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MISSION STATEMENT

The mission of the Neuroscience Research Institute is to foster knowledge and understanding of the nervous system by serving as a center for scientific research breakthroughs. The NRI is a group of investigators whose collective goal is to create an intellectual atmosphere conducive to exploration at the frontiers of human knowledge where disciplinary boundaries disappear. Investigators in the NRI recognize that the interests of neuroscience extend broadly from repair and prevention of human disease to the principles that underlie the earliest nervous systems, from the human mind to the single molecular building blocks of the brain.

Surface of a rabbit retina that has changed its shape due to the reactivity of the glial cells. This type of reactivity can occur following disease or injury to the retina. It was immunolabeled with antibodies to GFAP in green, and vimentin in red, and imaged using our Olympus FV1000 confocal microscope. Credit: Geoff Lewis
DIRECTORS’ STATEMENT

The mission of the Neuroscience Research Institute is to serve the research interests of the campus in all facets of neuroscience and to foster growth of strong neuroscience research at UCSB. As a central site for campus neuroscience research we particularly emphasize multi- and interdisciplinary research. We serve faculty from seven different academic departments and maintain a list of thirty additional UCSB NRI Affiliates. We provide our core members and affiliates the opportunity to highlight their work in the press and on social media. We provide an intellectual environment through our seminars, Gurley lectures and specialized centers. Our fourth Gurley lecture of the year featured Ralph Greenspan of UCSD and kicked off UCSB’s involvement in President Obama’s BRAIN research initiative.

Importantly, we provide knowledgeable staff support for grant submissions, maintaining accurate budget numbers, assisting with personnel matters in member labs, obtaining visas for foreign students, post-docs and visitors, and ordering supplies. This infrastructure has continued to be essential to the success of the unit during the 12–13 fiscal year. We welcomed Theresa Peña as Business Officer following the retirement of Jeanie Cornet and the transition was seamless thanks to the dedication of NRI personnel.

The NRI contributes to the academic needs of the University and enhances the instructional and research mission of the campus by operating the following cores: light microscopy, electron microscopy, deep sequencing, and stem cells. The purchase of an Ion Torrent DNA sequencer established our campus the first in the UC system with the technology. The NRI also houses three active centers: Alzheimer’s Disease Research Center, Center for Stem Cell Biology and Engineering and Center for the Study of Macular Degeneration. A 10,000 square-foot, $6.4 million renovation of dedicated stem cell research space was completed in October 2012.

The NRI has been instrumental in recruiting neuroscience faculty. This year we provided space for Craig and Denise Montell to establish a Drosophila behavior and neurophysiology unit and provided an FTE for a junior engineering faculty, Adele Doyle, to establish a stem cell lab that will interface with her interest in materials. Adele was also responsible, along with NRI affiliate Samir Mitragotri, for establishing the interdisciplinary Ph.D. emphasis in bioengineering that launched this year.

With the effects of the fiscal crisis largely behind us, NRI personnel are looking forward to exploring new technologies and collaborations in pursuit of better understanding the nervous system at all levels.

Stuart C. Feinstein
Co-Director

Kenneth S. Kosik
Co-Director
### OTHER PROJECTS + ACTIVITIES

#### ACADEMIC PROJECTS

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<td>Mark Humayun Stem Cell Center Keynote</td>
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<td>Gurley Lecture - Nancy Bonini, UoP</td>
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#### RESEARCH EXPERIENCES FOR GRADUATE STUDENTS

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#### RESEARCH EXPERIENCES FOR UNDERGRADUATES

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ALZHEIMER’S DISEASE RESEARCH CENTER

Investigations in the ADRC focus upon the normal and pathological action of the microtubule associated protein, tau, as well as mechanisms of neuronal plasticity and its impairment in neurodegeneration. A June 2013 paper from the Kosik Lab showed that dual kinase inhibitors reduced the phosphorylation of tau in mice, which may lead to an effective Alzheimer’s treatment for humans.

CENTER FOR STEM CELL BIOLOGY & ENGINEERING

The mission of the UCSB Center in Stem Cell Biology and Engineering is to foster an interdisciplinary program of stem cell research and teaching to develop new technologies in the emerging field of regenerative medicine. To accomplish this, the center supports collaboration and exchange of ideas among a wide range of disciplines, with research divided into three general areas: Molecular mechanisms of stem cell pluripotency, proliferation and differentiation; Biotechnology and Bioengineering of stem cell growth, differentiation, sorting and delivery; and Regenerative Medicine to translate discoveries to the clinic. Funded by a grant of $1.3 million in 2005 from the California Institute for Regenerative Medicine (CIRM), a stem cell training program has been developed to support graduate and postdoctoral fellows engaged in a variety of stem cell projects. The UCSB Laboratory for Stem Cell Biology and Engineering was established free of federal funding to allow research on all stem cell lines. Renovation of this facility, funded by a $2.2 million grant from CIRM, was completed in October 2012.

CENTER FOR THE STUDY OF MACULAR DEGENERATION

The Center for the Study of Macular Degeneration (CSMD) is a dedicated biomedical research unit and part of the Neuroscience Research Institute (NRI) at the University of California, Santa Barbara (UCSB). The mission of the CSMD is to advance basic biomedical research into the cellular, molecular, and genetic factors that contribute to the human ocular diseases that are known as macular degeneration. In pursuing its mission, the CSMD seeks to stimulate interactions between basic and clinical scientists, contribute to the training of students and postdoctoral fellows, and lay the groundwork for the development of new diagnostic and therapeutic approaches to treat macular degeneration.
DNA SEQUENCING FACILITY

In January 2013 the Facility acquired a Life Technologies Ion Torrent gene sequencer, the first of its kind for a UC campus. Besides core instrumentation, the genomics center includes UCSB faculty members, postdoctoral fellows, and graduate students -- as well as broader collaborations with research groups at UCLA and UC San Francisco. Mary L. Arcila, a doctoral student with Kosik, is assistant director of the sequencing facility. The purchase of the sequencer was preceded by a visit from the Ion Bus in October 2012. Campus and community members were able to see the Ion Torrent running samples and generating data in real time.

Mary Arcila and Kenneth Kosik Credit: George Foulsham

MICROSCOPY FACILITY

The NRI–MCDB Microscopy Facility, founded in 1990, is jointly maintained by the Neuroscience Research Institute and the Department of Molecular, Cellular and Developmental Biology at the University of California, Santa Barbara. The Facility’s mission is to promote and facilitate research necessitating microscopy. To achieve this mission the Facility houses state–of–the–art instruments, supports expert full–time support staff, hosts outreach events and provides individual and workshop based training in microscopy.

This centrally located Facility is based within the Neuroscience Research Institute, in the Biological Sciences II Building. Presently, the Facility maintains multiple sophisticated instruments including a JEOL JEM-1230 transmission electron microscope, a new Olympus Fluoview 1000 Spectral Confocal Laser Scanning Microscope, an Olympus Fluoview 1000 Multiphoton Laser Scanning Microscope and an Olympus Spinning Disk Confocal. The Facility also hosts five Olympus compound microscopes configured
with transmitted and fluorescent light-paths as well as a stereomicroscope configured with transmitted and reflected light. These microscopes are further equipped with research grade digital cameras and a high-end computer workstations for image acquisition, processing and analyses. The confocal and multiphoton microscopes are equipped with time-lapse software controls for automated long-term imaging and the Fluoview 1000 systems are equipped with a motorized X, Y stages for automated sampling of multiple locations. It also provides an Imaris 3D image processing and analysis workstation for the Facility users.

The Facility Director, Dr. Mary Raven is an experienced microscopist, scientist and manager. Dr. Raven is assisted by Dr. Geoff Lewis who oversees transmission electron microscopy and assists with confocal training as needed. Both Drs. Raven and Lewis have published numerous papers employing conventional, transmission electron microscopy and confocal microscopy. Drs. Raven and Lewis provide training on a daily bases and regularly meet with individuals to provide advice and to address additional microscopy needs.

The Facility offers an advanced microscopy workshop each year in which students are trained in a variety of modern light microscopy principles and techniques. The most recent workshop was offered in Jan 2013 and focused on advanced microscopy and live imaging. The workshops are designed to advance graduate and post-doctoral development and generally more than half the participants are graduate or post-doctoral researchers. The Facility also offers mini-workshops throughout the year on select topics and the Facility Director will be teaching a graduate and an undergraduate seminar on microscopy in the 2013–2014 academic year.

In the past year, the Facility supported 122 users, 60 Principal Investigators and more than 1800 reservations. The Facility users are requested acknowledge the Facility in their publications and report new publications supported by the Facility. 22 publications were reported to the Facility in 2012–2013.
We established The California Project to Cure Blindness, a “Disease Team” of investigators from USC, Caltech, City of Hope, University College London, and Regenerative Patch Technologies, LLC, to develop a stem cell therapy for Age-related Macular Degeneration. This work is funded by the California Institute for Regenerative Medicine. The California Project to Cure Blindness is an interdisciplinary research effort involving researchers at UCSB in the Center for the Study of Macular Degeneration and the Center for Stem Cell Biology and Engineering, along with retinal surgeons in the Keck School of Medicine at the University of Southern California, materials chemists at Caltech, and experts in cellular therapy from the City of Hope and University College London. The goal is to differentiate a monolayer of RPE on a synthetic substrate that can be implanted in the macula to replace damaged RPE and therefore preserve photoreceptors.

DOHENY EYE INSTITUTE

Peter Coffey 7/1/13-6/30/14 $1,00,000
Modeling AMD in a dish - a bioassay of disease pathogenesis 1312

It is likely that in degenerative diseases such as AMD that “wound responses” come into play. Epithelial cells have a potent capacity for stress response, however this does not come without costs. Responding results in loss of cell type specific function and left unchecked can lead to permanent changes at the cellular and tissue level. This project investigates the role of TGF-beta mediated wound response in early AMD and geographic atrophy. This proposal outlines experiments to 1) Assess whether activation of TGF-beta signaling in RPE cells is associated with macular degeneration, 2) Determine the direct effects of TGF-beta ligands on gene expression and complement reactivity in differentiated RPE, and 3) Assess the influence of the CFH AMD haplotypes on the complement attack of “wounded” RPE. The role of TGF-beta in atrophic AMD has been largely overlooked. Furthermore, the possible intersection between TGF-beta mediated wound response and changes in RPE complement reactivity are particularly intriguing in the context of disease progression. Should TGF-beta signaling be activated in AMD it would become an attractive target for pharmacological intervention.

CUREPSP

Stuart Feinstein 3/2/13-2/28/14 $75,000
Tau Dimerization: A Mechanism of Tau Function and Dysfunction? 509-13
The protein tau is critical for the maintenance of the nervous system. It is also a key contributor to many neurodegenerative diseases, including PSP and CBD. While we understand many functions performed by tau, our understanding of how it actually performs these functions remains primitive. One half of tau, (the “C-terminal half”), can associate with microtubules and regulate their essential behaviors. However, the ability this region of tau to perform these critical functions drops dramatically without the other half of the protein (the “N-terminal half”), which lacks the ability to bind and regulate microtubules. Unfortunately, the mechanism(s) by which the N-terminal half exerts its potent influence(s) upon the C-terminal region remain completely enigmatic. The relevance of normal tau action to pathological tau action in PSP and CBD is that a fragment of tau, derived from the N-terminal half of the protein, has been shown to accumulate in PSP and CBD affected brains and to be neurotoxic, but the mechanism of its neurotoxicity is unknown.

However, we and our collaborators have recently reported biochemical and biophysical evidence indicating that the N-terminal region of tau promotes dimerization and that this dimerization is necessary for normal tau function. This leads to the hypothesis that tau mediated neuronal cell death and dementia in PSP and CBD is mediated by N-terminal tau fragment mediated inhibition of normal tau action, leading to a “loss-of-function” effect resulting in neuronal cell death and dementia. Our work supporting this dimerization hypothesis is based presently solely on in vitro data. The goal of this proposal is to test the hypothesis that tau dimerization mediated by the N-terminal region occurs in neurons and is required for tau action in neurons. By better understanding normal tau action, we will gain completely novel insights into pathological tau action in PSP and CBD.

INTERNATIONAL RETINAL RESEARCH FOUNDATION, INC.

Steven Fisher 10/7/11-11/1/13 $10,000
Creating Brainbow Astrocytes, A New Tool for Studying Retinal and Optic Nerve Astrocytes

The goal of this project was to create a transgenic mouse randomly expressing a set of 4 fluorescent protein genes in retinal astrocytes. Cottage Hospital’s research program has on several occasions provided funds that lead to preliminary data used to submit a larger grant to a federal agency. To date, our lab has completed the design and construction of the brainbow plasmid, a critical part of the project. Using the funds provided by the Santa Barbara Cottage Hospital Research Program, we were able to clone 4 fluorescent proteins into a vector containing the human full-length glial fibrillary acidic protein promoter (GFAP). Subsequently this promoter was sequenced to verify that our design and engineered plasmid was correctly synthesized. Additionally, we were able to test this plasmid on cultured U-87 human glioblastoma cells, immortal mouse retinal astrocytes, and 293T kidney cells for cell-type specificity as well as transfection efficacy. We shipped our completed vector to UC Irvine’s transgenic mouse facility in June for final purification and DNA microinjection on July 3rd, 2012. This mouse will allow us to view retinal...
astrocytes stained with a nearly 90 different fluorescent hues allowing us to study the attributes of individual cells and their reaction to injury.

SANTA BARBARA COTTAGE HOSPITAL

Claudia Gottstein 5/21/13–5/20/14 $15,000
Discovery of specific antibodies against breast cancer stem cells 276

Our over-arching goal is to develop targeted drugs against breast cancer, which improve outcome and prevent recurrence. There is increasing evidence that breast cancer stem cells (BCSC) play a critical role in tumor initiation, metastasis and recurrence, but they are relatively resistant to conventional therapies. A combination treatment of Antibody-drug-conjugates (ADCs) against BCSC with conventional therapy might significantly improve treatment outcome. However, there are currently no suitable antibodies available that could be incorporated into an anti-cancer stem cell ADC. We hypothesize that a new antibody phage display library format will require 1,000–10,000 fold less cells for successful selection of antibodies, compared with existing technology. This is based on lower library size, higher diversity and higher antibody affinity of the new design. If true, this would allow for the first time selection of antibodies against primary breast cancer stem cells from such libraries.

NIH NEUROLOGICAL DISORDERS & STROKE

Scott Grafton 9/1/12–8/31/13 $1,154,135
Spatial and Temporal Scales of Motor Sequence Learning P01 NS044393

This project is a collaborative effort by a team of five motor systems laboratories seeking to probe the mechanisms that underlie the brain’s capacity for learning a new motor skill. The common thread for all groups is to focus on changes that occur within motor circuits of the brain as a new sequential skill is acquired. The work is central to the problem of understanding the mechanisms where practice leads to reorganization of the human motor system in the face of aging, neurodegeneration, stroke or brain injury. Understanding these mechanisms has an impact on the design of therapies directed at preserving function, developing compensatory movements and ultimately, developing novel motor capacity.

NATIONAL SCIENCE FOUNDATION

Michael Gurven 9/1/12–8/31/15 $22,746
Doctoral Dissertation Improvement: Maternal reproductive trade-offs and the duration of exclusive breastfeeding in a natural fertility population BCS-1232370

Exclusive breastfeeding (EBF) for 6 months may significantly reduce risk of infant
morbidity in non-industrialized populations, but is relatively rare. Early provisioning with non–milk solids and liquids may benefit mothers by reducing the time constraints and energetic costs of nursing, which in turn may lead to sustained positive energy balance and earlier resumption of menstruation. However, as early provisioning may negatively impact infant health, early provisioning reflects a trade–off in maternal reproductive interests. The proposed study will investigate how infant condition, maternal condition, number of existing young dependents, support from alloparents, perceptions of lactational performance, and beliefs about early feeding combine to influence EBF duration in a high–fertility population—the Tsimane of Bolivia. The study will examine if early non–milk provisioning by Tsimane mothers results in sustained positive energy balance and earlier resumption of menstruation, possible contributing to high fertility rates.

EISAI COMPANIES (JAPAN)

Mary Ann Jordan                  7/1/12–12/31/13                                $10,422
Mechanism of Suppression of Neuronal Transport and SB120148
Induction of Peripheral Neuropathy by Microtubule
Targeted Anti-cancer Drugs

Using immunofluorescence confocal microscopy, we will determine whether treatment of the axoplasm with microtubule–targeting drugs induces depolymerization or bundling of axonal microtubule. These experiments are key to determining the mechanism by which the drugs exert their inhibitory effects on axoplasmic transport. We believe that this technique should be sufficient to detect bundling or depolymerization of the microtubule network in response to microtubule–targeting drugs. In addition to tubulin, we can use this technique to look for changes in microtubule acetylation (a posttranslational modification of tubulin with known effects on kinesin), or at the distribution of microtubule end-binding proteins. We will verify that a given antibody is usable in squid by Western blots of homogenized axoplasm.

SANOFI-AVENTIS

Mary Ann Jordan                    9/1/12–8/31/14                             $969,158
Mechanism of Action of Cabazitaxel CW70839

Cabazitaxel is a semisynthetic dimethoxy derivative of the modified taxane known as docetaxel. Cabazitaxel has undergone clinical development due to its poor affinity for P–gp, enabling efficacy against docetaxel-refractory prostate cancer. It is also superior to paclitaxel and docetaxel in its ability to cross the blood–brain barrier in animal models. It was approved by the U.S. Food and Drug Administration in 2010 as a new option for patients with prostate cancer, increasing the overall median survival benefit by 2.4 months for men with docetaxel-resistant metastatic castration resistant prostate cancer (CRPC). Its adverse side effects include
neutropenia, diarrhea, and, rarely, neuropathy (Paller, Antonarakis, 2011, Drug Design, Development and Therapy 5:117-124). Since the drug is a taxane, it is believed to promote assembly of tubulin into microtubules and is hypothesized also to suppress microtubule dynamics, to arrest cells in mitosis, and to induce apoptosis. However, there appears to be little published data about its specific interactions with microtubules or its effects on microtubule polymerization or dynamics. Given the clinical efficacy of cabazitaxel and the interesting differences between the parent molecule docetaxel and cabazitaxel, it is essential to elucidate cabazitaxel's basic mode of action on microtubules, both with purified microtubules and in cancer cells, as compared with that of docetaxel.

UNIVERSITY OF ILLINOIS

Kenneth Kosik 9/19/12-8/31/13 $77,359
Dynamic regulation of translation by fragile X mental retardation protein FMRP 2010-06927-01

The Kosik lab will analyze samples of MOV10 immunoprecipitates to detect the associated mRNAs and small non-coding RNAs. This analysis will be performed on our SOLiD sequencer. A data analysis pipeline that includes BioScope will be used to infer the results and follow up validation will be performed.

THE LARRY L. HILLBLOM FOUNDATION

Kenneth Kosik 1/1/08-12/31/13 $367,566
The Pathobiology of Tau Inclusions 2007-A-003-NET

Diseases, like Alzheimer’s and frontotemporal dementia, with intracellular inclusions such as neurofibrillary tangles, represent a broad category in pathology, and yet the mechanism of their formation is poorly understood. While most research directed toward the biochemical mechanism of tangle formation focuses on a class of enzymes called kinases, the aims of this project are built on the hypothesis that the accumulation of tau protein is due to a failure of the protein degradation machinery.

UC IRVINE

Geoffrey Lewis 1/1/13-12/31/16 $993,757
Retinal progenitor cells for treatment of retinitis pigmentosa 2013-2919

Retinitis pigmentosa is an inherited, degenerative eye disease that causes severe vision impairment and blindness. The goal of our CIRM funded proposal “Human retinal progenitor cells as candidate therapy for retinitis pigmentosa” was to use retinal progenitor cells, injected into the eyes of rats with a similar genetic eye disease, to “deliver” survival factors and prevent the retina from degenerating. To date we have identified the injected cells using histological markers (antibodies)
to stem cells and have documented that they survive for months after injection into the eyes. In addition, we have shown that the stem cells greatly slow the degeneration of the retina without causing any adverse affects. The work is the result of an ongoing collaboration with colleagues at UC Irvine. In addition, UCSB undergraduates contributed greatly to the project by doing much of the routine histology on the rat eyes.

NIH GENERAL MEDICAL SCIENCES

Benjamin Lopez                  8/1/12-7/31/13                                $49,214
Determining How Tau and EB1 Affect Microtubule Structure & Kinesin Translocation

This project will determine how the presence of disease-linked microtubule associating proteins (MAPs) influence and regulate microtubule (MT) structure. In particular, we will investigate two important MAPs: tau and EB1. Normal tau binds to and regulates the growth and stability of MTs while certain tau mutations are known to cause neurological disease. EB1 binds to the growing plus-end of MTs, coordinates other end binding proteins and possibly changes the lattice structure of MTs. A wide array of cancer cells has been found to over express EB1. Using a custom-built multimodal microscope capable of simultaneous single-molecule fluorescence and optical trapping, this project will determine the effects of MAPs on MT stiffness and assess the ability of MAP-coated MTs to support kinesin-based transport. These investigations will first consider tau mutations known to cause frontotemporal dementia with parkinsonism–17, and progressive supranuclear palsy, devastating neurological disorders. The hypothesis that the GTP-rich EB1-coated plus end of a MT has differing stiffness and supports different rates of kinesin translocation compared to the rest of the MT will also be tested. These complimentary data sets will provide critical quantitative insight into the role of MT mechanics and structure in health and disease. In the short term, this project will answer fundamental biological questions about the role of MAPs in regulating the organization and function of the MT cytoskeleton. Ultimately, answers to these questions will contribute to improved medical diagnostics and/or treatments of diseases linked to MT and MAP dysfunction, including neuropathies, Alzheimer’s disease and cancer.

Craig Montell                     4/1/13-3/31/14                               $295,290
TRPA1: A Polymodal Sensor for Aversive Stimuli         7R01GM085335-06

The long-term goal of the research is to use the fruit fly, Drosophila melanogaster, as an animal model to unravel the mechanisms through which insects respond to sensory cues, ranging from changes in temperature to insect repellents. These questions are of potential relevance to the control of insect pests, since mosquitoes that spread diseases are attracted to humans through thermosensory, visual...
and chemical cues. Aversive temperatures and chemical repellents deter insects. Therefore, understanding the mechanisms underlying avoidance behavior may provide important insights into insect pest control. A key group of receptor proteins that sense environmental stimuli are Transient Receptor Potential (TRP) cation channels. Among the 13 Drosophila members, TRPA1 is of particular note as it is a detector for a wide array of noxious sensory inputs, including slightly warm or hot temperatures, insect repellents, and excessive light. Here, we propose to dissect the molecular, cellular and behavioral mechanisms through which TRPA1 allows larvae and adult flies to elude aversive stimuli. To accomplish our goals, we are employing a multidisciplinary approach, using a combination of molecular genetics, biochemistry, cell biology, electrophysiology and behavioral approaches.

NIH DEAFNESS & OTHER COMMUNICATION DISORDERS

Craig Montell 7/1/13-6/30/14 $308,869
Molecular Genetics of Contact Chemosensation 7R01DC007864-08

The long-term goal of this research project is to clarify the molecular mechanisms underlying the detection and discrimination of chemicals through contact chemosensation in the fruit fly, Drosophila melanogaster. Contact chemosensation allows flies to distinguish sweet from bitter molecules, as well as nonvolatile pheromones. Insect gustatory organs express a diversity of candidate molecular detectors. These include gustatory receptors (GRs), TRP channels, ionotropic receptors (IRs) and odorant binding proteins (OBPs), the latter of which promote the detection of chemicals by receptor proteins. However, the functions of most of these candidate gustatory receptors and binding proteins are unknown, or are understood poorly. This project is focusing on dissecting the mechanisms underlying contact chemosensation in flies using a multidisciplinary approach that includes electrophysiology, behavior, genetics, and cell biological approaches. During the last few years, the concept that GRs are required broadly for sensing sugars and bitter-tasting compounds has been confirmed. However, the biochemical functions of GRs are unclear. A long-term goal of this research is to apply the findings to the control of insect pests that spread disease.

NIH NATIONAL EYE INSTITUTE

Craig Montell 5/1/13-4/30/14 $363,375
Rhodopsins: from biosynthesis and degradation to unconventional functions 7R01EY008117-25

The goal of the research is to use the fruit fly, Drosophila melanogaster, as an animal model to unravel the molecular mechanisms underlying the biosynthesis, turnover and non-classical functions of rhodopsins. Rhodopsin is comprised of an opsin protein and a vitamin A-derived chromophore, which senses light. Among the most common forms of retinal degeneration are those that result from defects in
the visual cycle (retinoid cycle)—an enzymatic pathway required for regeneration of the chromophore. Until recently it was thought that flies do not employ a visual cycle, since the chromophore does not normally release from photoactivated rhodopsin. However, some rhodopsin is internalized and the opsin gets degraded, thereby releasing the chromophore. We have recently made the discovery that flies use a visual cycle to regenerate the released chromophore. To accomplish our goals, we are employing a multidisciplinary approach using a combination of genetic, cell biological, electrophysiological, molecular and biochemical techniques. The long-term goals of these studies are to 1) uncover mechanisms underlying the retinal degenerations that result from defects in the visual cycle with the ultimate goal of discovering new therapeutic approaches, and 2) uncover the roles of the enigmatic extra-retinal opsins.

Craig Montell 5/1/13-4/30/14 $309,783
Regulation of TRP channels and visual transduction 7R01EY010852-20

The long-term goal of this research project is to define the mechanisms through which the TRP channels in photoreceptor cells are activated and regulated in response to light. This project focuses on Drosophila phototransduction, which functions through a phospholipase C (PLC)-dependent signaling system, and culminates with Ca2+ and Na+ influx, via the TRP and TRPL channels. There exists a large family of mammalian TRPs, including channels in the intrinsically photosensitive retinal ganglion cells (ipRGCs) that are gated through a cascade that has notable parallels with fly phototransduction. The specific goals of this project are to answer major questions in Drosophila phototransduction concerning the mode of activation and regulation of the TRP channels. To accomplish our goals, we are employing a multidisciplinary approach, using a combination of molecular genetics, biochemistry, cell biology, and electrophysiology. The goal of aim 1 is to identify the molecule that directly gates the TRP and TRPL channels. Prior to activation of these channels, PLC causes hydrolysis of PIP2 to generate IP3, DAG and H+. However, despite the >20 years that have elapsed since the identification of the Drosophila TRP channels, the precise activation mechanism is not known. We recently identified a DAG metabolite that increased in concentration in a light-dependent manner. We suggest that the studies that are the focus of this project are significant because they offer to resolve the mechanisms by which the TRP channels in photoreceptor cells are gated, localized and regulated. We also suggest that these studies will provide the framework for answering similar questions relevant to the channels in the ipRGCs, which contribute to light-induced circadian rhythms, sleep patterns and rudimentary image formation in the absence of rods and cones.

SIGMA XI, THE SCIENTIFIC RESEARCH SOCIETY

Britney Pennington 5/1/13-5/31/14 $2,500
Characterization of a Novel Substrate for Stem Cell Based Therapies for Ocular Disease SB130138
Age-related macular degeneration (AMD) is the leading cause of blindness in the elderly and is characterized by the death of the retinal pigment epithelium (RPE), the cell layer located behind the retina. The RPE maintains the health of the primary cells responsible for vision, the photoreceptors. As AMD progresses, the RPE degrade, which causes the death of the photoreceptors and a debilitating loss of sight. Human embryonic stem cells (hESCs) can generate a limitless source of RPE for cellular therapies, therefore efforts to derive RPE from hESCs to graft into AMD patients are under development. However, to manufacture cells for clinical use, it is desirable for procedures to be performed under defined conditions sans animal products (xeno-free), and many studies are still optimizing the derivation with these parameters. Synthemax-II is a novel, xeno-free, biomimetic RGD-containing peptide copolymer designed for cell culture, but to date, no report has characterized hESCs nor RPE grown on Synthemax-II. Furthermore, Synthemax-II is compliant with good manufacturing practices (GMP) and can be used to coat synthetic scaffolds such as parylene in order to transplant hESC-derived RPE into AMD patients.

NIH NATIONAL EYE INSTITUTE

Benjamin Reese 1/1/13-12/31/13 $338,053
Development of Retinal Bipolar Cells R01 EY019968

This research program is identifying the molecular and genetic determinants controlling the natural variation in nerve cell number, examining the populations of synaptically connected photoreceptors, bipolar cells and amacrine cells in the retina. We are also determining how such variation in afferent and target cell number modulates the dendritic morphology of the post-receptoral cells. This program will, consequently, clarify the developmental events and their underlying mechanisms that produce the functional architecture and connectivity of the retina. These studies will contribute to our understanding of retinal development and degeneration, and will enlighten our approach in developing treatments for retinal disease, particularly where the latter seek to re-establish connectivity following cell replacement therapy.

NIH CHILD HEALTH & HUMAN DEVELOPMENT

Joel Rothman 6/1/12-5/31/13 $285,638
Specification and Differentiation of Endoderm in C. elegans R01 HD062922

We are continuing our studies on how cell division and growth are controlled by investigating the cellular components that switch dividing cells into non-dividing cells with specialized functions. These processes are critically important in the genesis of cancer and are uncontrolled in growing tumor cells. The project is providing training for graduate students and undergraduate researchers who are learning molecular genetic and cell biological experimental methods that effectively address these problems.
William Smith 7/1/12–6/30/13 $325,978
Morphomic analysis of a simple chordate R01 HD059217

This proposed collaborative project will investigate fundamental processes driving chordate embryogenesis. The project will combine the skills and expertise of two research groups: one that works in the area of developmental biology, and the other in the area of image analysis and computer vision. The goal of the project is to take a whole-embryo approach to investigating morphogenesis in live embryos in all 4 dimensions (x, y, z, and t). Specifically, we will collect and analyze confocal microscopy images to derive quantitative data on the division, shape, volume and movements of all cells in both selected developing organs and in whole embryos.

NIH GENERAL MEDICAL SCIENCES

William Smith 8/1/12–7/31/13 $271,253
Exploring Planar Cell Polarity in a Novel Invertebrate Chordate System R01 GM088997

The formation of organs and tissues in the developing embryo requires coordinated action of multiple cells. Cells are not uniform structures, but rather have distinct sides, a property we call polarity. For example, cells in an organ may adhere tightly to a substrate with one face, while actively secreting on another face. We use a simple model organism in which the organs are composed of only tens to hundreds of cells to investigate the cellular mechanisms by which cells sense directionality and coordinate polarity while they assemble into organs.

SANTA BARBARA COTTAGE HOSPITAL

Thomas Weimbs 11/1/12–10/31/13 $12,500
An unexpected mechanism underlying genomic stability defects in polycystic kidney disease 271

Genomic instability may contribute to renal cyst growth in polycystic kidney disease (PKD) but the underlying mechanism is unknown. We have discovered that polycystin-1, the protein affected in PKD, interacts with DNA-PK, a protein required for maintaining genomic stability. We hypothesize that loss of the polycystin-1/DNA-PK interaction in PKD patients leads to genomic instability and renal cyst growth.

NIH DIABETES, DIGESTIVE & KIDNEY DISEASES

Thomas Weimbs 9/21/12–7/31/13 $219,525
A Novel Role of Syntaxin 3 as a Transcription Regulator R21 DK095248

SNARE proteins mediate membrane fusion events in virtually all cellular membrane trafficking pathways. We have discovered an unexpected, novel function of the
SNARE protein syntaxin 3 (Stx3). Stx3 normally has a C-terminal trans-membrane anchor and is involved in trafficking to the apical plasma membrane domain of polarized epithelial cells. We found that Stx3 undergoes cleavage at an extremely conserved glutamine residue which removes its trans-membrane domain resulting in a soluble fragment, Stx3(1-225). Furthermore, a novel splice-isoform of Stx3 (Stx3E) lacks the trans-membrane anchor, and is expressed in human kidneys. Both, the cleavage fragment and Stx3E (collectively called “soluble Stx3”) bind to the nuclear import factor RanBP5, target to the nucleus and co-activate several transcription factors including ETV4. ETV4 is required for branching morphogenesis in kidney development, and associated with carcinogenesis and tumor metastasis. We found that kidneys from Autosomal Dominant Polycystic Kidney Disease (ADPKD) patients express a small Stx3 fragment – consistent with soluble Stx3. We hypothesize that cleavage and transcriptional regulation in the nucleus is a novel function that may be a common feature of syntaxin members of SNARE proteins. This may be a novel signaling mechanism that transduces information from cytoplasmic membrane trafficking events to the nucleus to affect changes in gene expression. If correct, this would introduce a new paradigm of SNARE function. More specifically, we hypothesize that soluble Stx3 plays a role in the regulation of renal epithelial morphogenesis, carcinogenesis and ADPKD.

UC CANCER RESEARCH COORDINATING COMMITTEE

Thomas Weimbs 7/1/12-6/30/14 $55,000
A Novel Role of Syntaxin 3 as a Transcription Regulator SB130052
Implicated in Epithelial Morphogenesis and Carcinogenesis

SNARE proteins mediate membrane fusion events in virtually all cellular membrane trafficking pathways. We have discovered an unexpected, novel function of the SNARE protein syntaxin 3 (Stx3). Stx3 normally has a C-terminal trans-membrane anchor and is involved in trafficking to the apical plasma membrane domain of polarized epithelial cells. We found that Stx3 undergoes cleavage at an extremely conserved glutamine residue which removes its trans-membrane domain resulting in a soluble fragment, Stx3(1-225). Furthermore, a novel splice-isoform of Stx3 (Stx3E) lacks the trans-membrane anchor, and is expressed in human kidneys. Both, the cleavage fragment and Stx3E (collectively called “soluble Stx3”) bind to the nuclear import factor RanBP5, target to the nucleus and co-activate several transcription factors including ETV4. ETV4 is required for branching morphogenesis in kidney development, and associated with carcinogenesis and tumor metastasis. We found that kidneys from Autosomal Dominant Polycystic Kidney Disease (ADPKD) patients express a small Stx3 fragment – consistent with soluble Stx3. We hypothesize that cleavage and transcriptional regulation in the nucleus is a novel function that may be a common feature of syntaxin members of SNARE proteins. This may be a novel signaling mechanism that transduces information from cytoplasmic membrane trafficking events to the nucleus to affect changes in gene expression. If correct, this would introduce a new paradigm of SNARE function. More specifically, we hypothesize that soluble Stx3 plays a role in the regulation of renal epithelial morphogenesis, carcinogenesis and ADPKD.
morphogenesis, carcinogenesis and ADPKD.

ENDOCYTE

Thomas Weimbs                           5/1/13-4/30/14                       $191,563
Pre-clinical efficacy of Folate-Conjugated mTOR                             SB130109
Inhibitors and related compounds in Polycystic Kidney Disease

Our previous work has established that the mTOR signaling pathway is aberrantly activated in polycystic kidney disease (PKD), and that the mTOR-specific inhibitor rapamycin is highly effective in reducing renal cyst growth and preserving renal function in mouse models of PKD 1,2,3. However, clinical trials to test the efficacy of mTOR inhibitors in ADPKD patients had disappointing results 4,5 which suggested that the clinically feasible doses of these mTOR inhibitors are insufficient to inhibit mTOR in the target tissue. Due to the strong side effects of high-dose rapamycin, increasing the treatment dose does not appear to be a viable approach. These results strongly support the need to target rapamycin to polycystic kidneys in order to inhibit renal cyst growth while circumventing most of the side-effects. In collaboration with Endocyte we have demonstrated that the folate receptor is expressed in epithelial cells in the disease state. We have characterized FC-rapamycin (FC-rapa) in vitro and demonstrated that FC-rapa inhibits mTORC1 in a dose-dependent manner that requires expression of the folate receptor. We have further shown that treatment with FC-rapa in vivo effectively ameliorates the cystic renal phenotype and preserves kidney function in the bpk mouse model of PKD. Biochemical analyses have demonstrated that in vivo treatment with FC-rapa inhibits the activity of the mTOR pathway in the target organ. Lower doses of FC-rapa effectively treat the disease while exhibiting increased renal specificity. These results were recently published 9. Taken together, these results suggest that FC-rapa represents a promising targeted therapeutic treatment for PKD. The purpose of the proposed research is to further validate these results using an independent, human-orthologous PKD mouse model, and to compare the potential efficacy between several related compounds to identify a lead compound.


PUBLICATIONS


34: Winking J, Stieglitz J, Kurten J, Kaplan H, Gurven M. Polygyny among the


Publications


### Statistical Summary

1. **Academic personnel engaged in research:**
   - Faculty: 25
   - Professional Researchers (including Visiting): 3
   - Project Scientists: 9
   - Specialists: 30
   - Postdoctoral Scholars: 22
   - Postgraduate Researchers: -
   - TOTAL: 89

2. **Graduate Students:**
   - Employed on contracts and grants: 28
   - Employed on other sources of funds: 4
   - Participating through assistantships: -
   - Participating through traineeships: 4
   - Other (specify): -
   - TOTAL: 36

3. **Undergraduate Students:**
   - Employed on contracts and grants: 22
   - Employed on other funds: 4
   - Number of volunteers, & unpaid interns: 24
   - TOTAL: 50

4. **Participation from outside UCSB: (optional)**
   - Academics (without Salary Academic Visitors): 5
   - Other (specify): -

5. **Staff (Univ. & Non-Univ. Funds):**
   - Technical: 1
   - Administrative/Clerical: 5

6. **Seminars, symposia, workshops sponsored:** 6

7. **Proposals submitted:** 50

8. **Number of different awarding agencies dealt with:** 52

9. **Number of extramural awards administered:** 31

10. **Dollar value of extramural awards administered during year:** $53,375,483

11. **Number of Principal Investigators:** 41

12. **Dollar value of other project awards:** $3,258,898

13. **Number of other projects administered:** 30

14. **Total base budget for the year (as of June 30, 2013):** $306,141

15. **Dollar value of intramural support:** $67,357

16. **Total assigned square footage in ORU:** 14,682

17. **Dollar value of awards for year (2013 Total):** $6,885,269
## BUDGET SUMMARY

### PERMANENT NR02

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<thead>
<tr>
<th>Item</th>
<th>Appropriation</th>
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<tbody>
<tr>
<td>Academic Salaries</td>
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<td>Co-Directors</td>
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<td>Supplies &amp; Expense</td>
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<td>Travel &amp; Equipment</td>
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<tr>
<td>Other: Copier Recharge</td>
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<td><strong>Total 2011-2012</strong></td>
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<td><strong>Adjusted total 2011-2012</strong></td>
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<td><strong>Carry forward/(overdraft)</strong></td>
<td>($119,758)</td>
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### TEMPORARY

Intramural Funding*—Funds allocated directly to Organized Research Unit

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<th>Person/Project—Source of funds</th>
<th>Appropriation</th>
<th>Expense</th>
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<td>Carry forward (overdraft from prior year)</td>
<td>$121,723</td>
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<td>447636-07427 Indirect Cost Return 2012-2013</td>
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<td>447633-07427 c/f Raven Microscopy Match</td>
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<tr>
<td>447636-07427 5th Floor Renovation BIO II</td>
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<td>447636-07427 OR Contribution 5th Flr Reno</td>
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<td>447636-07427 Coffey CIRM match 5th flr reno</td>
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<td>447636-07427 COE contribution SB Co Sci Fair</td>
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<td>447636-07427 NRI ICR CF</td>
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<td>447636-19900 Indirect Cost Return 2012-2013</td>
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<td>447636-19900 c/f Raven Microscopy Match</td>
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<td>Carry forward 19900 to 19941</td>
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<td>447636-19900 Kydland Salary</td>
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<td>447636-19900 Feinstein IACUC FY 2012/13</td>
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<td><strong>Total Appropriations/Expenses</strong></td>
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<td><strong>Carry forward/(overdraft)</strong></td>
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### Recharge/Income Account 447636-62190

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<th>Item</th>
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<td>Staff Salaries</td>
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<td>General Assistance</td>
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## BUDGET SUMMARY

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<tr>
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<th>INCOME</th>
<th>EXPENSE</th>
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<td><strong>S&amp;E</strong></td>
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<td><strong>Benefits</strong></td>
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<td><strong>Other: Equipment &amp; Facilities Unallocated</strong></td>
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<td><strong>Total Recharge Income/Expenses</strong></td>
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<td><strong>Carry forward/(overdraft)</strong></td>
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<table>
<thead>
<tr>
<th>Other Income (specify source and use)</th>
<th>INCOME</th>
<th>EXPENSE</th>
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<tbody>
<tr>
<td><strong>Donations/Gifts/Endowments</strong></td>
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<tr>
<td><strong>Various Donors</strong></td>
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<td><strong>Gifts</strong></td>
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<td><strong>Endowments</strong></td>
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<td><strong>Total Other Income/Expenses</strong></td>
<td>$4,279,449</td>
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| **Total Funding/Expenses for FY 2012-2013** | $5,206,436 | $1,986,985 |
| **Total carry forward/(overdraft)**        | $3,219,451 |          |

Live neurons visualized with confocal microscopy. Nuclei are stained with DAPI (blue). Membrane localization of p35 is observed with anti-p35 primary and Cy3 second antibodies (red). Credit: John Lew
ADVISORY COMMITTEE

MARK BRZEZINSKI, EEMB
PETER COFFEY, NRI/MCDB
SCOTT GRAFTON, Psychological + Brain Sciences
THOMAS HARRIMAN, Community
JANICE HARTOCH-TAYLOR, Development
RICHARD LEHMAN, Santa Barbara Cottage Hospital
JOANN KUCHERA-MORIN, Media Arts + Technology
B.S. MANJUNATH, Electrical + Computer Engineering

FYL PINCUS, Biomolecular Science + Engineering
ART ROSENBLATT, Olympus
THOMAS WEIMBS, CHAIR, NRI/MCDB

EX-OFFICIO

THERESA PEÑA, NRI
STUART FEINSTEIN, NRI
KENNETH KOSIK, NRI

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JUDY CUSHING, Payroll/Purchasing Assistant
KAREN CISNEROS, Administrative/Purchasing Manager
THERESA PEÑA, Business Officer
STUART FEINSTEIN, Co-Director
KENNETH S. KOSIK, Co-Director
MAX MCCUMBER, Purchasing Assistant
KATHLEEN MCINTOSH, Purchasing Assistant
JEN MESSECAR, Assistant to Kenneth Kosik
LAURA SUSIN, Contract + Grants Manager
BEE JAY YOKOI, Payroll Manager

TECHNICAL STAFF

MARY ARCILA, Sequencing Facility Assistant Director
HUNTER BUCHANAN, Computer Support
GEOFFREY LEWIS, Microscopy Support
MARY RAVEN, Microscopy Director
## PRINCIPAL INVESTIGATORS

<table>
<thead>
<tr>
<th>Name</th>
<th>Title</th>
<th>Department</th>
</tr>
</thead>
<tbody>
<tr>
<td>Don Anderson</td>
<td>Research Scientist</td>
<td>NRI</td>
</tr>
<tr>
<td>Osnat M. Ben Shahar</td>
<td>Researcher</td>
<td>Psychological + Brain Sciences</td>
</tr>
<tr>
<td>Dennis O. Clegg</td>
<td>Professor</td>
<td>MCDB</td>
</tr>
<tr>
<td>Adele Doyle</td>
<td>Assistant Researcher</td>
<td>NRI</td>
</tr>
<tr>
<td>Stuart C. Feinstein</td>
<td>Professor</td>
<td>MCDB</td>
</tr>
<tr>
<td>Steven K. Fisher</td>
<td>Professor</td>
<td>NRI</td>
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<tr>
<td>Osnat M. Ben Shahar</td>
<td>Researcher</td>
<td>Psychological + Brain Sciences</td>
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<td>NRI</td>
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<tr>
<td>Stuart C. Feinstein</td>
<td>Professor</td>
<td>MCDB</td>
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<tr>
<td>Claudia Gottstein</td>
<td>Adjunct Assistant Professor</td>
<td>MCDB</td>
</tr>
<tr>
<td>Scott Grafton</td>
<td>Professor</td>
<td>Psychological + Brain Sciences</td>
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<tr>
<td>Mary Ann Jordan</td>
<td>Research Scientist</td>
<td>NRI</td>
</tr>
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<tr>
<td>Tod Kippin</td>
<td>Associate Professor</td>
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<tr>
<td>Kenneth S. Kosik</td>
<td>Harriman Professor</td>
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<td>Todd Kippin</td>
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<tr>
<td>Tonya Kydland</td>
<td>Project Scientist</td>
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<tr>
<td>John Lew</td>
<td>Associate Professor</td>
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<tr>
<td>Geoff Lewis</td>
<td>Research Scientist</td>
<td>NRI</td>
</tr>
<tr>
<td>Dzwokai “Zach” Ma</td>
<td>Assistant Professor</td>
<td>MCDB</td>
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<tr>
<td>Craig Montell</td>
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<td>Denise Montell</td>
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<tr>
<td>Stanley M. Parsons</td>
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<td>Chemistry + Biochemistry</td>
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<tr>
<td>Monte Radeke</td>
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</tr>
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<td>Benjamin E. Reese</td>
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<td>Psychological + Brain Sciences</td>
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<td>Joel Rothman</td>
<td>Professor</td>
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<tr>
<td>Charles E. Samuel</td>
<td>C.A. Storke II Professor</td>
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<tr>
<td>William Smith</td>
<td>Professor</td>
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<tr>
<td>Karen Szumlinski</td>
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<td>Psychological + Brain Sciences</td>
</tr>
<tr>
<td>Megan T. Valentine</td>
<td>Assistant Professor</td>
<td>Mechanical Engineering</td>
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<tr>
<td>Carol Vandenberg</td>
<td>Professor</td>
<td>MCDB</td>
</tr>
<tr>
<td>Thomas Weimbs</td>
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<td>MCDB</td>
</tr>
<tr>
<td>Leslie Wilson</td>
<td>Professor</td>
<td>MCDB</td>
</tr>
</tbody>
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SPECIAL THANKS

The time-lapse cover image of the steps to the beach just below the Bio II building is courtesy of Gabe Luna. Thanks also go out to Geoff Lewis, John Lew, Peter Allen, George Foulsham, Mary Raven, Israel Hernandez, Panuakdet Suwannatat, Tobias Hollerer, and Steven Fisher for use of their images. We are also grateful to Business Officer Jeanie Cornet, who retired at the end of the year, for her 32 years of service at UCSB and time at NRI.

Please follow us on Twitter and Facebook for current updates on NRI research news.

@UCSB_NRI

UCSB NRI