow Roberto Zoncu reveals intricacies of melanoma cells’ dependence on leucine.*

Malignant melanoma cells depend on the amino acid leucine, according to recent research by JCC fellow Roberto Zoncu published in the May 2011 issue of *Cancer Cell*. Zoncu and his coauthors found that melanoma cells with a mutation in the RAS/MEK signaling pathway—the most common mutation found in the deadliest form of skin cancer—died when leucine was withheld.

When cells are starving, they repurpose some of their own old and damaged parts, breaking them down into their components and reusing the raw materials. This process, known as autophagy, is particularly relevant to cancer cells, which

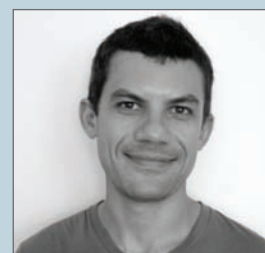
often grow in nutrient-limited conditions.

Autophagy is triggered when a specific pathway, the mTOR pathway, senses that nutrients are scarce. Zoncu and his coauthors discovered that leucine is a critical component. Without leucine, the melanoma cells did not recognize the nutrient shortage. But leucine is required for basic functions, so the cancer cells eventually died when they reached a metabolic crisis.

“My specific contribution to this work was to identify a cellular mechanism that prevents melanoma cells from triggering autophagy in response to leucine deprivation,” says Zoncu, who is a postdoc in David Sabatini’s lab at the Whitehead Institute.

Normally, leucine activates mTORC1 by recruiting it to lysosomes and suppressing autophagy. When leucine is removed, mTORC1 detaches from lysosomes and allows autophagy to proceed. Using confocal microscopy, Zoncu found that, in the melanoma cells studied, the mTORC1 remained attached to lysosomes even without leucine, preventing autophagy and leading to cell death.

The hope is that, eventually, the research could be useful in developing future melanoma treatments. “These findings are exciting because they reveal a novel mechanism through which the RAS oncogene may drive tumor growth, namely, by forcing



*Roberto Zoncu*

the localization and activation of mTORC1 to the lysosome,” says Zoncu. “While forced localization of mTORC1 likely contributes to driving tumor growth, it also introduces a selective liability of melanoma cells that occurs upon deprivation of leucine, and which may be exploited for therapeutic purposes.” \*

# Fellows Awarded Spring 2011

- **Lacramiora Bintu**  
**Frederic M. Richards Fellow**  
 Gene regulatory dynamics of cell differentiation, with Dr. Michael Elowitz, Department of Applied Physics California Institute of Technology, Pasadena, CA
- **Matthew T. Cabeen**  
**Merck Fellow**  
 Bacterial biofilm regulation by D-amino acids, with Dr. Richard M. Losick, Department of Molecular and Cellular Biology, Harvard University, Cambridge, MA
- **Pamela V. Chang**  
 Sensing gut microbiota through G-protein coupled receptors, with Ruslan Medzhitov, Department of Immunobiology, Yale University New Haven, CT
- **William S. Childers**  
 Exploring the role of kinase sub-cellular localization in bacterial cell cycle regulation, with Lucy Shapiro, Department of Developmental Biology, Stanford University, Stanford, CA
- **Bryan Dickinson**  
 Continuous directed evolution of proteases for cancer therapy, with David Liu, Department of Chemistry and Chemical Biology, Harvard University, Cambridge, MA
- **Megha Ghildiyal**  
 Repair and regeneration of *Drosophila* musculature: A potential role for muscle stem cells, with Allan Spradling, Department of Embryology, Carnegie Institution of Washington, Baltimore, MD
- **Kristin R. Gleitsman**  
 A comparative approach to uncover mechanisms of RNA molecular recognition, with Daniel Herschlag, Department of Biochemistry, Stanford University, Stanford, CA
- **Martin Haesemeyer**  
 Circuits mediating learning and sensory processing in the context of memory in zebrafish, with Florian Engert, Department of Molecular and Cellular Biology, Harvard University, Cambridge, MA
- **Chanhee Kang**  
**HHMI Fellow**  
 Elucidation of senescence regulatory networks using unbiased genetic screens, with Stephen Elledge, Department of Genetics, Harvard Medical School, Brigham & Women's Hospital, Cambridge, MA
- **Peri Kurshan**  
 A search for determinants of synaptic size and function, with Kang Shen, Department of Biology, Stanford University, Stanford, CA
- **Minh TN Le**  
 The role of secreted microRNA's in breast cancer metastasis, with Judy Lieberman, Immune Disease Institute, Children's Hospital, Boston, MA
- **Bo Li**  
**HHMI Fellow**  
 Unusual redox chemistry in the biosynthesis and action of the dithiopyrrolone natural product holomycin, with Christopher T. Walsh, Department of Biological Chemistry and Molecular Pharmacology, Harvard Medical School, Boston, MA
- **Xin Li**  
**HHMI Fellow**  
 Understanding the function and regulation of piRNAs in mammals, with Phillip Zamore, Department of Biochemistry and Molecular Pharmacology, University of Massachusetts Medical School, Worcester, MA
- **Jesse Lipp**  
 A chemical genetics approach on how signaling controls aberrant mRNA splicing in cancer, with Kevan Shokat, Department of Cellular and Molecular Pharmacology, University of California, San Francisco, CA
- **Xin Lu**  
**HHMI Fellow**  
 Genetic and genomic analysis of prostate cancer progression, with Ronald DePinho, Department of Medical Oncology, Dana-Farber Cancer Institute, Boston, MA
- **Benjamin J. Matthews**  
**HHMI Fellow**  
 Molecular genetics of water sensation and oviposition site preference in the yellow fever mosquito, with Leslie Vosshall, Laboratory of Neurogenetics and Behavior, The Rockefeller University, New York, NY
- **Christina K. McPhee**  
**HHMI Fellow**  
 The role of PHA-4 binding in cell fate specification during organ formation, with Susan Mango, Department of Molecular and Cellular Biology, Harvard University, Cambridge, MA
- **Michael J. Moore**  
 Defining layers of post-transcriptional control in chronic inflammatory disease, with Robert Darnell, Laboratory of Neuro-Oncology, The Rockefeller University, New York, NY
- **Kassandra M. Ori-McKenney**  
 Modes of microtubule nucleation in *Drosophila* neurons, with Yuh Nung Jan, Department of Physiology, University of California, San Francisco, CA
- **Rachel H. Roberts-Galbraith**  
 Investigating mechanisms underlying nervous system regeneration in the planarian, *Schmidtea mediterranea*, with Phillip Newmark, Department of Cell and Developmental Biology, University of Illinois Urbana-Champaign, Urbana, IL
- **Thomas E. Schaus**  
 Intelligent drug delivery by dynamic nucleic acid nano-devices, with Peng Yin, Wyss Institute for Biologically Inspired Engineering and Department of Systems Biology, Harvard University, Boston, MA
- **David Shechner**  
**HHMI Fellow**  
 Structure and function of large non-coding RNAs regulated by p53, with John Rinn, Department of Stem Cell and Regenerative Biology, Harvard University, Broad Institute, Cambridge, MA
- **Kristan K. Steffen**  
 Cell non-autonomous modulation of electron transport, with Andrew Dillin, Molecular and Cell Biology Laboratory, The Salk Institute for Biological Studies, La Jolla, CA
- **Margaret M. Stratton**  
**HHMI Fellow**  
 The molecular mechanism of CaMKII activation by specific calcium-spike frequencies, with John Kuriyan, Department of Molecular and Cell Biology, University of California, Berkeley, CA
- **Hao Wu**  
**Merck Fellow**  
 Dissecting functions of long noncoding RNAs in cardiac progenitors and heart development, with Kenneth Chein, Department of Stem Cell and Regenerative Biology, Massachusetts General Hospital, Harvard Stem Cell Institute, Boston, MA
- **Jai Y. Yu**  
 Investigating the mechanism of memory recall, with Loren Frank, Department of Physiology, University of California, San Francisco, CA

## Fellowship Application Information

The Fund awards fellowships to qualified individuals for full time postdoctoral research on cancer and related subject areas. Applicants should not have more than one year of postdoctoral experience and should hold either an M.D. or a Ph.D. in the field in which they propose to study. In some cases, evidence of equivalent training and experience will be accepted. The appointment normally lasts three years. The basic stipend for the 2012 recipients will be \$45,000 the first year, \$46,000 the second, and \$48,000 the third. Applications for 2012 must be submitted electronically and received by Wednesday, February 1, 2012. **For details, please visit the Fund's website at [www.jccfund.org](http://www.jccfund.org)**

## DIRECTOR'S CORNER

## Tough Times for Federal Funding



*From the National Library of Medicine and the Department of Special Collections and Archives of Stanford University:*

. . . We have completed our selection of graduate students. It was unusually difficult this year, I thought, because of the very large number of first-rate candidates . . . I think they are a very bright group; many of them have had extensive research experience and all of them are eager to conquer the world! The only concern I have is what will happen to these people four to five years hence. If there is in fact a constriction in the opportunities for biochemically trained people at that time, we shall have on our hands the responsibility of trying to figure out what to do with them and how to place them. This doesn't even take into account the problem of what will happen if the Training Grant funds are eliminated (more about that later).

The grant situation seems to be deteriorating rapidly. There are all kinds of rumors many of which are hard to believe but the gist of the story is that both the level of support and the type of work that will be supported by, for example NIH is undergoing serious review.

— from a letter written to Arthur Kornberg by Paul Berg, dated April 3, 1970

Yes, the current federal funding situation is bleak, but as the note above documents, times have been tough before. The group of students to whom Berg refers, including yours truly, survived and flourished through generations of up and down funding at the NIH. I have every confidence that we will weather this funding contraction and emerge with an essentially intact biomedical enterprise. But how to deal with the current crisis in which NIH pay lines are likely to hover around the 10% level of success? Clearly, the situation calls for changes in funding mechanisms to ensure that beginning and worthy established research groups have an opportunity to continue at some level of support. Simple policy changes could help during this difficult period. For example, the Stanford Biochemistry Department, of which Berg and Kornberg were charter members, had a policy of sharing all reagents and making joint decisions on major equipment purchases. The resulting efficiencies provided a buffer to keep their enterprise afloat. Current accounting procedures for federal grants greatly discourage such consolidation, but is this really necessary if the result is to foreclose opportunities for group collaboration? The R01 mechanism remains the preferred path for most investigators, but without greater flexibility, we will lose many established and early career scholars who fail to meet the impossible standards that the budget situation dictates. One might even consider giving special consideration to joint R01 proposals from groups of investigators who wish to consolidate their programs into a single collaborative effort.

Fortunately, the situation in funding of Jane Coffin Childs fellows is not so bleak. We enjoy continued support from the Childs' endowment and crucial additional support from the Howard Hughes Medical Institute, the Genentech Foundation, The Anna Fuller Fund and Merck Corporation. This year we were able to increase the number of new fellows from 23 to 26. The need is great and we continue to look for new funding opportunities.

As always, I am grateful to my colleagues on the Board of Scientific Advisors for their efforts on behalf of our fellows. One of our enduring strengths is the scientific judgment and collegiality of this group. Of course, as we renew the team, we lose the services of veteran members. This year, we say goodbye to Elaine Fuchs (Rockefeller University) but welcome Carol Prives (Columbia University) and Haifan Lin (Yale Medical School). I am so pleased that such notable scholars are willing to devote precious time to this effort. \*

Randy Schekman, Director of the Board of Scientific Advisors

## A Conversation with Jeremy Berg

*Former JCC fellow Jeremy Berg concludes his service as director of the basic sciences institute at the National Institutes of Health this year.*

When Jeremy Berg became director of the basic sciences institute at the National Institutes of Health (NIH) in 2003, a period of growth was ending. The Institute's budget had increased substantially over the previous five years, and Berg stepped in during what some called the "post-doubling hangover." Instead of tackling fun challenges like how to spend money and start new initiatives, Berg faced questions of how to stretch money, identify priorities, and decide which opportunities to forgo.

As director of the National Institute of General Medical Sciences (NIGMS), he oversaw a \$2 billion budget funding more than 4,500 research grants—about 10 percent of the grants funded by NIH as a whole—in cell biology, biophysics, genetics, developmental biology, pharmacology, physiology, biological chemistry, bioinformatics and computational biology. This year, he steps down from the position.

Tough economic times didn't stop Berg from leaving his mark at NIGMS during his eight years there. In one move toward greater transparency, he publicized information about how peer-reviewed grant proposals are chosen for funding. Both scientific quality and alignment with NIGMS programmatic goals matter, a fact that emerges from the funding curve he published yearly on the blog he created, called the Feedback Loop.

Berg aimed to make the most of NIH dollars. "The fear is that when funding is tight, research becomes more conservative, and that's terrible for science," Berg says, explaining that money could end up supporting

sure-fire, but less interesting, projects. To counter that possibility, he championed special programs targeting cutting-edge projects and investigators by leading the NIH Director's Pioneer Award and New Innovator Award programs. Plus, he changed the nomination process to ensure that the awardees represent a better cross-section of age groups and gender. He also discovered that lab productivity peaks at a funding level of about \$700,000 per year, a finding that supports the NIH policy of financing more scientists instead of concentrating dollars in the labs of a few high-profile researchers.

"Under Jeremy's leadership, NIGMS continued its impressive record of supporting outstanding research and training programs," said NIH Director Francis Collins. "He has been a leader who is always willing to roll up his sleeves and pitch in."

Becoming director of NIGMS wasn't part of some grand career plan, according to Berg. He described himself as a "hopeless nerd" in school. As an undergraduate, he worked in Keith Hodgson's lab at Stanford University, and he followed his passion for chemistry through the end of his PhD at Harvard.

Then Berg realized he needed to shift directions. "I was totally in love with chemistry and wanted to understand biology," he said. He won a fellowship from the Jane Coffin Childs Fund and joined Carl Pabo's lab at Johns Hopkins.

"JCC funding turned out to be so valuable because it allowed me to change directions quite dramatically," Berg said. With his own funding, he had the freedom to move away from his

current strengths and try something new.

When deciding how to spend his postdoctoral years during the 1980s, Berg read a "relatively obscure paper on an obscure topic" that led him to study zinc fingers, structural components of proteins that bind other molecules. The field was on the rise and it combined inorganic chemistry with his new interest in biology. In part, Berg credits his early successes to his strong foundation in chemistry, which he could then apply to biological questions.

Berg went on to spend 17 years as a professor at Johns Hopkins University, first in the chemistry department and then as director of the biophysics and biophysical chemistry department in the School of Medicine, where he also became director of the Institute for Basic Biomedical Sciences.

Then, in 2003, Berg received a phone call from a search committee at NIH. "I had no idea what the [NIGMS] job entailed," he said. "But it seemed like an intriguing opportunity." He wanted the chance to make substantial decisions that really mattered. Plus, he jokes that if he had turned down the offer, he would have forever relinquished the right to complain about the state of affairs in science.

Now, he's transitioning again, moving on to become associate senior vice chancellor for science strategy and planning in



*Jeremy Berg*

the health sciences at the University of Pittsburgh and a faculty member in the department of computational and systems biology.

"I had no intention of leaving NIGMS at this point, but am doing so in support of the career of my wife, a leading breast imaging clinical researcher," Berg said. "After a change in her situation earlier this year, she was recruited by many institutions around the country, and the University of Pittsburgh offered tremendous opportunities for each of us." Dr. Wendie Berg joined the University of Pittsburgh School of Medicine as a professor earlier this year.

As Berg shifts from the government world to the university setting, he emphasizes that there are many career paths in scientific research. And young people, like JCC fellows, have a particular advantage to achieve bold things while they have fire in their bellies. \*

*The Feedback Loop blog alerts researchers to NIGMS funding opportunities, trends, and plans. Anyone can offer input and feedback on Institute activities by commenting on posts.*

*Read the blog at <https://loop.nigms.nih.gov/>*

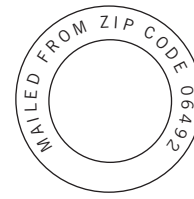


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## Thirty-fifth Symposia

### Challenges in Biomedical Sciences: *Research from the Fellows' Perspective*

October 21-23, 2011 • Interlaken Inn, Lakeville, Connecticut

HOSTED BY: Dr. Bonnie Bassler and Dr. Richard M. Losick



*Jack Dixon, Vice President and Chief Scientific Officer of the Howard Hughes Medical Institute, spoke at the 2010 Symposium.*



*JCC Fellow Rachel Mitton-Fry speaks with Board of Managers member William G. Gridley, Jr and his wife Barbara Gridley.*



*Board of Managers members at the 2010 Symposium*

*Standing: John W. Childs, William G. Gridley, Jr., James E. Childs, Elisabeth Childs Gill, Gardner M. Mundy, Bronwen A. Childs  
Seated: John D. Childs, Richard S. Childs, Elizabeth Borden, Alice Childs Anderson, Joan A. Steitz*

#### The 2011 Symposium will consist of presentations by third year fellows:

- **Eftychia (Effie) Apostolou**  
Konrad Hochedlinger, Massachusetts General Hospital
- **Joshua Black**  
Johnathan Whetstone, Massachusetts General Hospital
- **Bryan W. Davies**  
John H. Mekalanos, Harvard Medical School
- **Robert Driscoll**  
Karlene Cimprich, Stanford University
- **Andrew E. H. Elia**  
Stephen J. Elledge, Harvard Medical School
- **Liangcai Gu**  
George M. Church, Harvard Medical School
- **Suzanne K.L. Komili**  
Hiten Madhani, University of California, San Francisco
- **Claus Dieter-Kuhn**  
Leemor Joshua-Tor, Cold Spring Harbor Laboratories
- **Prabhat Kunwar**  
David Anderson, California Institute of Technology
- **Vicki P. Losick**  
Allan C. Spradling, Carnegie Institute, Baltimore Maryland
- **David G. Mets**  
Michael Brainard, University of California San Francisco
- **Brant Peterson**  
Hopi Hoekstra, Harvard University, Cambridge
- **Elizabeth Read**  
Arup Chakraborty, Massachusetts Institute of Technology
- **Dragana Rogulja**  
Michael Young, The Rockefeller University
- **June Round**  
Sarkis Mazmanian, California Institute of Technology
- **Edmund Schwartz**  
Richard Axel, Columbia University, New York
- **Alina Vrabioiu**  
Gary Struhl, Columbia University New York
- **Zhiping Wang**  
Yishi Jin, University of California, San Diego
- **Zeba Wunderlich**  
Angela DePace, Harvard Medical School, Boston
- **Oh-Kyu Yoon**  
Rachel B. Brem, University of California, Berkeley
- **Roberto Zoncu**  
David Sabatini, Whitehead Institute of Biomedical Research, Cambridge

#### Saturday evening October 22nd Speaker

- **Dr. Peter S. Kim**  
President, Merck Research Laboratories  
Merck & Co., Inc.  
North Wales, PA