MCB Transcript

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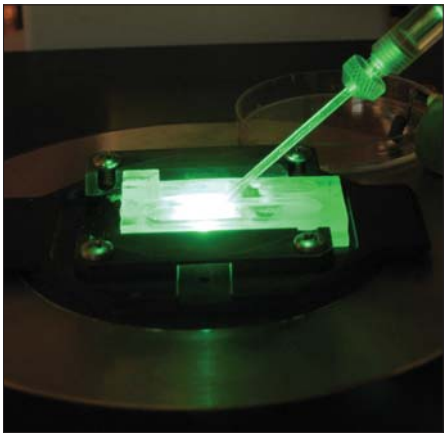
Newsletter for Members and Alumni of the Department of Molecular & Cell Biology at the University of California, Berkeley

NANOMEDICINE CENTER VIEWS PROTEINS IN A DIFFERENT LIGHT

Remote-controlled enzymes and artificial photoreceptors are among the innovations likely to emerge from a new collaborative effort that draws upon the research of three MCB faculty members. Neurobiologists Ehud Isacoff, Richard Kramer and John Flannery, together with chemistry professor Dirk Trauner, are the founding investigators of the Nanomedicine Development Center for the Optical Control of Biological Function, set up in April with a $6-million, five-year grant from the National Institutes of Health.

The aim of the research center is to modify ordinary proteins, such as ion channels and enzymes, so that they can be flipped on and off instantaneously by a flash of light. Such photo-switchable proteins have tremendous potential for both basic research and medicine, says Isacoff. "With this technology we can do something that has really been impossible before," he says. "How far it can be taken remains to be seen."



Illuminating work: A glass electrode is used to measure the conductance of a membrane bearing light-switchable ion channels.

Many functional proteins rely on some sort of effector molecule, known as a ligand, to regulate their activity. Isacoff and colleagues tether cellular ligands to their cognate proteins by a photosensitive linker that changes shape in response to light. In one state, the linker holds the ligand at arm's length from its binding pocket on the protein surface. When struck by just the right wavelength of light, however, the linker either shortens or bends in such a way as to allow the ligand to bind, thus either activating or shutting down the protein.

The team has used the approach to create remote control neurons that fire when light-activated. In one case, the light-responsive linker molecule azobenzene was used to tether a pore blocker, which acts like a drain plug, to a nerve cell potassium channel. With azobenzene in one configuration, the channel is plugged, ions do not flow, and the neuron will not fire. Short-wavelength light shifts the azobenzene conformation so the plug is literally pulled out of the channel and the nerve can conduct a signal (Banghart, M. et al. *Nat.*

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NEW PROFS STUDY CHANNELS, SUICIDE

BRYAN KRANTZ

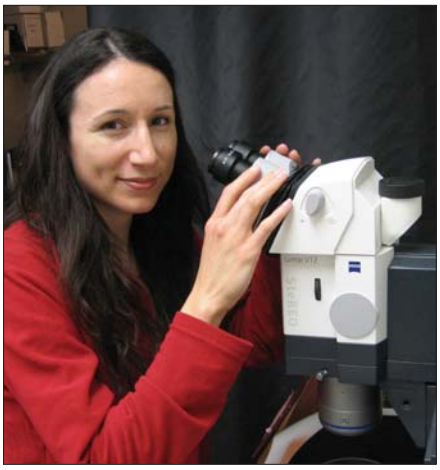
Membranes abound in the cytoplasm of eukaryotic cells. Specialized compartments like the endoplasmic reticulum, Golgi apparatus, lysosomes, endosomes, peroxisomes, vacuoles and vesicles of all sorts flourish between the double-walled nucleus and the outer membrane. Their greasy barriers do a good job of keeping things in place, but legitimate traffic needs to find a way through. The job of membrane channels is to provide regulated portals for everything from ions to macromolecules that need to move around in this wall-filled world.

Translocase channels (TCs), which provide passage to proteins, are among the most remarkable of these. An estimated half of all proteins in the cell make use of them, and in order to do so, each

CONTINUED ON PAGE 3. . .

Neurosci. **7**, 1381-1386; 2004). In a variation on this theme, light is used to reversibly gate a glutamate receptor channel. A tethered glutamate molecule evokes action potentials upon correct stimulation of the photoisomerizable linker (Volgraf. M. et al., *Nat. Chem. Bio*. **2**, 47-52; 2006; Gorostiza et al., *PNAS*).

The approach has several advantages for neurological research, Isacoff says. Nerves grouped in bundles tend to get activated en masse by traditional electrical or chemical stimulation, whereas light can pinpoint single neurons or specific groups of neurons. This can be done *in vitro* or *in vivo*, and has already been used to manipulate behavior. Zebrafish carrying remote control channels in specific subsets of sensory neurons can have their escape response to touch reversibly turned off with no effect on other swimming or chasing behaviors (Szobota et al., *Neuron* **54**, 535-545; 2007). Other methods, such as altering gene expression or releasing optically caged neurotransmitters, while effective, can be slow to reverse and hard to direct to specific cell types. In contrast, the conformation of azobenzene can shift instantaneously when hit with a different wavelength of light, and the protein to which it is attached can be expressed selectively in certain cell types.



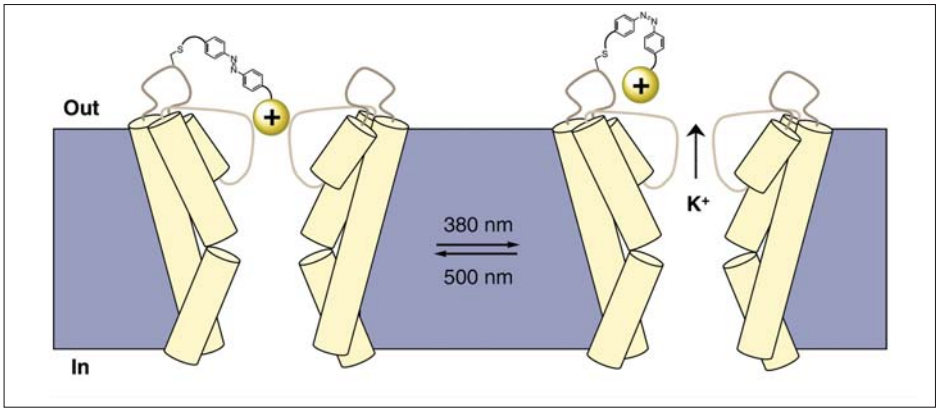
Stephanie Szobota, one of the graduate students on the project, at the dissecting scope used for neuron and zebrafish preparations.

Isacoff foresees a wide range applications in biology. Basic molecular research, he suggests, often involves altering protein function, either genetically or chemically. But even the most powerful genetic methods for controlling protein activity, such as RNA interference and gene manipulation, are not easily or quickly reversible. Furthermore, pharmacological approaches are hard to target to specific cells or tissues. In fact, drugs have an inherent tradeoff: the more specific they become for a molecular target, the more tightly they bind and the harder they are to wash away again. The light switch feature of photoactivation overcomes these limitations and allows reversible remote switching on a time scale of milliseconds or less. "There is a huge payoff here for basic research," Isacoff says.

Of course the potential for the treatment of blindness has not been overlooked. Many blinding diseases such as retinitis pigmentosa and macular degeneration are characterized by the loss of photoreceptor cells, at least initially leaving the rest of the retina intact. One approach to restoring the retina's sensitivity to light is to link photoswitchable pore blockers to ion channels in the retinal ganglion cells, the output neurons that transmit photoreceptor signals to the brain. Although this bypasses several layers of information processing that occur in the healthy retina, the team hopes that the brain will learn to interpret the new signals, allowing at least partial vision. After all, he says, the brain is extremely adaptable, as demonstrated by amputees who learn to control prosthetic limbs. Initial experiments applying this approach to rodents are just getting underway.

The center is one of eight Nanomedicine Development Centers established as part of the NIH Roadmap for Medical Research, a funding mechanism that emphasizes the translation of basic research into clinical developments. Isacoff took advantage of a Berkeley Research Futures Grant in 2005 to complete some of the groundwork needed to put together an competitive proposal for the center. The NIH says it intends to renew funding after five years to those centers that show progress towards clinical applications.

Bright future: The zebrafish (Danio rerio) *has been endowed with light-switchable sensory neurons that let certain behaviors be controlled by light.*



Unplugged: A pore blocker tethered by a light-sensitive linker gets pulled from a potassium channel when the wavelength of applied light is changed.

MCB Transcript

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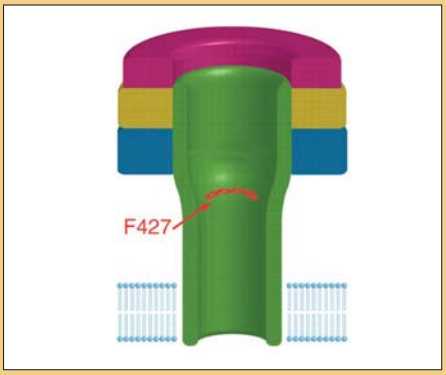
NEW FACULTY CONTINUED FROM PAGE 1. . .

protein must first unfold and snake through the channel's narrow opening. Since their discovery in the 1960's, TCs have been thought of as solvent-filled holes. But recent structural studies have shown that the passage is in fact so narrow, that it must make intimate contact with the polypeptide chains as they wriggle through, perhaps even helping to push them to the other side. Yet how they do this remains a mystery.

Bryan Krantz, who joined the department last summer as an associate professor of biochemistry and molecular biology with a joint appointment in chemistry, believes the questions surrounding TC function are exciting biophysical problems. "The prevailing model was that the channel didn't do much to unfold the protein. But I think as people get more structural detail, we will learn there are critical components of the channel that interact with the chain," he says.

The Krantz lab uses anthrax toxin as a model for protein translocation. Released by the bacterial pathogen *Bacillus anthracis,* anthrax toxin consists of three proteins that work together to invade cells. One of these, known as PA for protective antigen, forms a channel through the membrane of endosomes, the vesicle in which the toxin finds itself trapped after the host cell absorbs it. Then, through this channel and into the cytoplasm slither edema factor and lethal factor, PA's irritating and sometimes deadly partners in crime. The toxin makes a convenient system for studying protein channels, because PA is capable of self-insertion into any membrane you show it, making it much easier to work with than most eukaryotic TCs.

To create a model membrane for his experiments, Krantz spreads a thin film of dissolved lipid in organic solvent over a pinhole aperture with a paintbrush bristle. The result is a single lipid bilayer with the essential properties of a cell membrane. The toxin is then applied to the bilayer. With a small enough aperture, Krantz can even get a single PA channel to insert. He then measures the ionic current flowing through the channel under an applied membrane potential to determine whether or not a protein is in the channel.



Tight squeeze: schematic diagram of the anthrax toxin protective antigen forming a protein translocation channel in a lipid bilayer. The phenylalanine collar that regulates the passage of unfolded polypeptide chains is shown in red.

Krantz has described a seven-member phenylalanine ring that forms a tight collar part-way through the channel. This hydrophobic heptad, known as the ϕ clamp, is required for translocation of proteins. Its hydrophobic nature stands in contrast to the neighboring amino acid side-chains in the channel, which are hydrophilic. How the ϕ clamp works is a current focus of work in the Krantz lab.

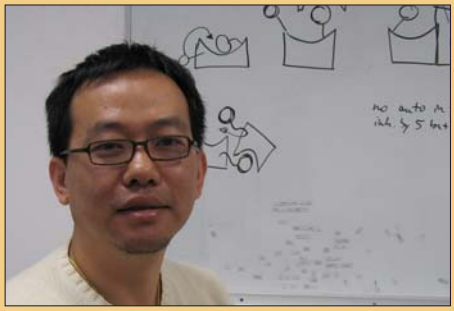
Krantz says he is looking forward to growing his lab slowly over time by taking on a combination of chemistry and MCB students. In fact, the graduate students were one of the main attractions of Berkeley, he says. Not only do they have a reputation for intelligence and creativity, but they are more willing than most to give new faculty a fair consideration when choosing a mentor. "It's a good place to become a junior professor," he says.

QING ZHONG

Death is part of life, particularly at the cellular level. Every cell in the body has the ability to commit suicide for the good of the whole, and ultimately quite a few do so, both as a part of normal growth and in response to damage or disease. Not to be undertaken lightly, programmed cell death is a carefully controlled process involving a number of cellular signaling pathways. Some of these are well understood, yet certain key features that tip the balance between life and death remain shrouded in darkness.

Qing Zhong, who joined MCB as an assistant professor of biochemistry and molecular biology last fall, is tackling the problem with a strictly biochemical approach. Using high resolution chromatography methods to fractionate cell extracts into discrete enzymatic activities, he is attempting to rebuild cell death signaling in a test tube. Ultimately he hopes to learn how cells activate two key suicide pathways: apoptosis, and the recently implicated process of autophagy, or self-ingestion.

Zhong became enamored of these questions as a postdoc in Xiaodong Wang's lab at the University of Texas Southwestern Medical Center in Dallas. Wang is the scientist who first established the critical role of mitochondria in apoptosis. He showed that a DNA damage signal from the nucleus causes the



outer mitochondrial membrane to open and release the cell death initiator cytochrome *c*. Yet the details of the signal itself remain unclear. "The key question we don't understand is how the DNA damage signal is transmitted all the way from the nucleus to the mitochondria," Zhong says.

To get at the answer, Zhong set up an *in vitro* assay for cytochrome *c* release while in the Wang lab. This allowed him to reconstitute the pathway in a tube and see when apoptosis was successfully initiated. The approach paid off when Zhong identified a key protein in the pathway he dubbed Mule. Also known as ARF-BP1, this protein tags the anti-apoptotic protein Mcl-1 with ubiquitin, marking it for destruction by the proteosome (*Cell* **121**, 1085-1095; 2005). It was both an important step in unravelling the signal cascade and a vindication of the biochemical approach. "No one had ever pulled out an enzyme like this through biochemical fractionation before," Zhong says.

Work on Mule continues in Zhong's new lab in Barker Hall. He wants to understand what upstream factors stimulate Mule upon DNA damage. He is also curious about Mule's other putative roles in protein regulation. It is thought to ubiquitinate the oncogene c-Myc, and there is weak evidence that it may even target histones. Perhaps more significantly, another substrate of Mule appears to be p53, a tumor suppressor gene that is mutated in most human cancers. Since tagging by Mule is a death sentence for a protein, it is interesting that Mule is overproduced in many human cancers as well. Whether its action on p53 will constitute an important biological mechanism of cancer progression remains to be seen, but Zhong is actively pursuing this possibility.

In another area of endeavor, Zhong is now turning the biochemical fractionation approach on autophagy. It has been known for half a century that cells can cannibalize parts of themselves, mostly for the purpose of self-renewal, but also as a defense. Some infectious bacteria, for instance, can only effectively be destroyed by incorporation into the autophagosome, a double-membrane vesicle that gobbles up junk inside the cell and exposes it to lytic enzymes. Now autophagy has also been implicated in programmed cell death.

CONTINUED ON PAGE 4. . .

MCB FACULTY LAUNCH PATHOGEN RESEARCH CENTER

A group of MCB immunologists has received a 5-year project grant from the National Institutes of Health to study the immune system's reaction to a variety of pathogens, including potential bio-terror agents and emerging infectious disease organisms. The Center for Host-Pathogen Studies capitalizes on existing expertise in mouse strain development and imaging to find out how specific molecules and cells react to infections. "The idea is to work collaboratively to gain a better understanding of host-pathogen interactions," says Ellen Robey, the program director on the grant.

The center, which was established last fall, includes three independent but overlapping projects. Robey and Nilabh Shastri are studying innate and adaptive immunity to *Toxoplasma gondii*, an intracellular parasite sometimes found in cat feces and uncooked meat. *Toxoplasma*, which can cause birth defects, makes an excellent model system for studying pathogens that evade the initial immune response to take up long term residence in the host. Not only can it be manipulated genetically, but it also infects mice much the same way as people. Thus the team can use the latest two-photon laser scanning microscopy to track the spread of the parasites in both normal mice and mice with mutations in components of their immune systems.

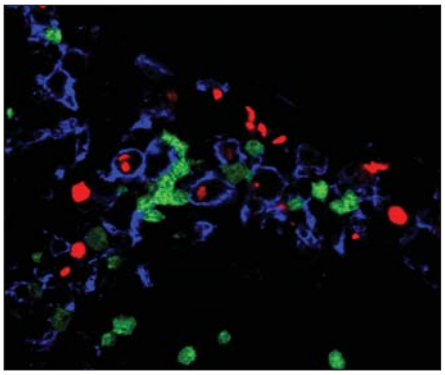
Astar Winoto, who heads the second project, is working on two major molecular pathways of innate immunity: the Toll-like receptors (TLRs), which identify pathogens by their molecular signatures, and the much less well understood RIP1/FADD pathway, which appears to be involved in responding to infection by double-stranded RNA viruses. In collaboration with Laurent Coscoy and Robey, Winoto hopes to elucidate the roles of FADD and an inhibitor of the TLR pathway known as TRAIL-R in regulating the host response to *Toxoplasma* and a number of virus classes, including several that are important to biodefense. Winoto hopes to identify novel FADD and TRAIL-R associated proteins that operate in these regulatory pathways, and then test their individual roles in innate immunity.

The third project team, headed by Coscoy and David Raulet, is interested in natural killer (NK) cells, components of the immune system that recognize and destroy cells in the body that have become infected with a pathogen. In particular, the NKG2D cell surface receptor helps NK cells spot viral infections by binding to specific ligands that are strongly up-regulated in virally infected cells. Exactly how cells know to boost production of these molecular markers of infection is still not well understood, but getting a handle on the mechanism could lead to ways of enhancing the NK response to viruses.

All three projects rely on several core facilities established by the $5 million grant. The Mouse and Virus Core, run by Winoto and core manager Namsil An, generates, maintains and distributes transgenic strains of mice as needed by center investigators. The core includes a biosafety level 2 facility for isolating pathogenic virus strains such as influenza and adenovirus. The Imaging Core, run by Robey and specialist Paul Herzmark, has state-of-the-art microscopes for imaging fluorescently tagged molecules in live preparations. The cores also form an integral part of the existing Cancer Research Laboratory transgenic and imaging facilities.

The combination of the cores is very powerful, says Robey. Pathogens bearing a fluorescent tag can be viewed during an active infection of live mouse tissue in which various cell types have been marked with distinct fluorescent proteins, providing an unparalleled view of the immune response whose various components have mainly been studied in isolation until now (see image). "We feel that the ability to visualize the pathogen and the host immune cells in real time during on-going immune responses will provide a lot of new insights," Robey says.

For more on the center, visit *http://mcb.berkeley.edu/chpr/*.



Rapid response: fluorescently labeled macrophages (blue) and memory T cells (green) respond to infection with Toxoplasma *(red) in a lymph node from an infected mouse.*

NEW PROFS CONTINUED FROM PAGE 3. . .

Hardcore biochemistry: The liquid chromatography apparatus that allows Zhong to fractionate cellular extracts into individual proteins.

Although little is known about the mechanism, it is clear that if apoptosis is blocked, autophagy can mediate death in damaged cells. Ironically, this appears to be important for longevity: stimulating autophagy extends the life-span of the roundworm *C. elegans*. In its classical conception, autophagy was not considered a highly regulated process, but now its dysregulation has been implicated in neurodegenerative disease and cancer. To find the signals involved, Zhong has an autophagy assay up and running.

Zhong says he was particularly attracted to Berkeley as one of the best places in the world to work on the biochemical analysis of signaling pathways. He notes the expertise of people like Robert Tjian, Michael Botchan and Randy Schekman who have so successfully applied the approach to problems such as transcription, replication and secretion. But perhaps just as important, Zhong says, is Berkeley's reputation for free and creative thought. "I want an environment that stimulates," he says. "I admire the free spirit of the place."

FACULTY NEWS

▼ **Steven Brenner** (G&D) will be a Miller Research Professor for the 2007-2008 academic year.

■ The second edition of the seminal reference "The Bacteriophages", edited by **Richard Calendar** (BMB), was published this spring by Oxford University Press.

▼ **Abby Dernburg** (CDB) was selected as the 2007 recipient of the American Society for Cell Biology Early Career Life Scientist Award, which will be presented in December at the annual ASCB meeting in Washington, DC. This will be the fourth time in the award's eight year history that an MCB faculty member has won. The previous MCB recipients were Karsten Weis (2006), Eva Nogales (2005), and Kathleen Collins (2002). Dernburg also received a Research Scholar Grant from the American Cancer Society and has been promoted from Assistant Professor in Residence to Associate Professor, effective July 1, 2007.

■ **Caroline Kane** received the Chancellor's Award for Advancing Institutional Excellence, which comes with $30K to be used for her diversity and education work. She was also honored with the California Alumni Association 2007 Excellence in Service Award.

■ **Gary Karpen** (CDB) has been awarded one of ten new grants under the modENCODE project, a 5-year, $57 million effort by the National Human Genome Research Institute to identify all functional elements in the genomes of the fruit fly and roundworm. Karpen's project is entitled "Genome-Wide Mapping of Chromosomal Proteins in *Drosophila*."

▲ **Michael Marletta** (BMB) was honored with three significant awards this spring. The American Chemical Society (ACS) selected him for the 2007 Gustavus John Esselen Award for Chemistry in the Public Interest in recognition of "his work in nitric oxide biology and malaria, and his communication of fundamental chemical research to non-science audiences." Marletta also received the 2007 Emil Thomas Kaiser Award given by the Protein Society for his application of chemistry to the study of proteins. Randy Schekman (BMB) won the Kaiser in 1999. And finally, Marletta won the Repligen Award in Biological Processes from the ACS's Division of Biological Chemistry.

■ **Michael Rape** (CDB) was honored with two junior researcher awards, a Kimmel Scholars Award from the Sydney Kimmel Foundation for Cancer Research (www.kimmel.org) and a Pew Scholars Award (www.futurehealth.ucsf.edu/pewscholar.html), funded by the Pew Charitable Trusts. In accordance with the rules of both programs, he was only permitted to accept one. He chose the Pew.

■ **Jeremy Thorner** (BMB) was elected to the American Academy of Arts and Sciences. Also inducted this year were former Vice President Albert Gore, cosmic dust astronomer Donald Brownlee and filmmaker Spike Lee.

■ **Matthew Welch** (CDB) received the R.R. Bensley Award in Cell Biology from the American Association of Anatomists in April. The award is in recognition of his contributions to the understanding of the function and regulation of the actin cytoskeleton.

▼ Scientific American published feature articles by two faculty members this spring. **Frank Werblin** (Neuro) wrote in the April issue about the intense visual processing of the retina under the title "The Movies in Our Eyes." **Peter Duesberg's** (BMB) piece in May entitled "Chromosomal Chaos and Cancer" explores the evidence that aneuploidy, rather than accumulated point mutations, causes cancer.

■ **Jeffery Winer** was recognized as an Outstanding Graduate Student Instructor Mentor by the Graduate Council. He also gave the keynote address at the International Conference on the Auditory Cortex in Birmingham, England, in September 2006. The meeting, sponsored by the Medical Research Council, was entitled "The Listening Brain."

2007 AWARDS

OUTSTANDING GRADUATE STUDENT INSTRUCTORS

The following GSIs for MCB courses were among the 268 honored by the Graduate Division in a May 7 event at the Alumni House for outstanding teaching.

Abigail Gerhold (Hariharan lab)

Ksenia Krasileva (Plant and Microbial Biology)

Melinda Modrell (Patel lab, IB)

Rebecca Pferdehirt (Meyer lab)

UC Berkeley NewsCenter

Sunita Puri (Joint Medical Program)

Stacia Rodenbusch (Dernburg lab)

Tami Rowen (Health & Medical Sciences Program)

Allyn Schoeffler (Berger lab)

Elizabeth Slawson (Rine lab)

Samuel Stephenson (Krantz lab)

Brian Swartz (Integrative Biology)

Jennifer Thompson (Winoto lab)

S. Derek Turner (Public Policy)

Andrea Wills (Harland lab)

Veronica Zepeda (Cate lab)

photos not available for

Rohini Dhand (Public Health)

Katherine McHenry (Plant and Microbial Biology)

UNDERGRADUATE AWARDS

University Medal Finalist

- **Amar Kishan** (Jeffery Winer lab)

DEPARTMENTAL AWARDS

Departmental Citation

- **Amar Kishan**

Outstanding Scholar

- **Scott Coyle** (Jennifer Doudna lab)

DIVISION OF BIOCHEMISTRY & MOLECULAR BIOLOGY

Grace Fimognari Memorial Prize

- **Leila Ross** (Sheng Luan lab, PMB)

Kazuo Gerald Yanaba & Ting Jung Memorial Prize

- **Carrie Shiau** (Carolyn Bertozzi lab)

Jesse Rabinowitz Memorial Prize (for outstanding junior in BMB)

- **Panid Sharifnia** (Laurence Tecott lab, UCSF)

DIVISION OF GENETICS & DEVELOPMENT

Spencer Brown Award

- **Elizabeth Lally** (Lewis Feldman lab, PMB) and
- **Darien Reed** (Michael Levine lab)

Edward Blount Award

- **Geoffrey Friedman** (Urnov lab at Sangamo BioSciences)

DIVISION OF IMMUNOLOGY

Outstanding Undergraduate Immunologist

- **Judy Chang** (Mark Schlissel lab)

DIVISIONS OF CELL & DEVELOPMENT BIOLOGY AND NEUROBIOLOGY

I.L. Chaikoff Memorial Awards

- **Arash Calafi** (David Schaffer lab, Chem. Engineering)
- **Ellen Cheang** (Rong Wang lab, UCSF)
- **Jennifer Chen** (Leonard Bjeldanes lab, Nutr. Sci. & Tox.)
- **Phillip Ge** (John Flannery lab)
- **Jessica Lin** (George Sensabaugh lab, Public Health)
- **Olga Kochan** (Robert Zucker lab)
- **David Noorvash** (Sharif Taha lab, UCSF)
- **Bora Shin** (Gail Martin lab, UCSF)
- **Halley Tsai** (Yang Dan lab)
- **Lisa Ai-Jin Tseng** (Paola Timiras lab)
- **Carter Wystrach** (Cindy Chang lab, University Health Services)
- **Kathy Zhang** (Steven Yannone lab, LBNL)

CLASS NOTES

- **Pei Chen** (BA 2001) earned her MD from the University of Southern California and is now a resident in family medicine at Kaiser Permanente in Woodland Hills. (verysunny@hotmail.com)

- **Matthew Levine** (BA 1994) earned his MD from the University of Southern California in 2000. He completed a residency in internal medicine at Santa Barbara Cottage Hospital in 2003 and fellowship in endocrinology and metabolism at Scripps Green in La Jolla in 2005. He is currently in clinical practice in endocrinology in Laguna Hills, California. (clonedoc@earthlink.net)

- **Soheil Najibi** (BA 1990) completed the MD/PhD program at Boston University School of Medicine in 1997. Soheil went on to an orthopedic surgery residency at the BU Medical Center followed by a sports medicine fellowship at the University of Iowa in 2002. This culminated in a senior staff position as an orthopedic surgeon with the Henry Ford Health System in Detroit. (snajibi@pol.net)

- **Melissa Rusli** (BA 2003) after graduation moved back home to New Jersey where she worked at a company involved in radiology and clinical trials. She later decided to go back to school and is now in her third year at the University of Medicine and Dentistry of New Jersey working toward a Doctor of Osteopathy degree. She wishes all her MCB classmates much luck and success. (ruslime@umdnj.edu)

- **Jay Tung** (BA 1984) was appointed Vice President of Drug Discovery for the Myelin Repair Foundation, a non-profit medical research organization which seeks the rapid development of treatments for multiple sclerosis.

HAMMER WINS WEINTRAUB

Gianna Hammer, who completed her Ph.D. last year in Nilabh Shastri's lab, is one of twelve nationwide recipients of the 2007 Harold M. Weintraub Graduate Student Award, given by the Fred Hutchinson Cancer Research Center in Seattle to recognize outstanding achievement during graduate studies in the biological sciences.

CLASS NOTES WANTS TO HEAR FROM YOU

Do you have a bachelor's, master's or Ph.D. in Molecular and Cell Biology from Berkeley? Let your classmates know what you are up to by sending in a Class Note for publication in the next issue.

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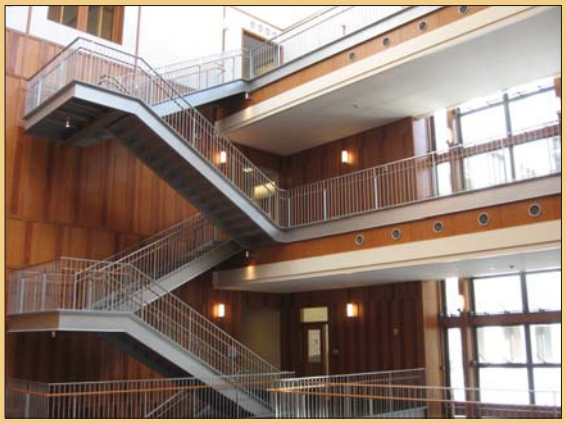
STANLEY HALL OPEN FOR SCIENCE

The new Stanley Hall is finished and labs have begun to move in. The 285,000 square-foot building houses the Berkeley arm of the California Institute for Quantitative Biomedical Research (QB3), including all QB3 administrative staff, some 40 affiliated research labs (many of them in MCB), and the Department of Bioengineering. Teaching and meeting facilities include three auditoria of 300, 120 and 45 seats and a multi-media flexible-space classroom for 45. The $162 million building replaces the original Stanley Hall, completed in 1952 (see below), which was around a fourth the size. The building's namesake, Nobel laureate Wendell M. Stanley, founded the Berkeley Virus Laboratory in 1948 and ultimately helped establish the Department of Molecular Biology, a predecessor of MCB.



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