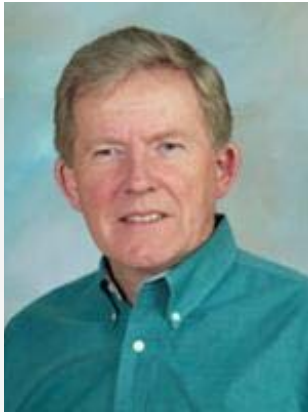


# Integrative Biology & Pharmacology

## Newsletter



## Dr. O'Neil Receives Two Grants

Roger O'Neil, Ph.D.

### AHA Grant-In-Aid Project

Roger G. O'Neil. American Heart Association, Grant-In-Aid. *Flow-Dependent TRPV Channels and Mechanical Control of Collecting Duct Function*. 1 July 2014 – 30 June 2016.

The goal of the project is to elucidate the micromechanical control and molecular basis of local calcium signaling events in regulating downstream, flow-dependent, mechanosensitive effector pathways in kidney cortical collecting duct (CCD) cells of the late distal tubule. While many processes in the kidney display a mechanosensitivity to tubular flow (e.g., calcium signaling; nucleotide and G-protein signaling, cell growth, and water and solute excretion), flow-dependent processes are often at the basis of numerous pathophysiological states. This is particularly true for mechanosensitive calcium signaling events in late distal tubule cells which can lead to markedly altered potassium excretion patterns (calcium-dependent potassium excretion) resulting in potassium wasting states leading to hypokalemia, loss of body fluids and low blood pressure. The focus of this project is to understand the molecular basis of the upstream mechanosensitive calcium signaling events in specific cells of the cortical collecting duct that can lead to altered potassium secretion patterns by these cells. The emphasis is on elucidating the basis of the mechanosensitive signaling pathways, the identity of the calcium-permeable TRPV channels underlying the calcium signal, and the calcium signaling complex that allows the channels to function in a mechanosensitive manner in the CCD cells. The project broadens to understand the specific role of the various cells types in the CCD (principal cells, intercalated cells, etc) and the expression patterns of the mechanosensitive signaling

molecules in each of the cell types. Finally, once the role of the cell types and TRPV channels are clarified, the studies will be extended to define the role of the cell types and the expressed TRPV channels in controlling potassium secretion events in each of the cell types to establish their participation in the pathogenesis of dysfunctional potassium balance states. The outcome of these studies will provide new insights into our understanding of the molecular basis of flow-sensitive calcium signaling in the kidney, the importance of this process in controlling downstream effector pathways, and the potential identification of new therapeutic targets for calcium-dependent pathologies of the kidney.

### NIH R01 Project

Roger G. O'Neil. National Institutes of Digestive, Diabetes, and Kidney Diseases. *Regulation of Flow-Induced Potassium Wasting*. 15 August 2014 -31 July 2017.

The overall goal of the project is to elucidate the mechanism by which flow in the nephron regulates potassium secretion, often leading to potassium wasting states, by the cells of the cortical collecting ducts (CCD) of the late distal tubule. It is well known that states of enhance fluid delivery to the late distal tubule stimulates potassium secretion which results in excess potassium excretion by the kidney. Such flow-dependent potassium wasting states are wide-spread occurring in conditions of volume expansion, loop-diuretic use and in salt-losing tubulopathies, such as Bartter and Gitelman syndromes. It can quickly lead to hypokalemia, volume depletion, and low blood pressure. The mechanism of this flow-dependent potassium wasting is thought to involve flow-induced calcium influx into cells (the basis of the AHA project, above) which, in turn, activates calcium-dependent potassium excretion. This induced potassium excretion appears, in part, to be due to activation of maxi-potassium

*continued from page 1*

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# Departmental News & Events

channels (BK channels) in the late distal tubule. However, the mechanism of the induced potassium secretion remains controversial and poorly understood. We have recently identified the expression of a new class of calcium-dependent potassium channel in the kidney, the “small conductance” potassium channels (the SK channels). The SK channels are unique in that they are highly sensitive to cellular calcium levels due to the constitutive binding of a calcium-sensing protein, calmodulin, to a C-terminal site of the channel. The emphasis of the study is to determine which calcium-sensitive potassium channels underlie the phenomena of flow-dependent potassium excretion, how these channels interact to lead to enhanced potassium secretion in the CCD, and, ultimately, how these channels are so tightly regulated by small changes in intracellular calcium levels and

the upstream calcium-permeable TRPV channels (AHA project, above). The project makes extensive use of genetically modified animal models of the potassium channels, the calcium-permeable TRPV channels, and the upstream flow-sensitive purinergic signaling pathways to provide an indepth assessment of the flow-dependent potassium-wasting states and the interacting signaling pathways that give rise to these pathological conditions. The outcome of these studies will provide new insights into our understanding of the molecular basis of flow-sensitive potassium excretion in the kidney and will identify potential new therapeutic targets for development of treatment strategies in potassium wasting pathologies.



Kartik Venkatachalam has received a gift from the Homejoy Foundation <https://www.homejoy.org/> for Amyotrophic Lateral Sclerosis, known as ALS or Lou Gehrig’s Disease Research. The research the lab is doing on the ALS related project involves heavy usage of a microscopy facility available at UT Houston. Specifically, they study alterations in calcium levels in neurons expressing ALS related mutations and also examine the consequences of these mutations on motor neuron development and function.

***Congratulations to Dr. Venkatachalam and his lab!***

## Teaching Excellence Awardees

May is designated as teaching excellence month for the Medical School. Below are this year’s recipients of the Dean’s Teaching Excellence Awards by department.

Rebecca Berdeaux, Ph.D.  
Lenard M. Lichtenberger, Ph.D.  
Roger G. O’Neil, Ph.D.  
Kartik Venkatachalam, Ph.D.

Raymond J. Grill, Ph.D.  
David S. Loose, Ph.D.  
Gary C. Rosenfeld, Ph.D.  
Edgar T. Walters, Ph.D.

***Congratulations to these wonderful professors!***

The **IBP Newsletter** is published quarterly by the department and distributed to faculty, staff and students. An electronic copy is available on the IBP website at <http://ibp.med.uth.tmc.edu/>

**Chair, IBP**  
Dr. John Hancock

**Vice Chair, IBP**  
Dr. Roger O’Neil

**Director of Management Services**  
Monica Gardner

**Editor**  
Anne Dybala

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# Departmental News & Events

## New Members of the Team



**Joseph Lorent**

Post Doctoral Research  
Fellow

Ilya Levental, PhD



**Maria Mendoza-  
Rodriguez**

Research Assistant II

Rebecca Berdeaux, PhD



**Li Wei Rachel Tay**

Research Assistant I

Guangwei Du, PhD



**Karolina Tulodziecka**

Post Doctoral Research  
Fellow

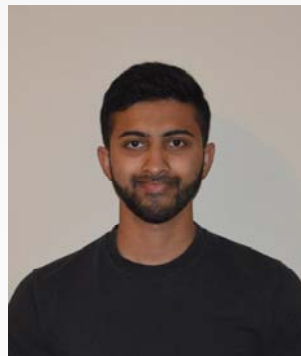
Ilya Levental, PhD



**Emily Huang**

Visiting Student Trainee

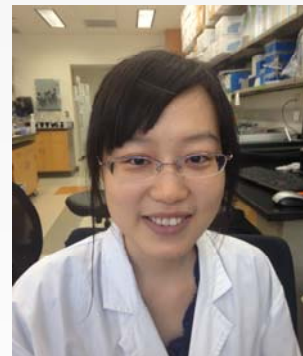
Lenard Lichtenberger,  
PhD



**Mashfee Khan**

Visiting Student Trainee

Shane Cunha, PhD

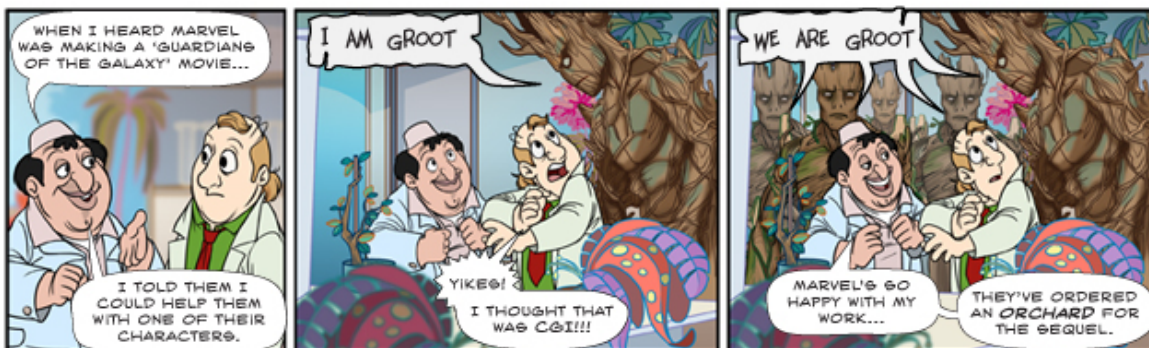


**Pa Wu**

Visiting Student

Guangwei Du, PhD

Lab Bratz Episode #393 "I am Groot"



Lab Bratz by Dunphy & Soares

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<http://www.LabBratz.com>

# Faculty Spotlight



Ching-On Wong



Kartik Venkatachalam, Ph.D.

## Venkatachalam Lab

microscopy, we found that a TRPV channel called Inactive (*lav*) is required in presynaptically for the development of NMJ synapses. *In vivo*  $Ca^{2+}$  measurements using the  $Ca^{2+}$  sensor, GCaMP5, revealed that *lav* is required for the maintenance of presynaptic resting  $Ca^{2+}$ . *lav*-dependent presynaptic  $Ca^{2+}$  activates the phosphatase, calcineurin, which is essential for the stabilization of presynaptic microtubules—a critical determinant of the number of presynaptic termini. Thus, loss of *lav* induces microtubule destabilization resulting in fewer presynaptic termini. These defects were rescued by expression of a human ortholog of *lav* called TRPV1, suggesting conservation of function between flies and humans. By determining presynaptic resting  $Ca^{2+}$  levels, *lav* also affects the number of SVs released in response to a given electrical stimulus, and therefore regulates the gain of synaptic transmission. In summary, we present evidence for a novel mechanism explaining the ability of synapses to exquisitely transform both the spatial dynamics and gain of electrical signals as they move from one neuron to another, which might contribute to the rich computational capacity of the brain.

### Reference:

**Wong, CO.**, Chen, K., Lin, YQ., **Chao, Y.**, Duraine, L., Lu, Z., Yoon, WH., Sullivan, J.M., Broadhead, G.T., Sumner, C.J. Lloyd, T.E., Macleod, G.T., Bellen, H.J., and **Venkatachalam, K.** (2014) A TRPV Channel in *Drosophila* Motor Neurons Regulates Presynaptic Resting  $Ca^{2+}$  Levels, Synapse Growth, and Synaptic Transmission *Neuron*, **In Press**

We recently had a paper get accepted in the prestigious neuroscience journal *Neuron*. The following is a brief description of our study:

Communication between neurons occurs at intercellular contacts called synapses. Electrical signals at presynaptic termini elicit cytosolic  $Ca^{2+}$  elevation leading to the release of neurotransmitter-laden synaptic vesicles (SVs). The released neurotransmitter then generates electrical activity in the postsynaptic neurons leading to transsynaptic flow of electrical activity. However, simple propagation of electrical activity between neurons does not explain the vast computational capacity of the brain. *Therefore, we hypothesized that synapses also possess electrical activity-independent mechanisms to sculpt the spatial characteristics and strength of synaptic transmission.* To test this hypothesis, we used the *Drosophila* neuromuscular junction (NMJ) as a model synapse, and screened for the synaptic functions of TRP channels—a cohort of cation channels not activated by electrical activity. By visualizing NMJ synapses by light

## Cancer Chain in the Membrane

The University of Texas at Austin conducted an interview with Dr. Alex Gorfe as well as his post-doctoral fellow, Priyanka Srivastava regarding supercomputing simulations crucial to the study of Ras protein in determining anticancer drugs. The story can be found at the following link: <https://www.tacc.utexas.edu/news/feature-stories/2014/cancer-chain-in-the-membrane> .

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# Levental Lab Publishes New Work

Ilya Levental, Ph.D.



Members of the Levental lab recently published their work titled "Membrane raft association is a determinant of plasma membrane localization" in the *Proceedings of the National Academy of Sciences*. Co-primary authors Dr. Kandice Levental and incoming CRB graduate student

for distributing components between various cellular lab locations. The panel of mutants showed a striking correlation between their affinity for lipid rafts and their subcellular localization. The "rafty" mutants all sorted to the plasma membrane, in accordance with expectations from the wild-type protein. However, all variants whose raft association was disrupted were mis-sorted to lysosomes – the cell's waste bin. As it turned out, this mis-targeting was a consequence of the failure of these mutants to be "rescued" by recycling back to the plasma membrane from the endocytotic pathway. This observation revealed membrane microdomains to be a key mechanism by which membranes and their associated transmembrane proteins are recycled back to the cell surface after internalization.

Barbara Diaz-Rohrer set out to examine the structural bases of the association between transmembrane proteins and plasma membrane microdomains known as lipid rafts. By generating and testing a large panel of transmembrane domain mutants, Kandice and Barbara definitively identified the length of a protein's transmembrane domain as being a key determinant of lipid raft targeting, with longer transmembrane domains imparting preference for raft domains. This observation was the first experimental proof of a long-standing theoretical hypothesis, and therefore a significant breakthrough for the field.

However, what started as a directed examination of structural protein characteristics led to a discovery of a mechanism by which cells use membrane microdomains

Because of the impact of the observations – and because scientists, like children, love pictures of bubbles – an image from the paper was selected for the cover of the June 10 issue of *PNAS*.

The discovery from the Levental lab sparked a new research interest among the co-authors; how are lipid rafts involved in biosynthetic sorting? An extensive literature search led to the publishing of a review article in *Biochimica et Biophysica Acta – Biomembranes* entitled "Rafting through traffic: Membrane domains in cellular logistics". This review presents evidence for the role of lipid rafts in subcellular membrane sorting, but also highlights how little is currently understood about this fascinating topic.

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## Dr. Gorfe's Work Featured

Priyanka Srivastava, Postdoctoral Research Fellow



The Texas Advanced Computing Center has recently highlighted Dr Gorfe's work as a featured news story: <https://www.tacc.utexas.edu/news/feature-stories/2014/cancer-chain-in-the-membrane>. The story revolves around the primary focus of Dr. Gorfe's lab, unraveling the atomic details of Ras signaling, and was inspired by their recent paper in the *Journal of Physical Chemistry Letters* (2014, 5(8): 1457-1462)

# UTHealth research: What squid can teach us about the purpose of pain

Published June 19, 2014



Injured squid (*Doryteuthis pealeii*) behave with extra vigilance after even a minor injury.  
Photo by Roger Hanlon

Many of us have felt that lingering sense of anxiety or pain after enduring an accident or injury. Now researchers at UTHealth Medical School have the first evidence that there may be a good reason for that heightened sensitivity—at least in the case of squid versus fish.

Squid that behave with extra vigilance after experiencing even a minor injury are more likely to live to see another day, according to a report that appeared in the May 8 issue of the Cell Press journal *Current Biology*.

The findings suggest that behaviors that appear counterproductive might sometimes have an advantage when viewed from an evolutionary perspective, the researchers say.

“Many pain researchers and clinicians consider long-lasting sensitization and associated pain to be maladaptive, rarely considering whether it might be evolutionarily adaptive,” says Edgar T. Walters, PhD, professor of integrative biology and pharmacology at the UTHealth Medical School. “Intense pain is certainly maladaptive in many human contexts when modern medical care is available. However, this study provides the first direct evidence for the plausible evolutionary hypothesis that sensitization mechanisms – which in some animals are known to promote pain – have been shaped by strong evolutionary selection pressures, including pressures from

predators.” Walters is also on the faculty of The University of Texas Graduate School of Biomedical Sciences at Houston.

Walters and UTHealth researcher Robyn Crook, PhD, first author of the study, had an interest in the evolution of mechanisms associated with lasting pain. They realized they had a rare opportunity to study related behaviors through the interaction of squid with their natural black sea bass predators.

“Squid perform a stepwise and quite stereotyped sequence of defensive behaviors when they feel threatened, often starting when the predator is still quite distant,” Crook explains. “Because we can grade their responses from low to high levels of perceived danger, it gives us a way to measure how injured and normal squid assess danger differently as a predator approaches them and initiates an attack.”

In their observations of squid and black sea bass swimming freely in laboratory tanks, the researchers found that squid could get around perfectly well after a slight injury to one of their arms. But they were still at a considerable disadvantage under those circumstances, as the bass continued to pursue injured squid over their uninjured fellows, and from greater distances, too. Once the injured squid became the subject of pursuit, they acted more defensively as well.

What the researchers found most intriguing was the survival value of heightened vigilance to injured squid. They found that squid treated briefly with anesthetic (which prevented their nervous systems from registering their injuries) failed to respond with enhanced defensive behaviors that otherwise would have helped protect them.

The sensory activity driving the squids’ heightened vigilance may be similar to sensory processes that trigger pain after injury in humans, but the researchers say there is no evidence that squid feel what humans would consider pain. Nevertheless, the findings in squid suggest a new way to think about our human reactions to injury and pain, the researchers say.

“If we can understand more about what the natural, ‘intended’ purpose of nociceptive sensitization is, we might be in a better position to find new ways to treat its pathological expression in humans,” Crook said.

*Press release courtesy of Cell Press*

[Inside UT Health](#)



## Xiaodong Cheng, Ph.D.

Xiaodong Cheng has been appointed to the Editorial and Advisory Board of Molecular Pharmacology.  
**Congratulations!**

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# Student Awards & Activities

## GSEC Selects 9 Finalists for Scholarships

The [Graduate Student Education Committee](#) reviewed applications and selected nine finalists to receive the Dean's Research Scholarship.

Finalists for 2014 are **Rita Sirrieh**, [Biochemistry and Molecular Biology](#); **Veronica Wells Rowlett**, [Microbiology and Molecular Genetics](#); **Natalie Sirisaengtaksin**, [Neurobiology and Anatomy](#); **Cameron Brand**, [Integrative Biology and Pharmacology](#); **Stuart Red**, Neurobiology and Anatomy; **Meredith Rees Rodriguez**, [Internal Medicine](#); **Swarna Ramaswamy**, Biochemistry and Molecular Biology; **Melissa Reardon Robinson**, Microbiology and Molecular Genetics; and **Maria Camila Montealegre**, Microbiology and Molecular Genetics.

These \$2,500 awards were established to recognize the achievements of Medical School graduate students who have achieved distinction in their academic and research programs.

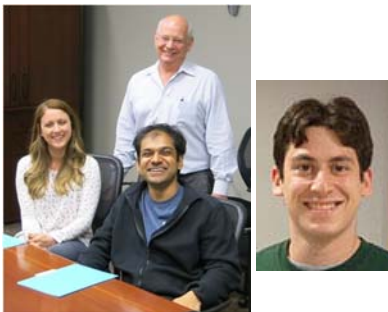
The awarding of these scholarships is based in part on the student's ability to design, conduct, and critically analyze the results of their research. The award is contingent upon the students' ability to skillfully communicate the significance of their findings at the award ceremony and symposium Sept. 18, 2014 in MSB 5.001. Everyone is welcomed to attend.

<https://med.uth.edu/news/gsec-selects-9-finalists-for-scholarships/>

## CRB Retreat's Outstanding Students!

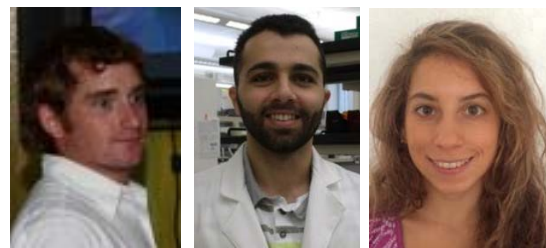


### Student Oral Presentation: *Three Way Tie for 1st Place!*



Randi Stewart, Dhananjay Thakur, Cameron Brand

### Student Poster Awards:



1st Place:  
Andrew  
Peters

2nd Place:  
Muayad  
Almahariq

3rd Place:  
Leandra  
Mangieri

### Fellows Oral Presentation Winners:



1st Place:  
Mykola Mamenko

2nd Place:  
Jialie Luo

### Fellow Poster Awards:



1st Place:  
Blanca Diaz-  
Rohrer

2nd Place:  
Bangxing Hong



# Mykola Mamenko Wins AHA Award

Mykola Mamenko, Postdoctoral Research Fellow



Kidneys are paired organs in the body of mammals and higher animals, performing a number of vital homeostatic functions by the means of uropoiesis (or urine formation). The main physiological role of the kidneys

lies in the excretion of final products of nitrogen-bearing compounds metabolism, regulation of water-electrolyte balance in the body, maintenance of stable osmolality and constant volume of bodily fluids. The key regulator of water homeostasis in the body is a hormone – vasopressin. It prevents excessive loss of water by increasing its re-uptake in the kidneys. Inability of the kidney to properly retain water in response to vasopressin causes a devastating pathology of nephrogenic Diabetes insipidus. This disorder is characterized by intense thirst and production of large amounts of urine, usually more than 3-15 liters per day. Vasopressin promotes fusion of the vesicles containing Aquaporin 2 water channels with the luminal plasma membrane of collecting duct (CD) cells. Kidneys conserve water as it enters the cells and returns to the circulation. Both fusion of vesicles containing water channels and water transport in the collecting duct are  $\text{Ca}^{2+}$ -dependent. Recent studies indicate that the required  $\text{Ca}^{2+}$  is released from intracellular stores, such as endoplasmic reticulum (ER). However,  $\text{Ca}^{2+}$  release from ER cannot account for sustained intracellular  $\text{Ca}^{2+}$  elevations observed in response to vasopressin. Factors contributing to these intracellular  $\text{Ca}^{2+}$  elevations in CD cells remain poorly understood. In his project “...” Mykola hypothesizes that the main role here belongs to ER  $\text{Ca}^{2+}$  sensor – STIM1. STIM1 senses the decrease of  $\text{Ca}^{2+}$

concentration in the ER, translocates towards the plasma membrane, where it stimulates  $\text{Ca}^{2+}$  permeable channels to induce/allow  $\text{Ca}^{2+}$  entry into the cell. This process is called store-operated calcium entry (SOCE). The entering  $\text{Ca}^{2+}$  can be subsequently re-uptaken by the ER  $\text{Ca}^{2+}$  pumps. However, molecular determinants of SOCE in the CD cells remain obscure and little is known about the relation of SOCE to vasopressin-induced signal in the CD cells. To probe this Mykola will study SOCE, vasopressin-induced  $\text{Ca}^{2+}$  signal and urine concentrating ability in CDs from two closely related rat strains, one of which has a defective STIM1 protein. This defect in the ER  $\text{Ca}^{2+}$  sensor is caused by a mutation in STIM1 gene resulting in the production of truncated protein, which is unable to properly activate plasma membrane  $\text{Ca}^{2+}$ -channels. Mykola has found that SOCE is functional in the CD cells from rats with intact STIM1, but is disrupted in animals with defective  $\text{Ca}^{2+}$  sensor. Moreover, CD cells with defective STIM1 respond to vasopressin with only a transient elevation of intracellular  $\text{Ca}^{2+}$ . They are unable to sustain the response to vasopressin, in contrast to the cells with intact STIM1. Finally, Mykola’s preliminary data indicate that rats with defective STIM1 protein excrete less concentrated urine. Overall, Mykola’s project reveals a novel mechanism regulating vasopressin-dependent water reabsorption in the kidney’s collecting ducts. The obtained results indicate that SOCE is an important molecular switch maintaining vasopressin-induced water transport in the CD. Disruption of STIM1 will, likely, impair  $\text{Ca}^{2+}$  signaling in CD cells and compromise the ability of kidney to conserve water, leading to a pathological syndrome of nephrogenic Diabetes insipidus.

## Students Excel



Sara Prijic was awarded a post-doctoral fellowship in the UTHealth Innovation in Cancer Prevention Research Training Program funded by the Cancer Prevention and Research

Institute of Texas (CPRIT). This award will fund Sara to develop innovative approaches to eliminating metastases and deaths from breast cancer.



Muayad Almahariq, a MD/PhD student, has successfully defended this PhD thesis on August 5 and is back in medical school for clinical rotations. We wish Muayad

continued success.



Jian Xiong defended his Master’s thesis on July 24th. We wish Jian continued success.



# Proposals & Awards

Anne Dybala & Deborah Brougher

Twenty-six proposals were submitted by the Department of Integrative Biology & Pharmacology in the third quarter of Fiscal Year 2014 by Drs. Gorfe, Berdeaux, Breton, Chang, Cheng, Denicourt, Dessauer, Grill, Hu, Li, Lichtenberger, Walters, Yang, and Zhu.

Nine proposals were awarded this quarter. Faculty receiving awards include Drs. Cheng, Dessauer, Du, Frost, Hu, Lichtenberger, and Walters. This brings the total number of proposals this year to 65 and

awards to 19, a success rate of about 29%.

~Data provided by Deborah Brougher, Sr. Grants and Contracts Specialist

**Proposals Submitted FY2014 3rd QTR**

# Subt'd	Federal	Federal Continuation	Non Profit	Total
10	13,710,623.00			<b>13,710,623.00</b>
5		1,673,229.00		<b>1,673,229.00</b>
6			482,352.00	<b>482,352.00</b>
<b>21</b>	<b>13,710,623.00</b>	<b>1,673,229.00</b>	<b>482,352.00</b>	<b>15,869,204.00</b>

**Awards Received FY2014 3rd QTR**

# Rec'd	State	Federal	Federal Continuation	Total
2	450,000.00			<b>450,000.00</b>
5		2,691,647.00		<b>2,691,647.00</b>
4			1,019,149.00	<b>1,019,149.00</b>
<b>11</b>	<b>450,000.00</b>	<b>2,691,647.00</b>	<b>1,019,149.00</b>	<b>4,160,796.00</b>

## New Awards

Awards received during the third quarter of Fiscal Year 2014 include:

**Chang, Jeffrey** CPRIT *Collaborative Training of A New Care of Innovative Cancer Prevention Researchers*

**Cheng, Xiodong** NIGMS *Epac/cAMP-GEF, A Novel Intracellular cAMP Receptor*

**Cheng, Xiodong** NIGMS *Novel Pharmacological Probes Targeting Exchange Proteins Activated by cAMP (EPAC)*

**Cheng, Xiodong** NIGMS *Preclinical Development of Novel Rickettsiosis Therapeutics Targeting EPAC1*

**Dessauer, Carmen**

**Du, Gwangwei** National heart Lung & Blood Institute *Phospholipase D2 regulation of vascular smooth muscle cell migration.*

**Du, Gwangwei** CPRIT *Elucidating Mechanisms of Altered Lipid Metabolism in Cancer.*

**Frost, Jeffrey** NCI *Role of the RhoGEF Net1 in breast cancer cell motility and metastasis*

**Hancock, John** CPRIT *K-ras Spatiotemporal Dynamics Novel Therapeutic Target*

**Lichtenberger, Lenard** NCI *Effects of Antiplatelet Drugs on Colon Cancer in the Elderly*

**Walters, Terry**

# IBP Seminar Series

~Directed by Drs. Shane Cunba and Kartik Venkatachalam



**September 8, 2014**

**Oleh Pochynyuk, Ph.D.**  
Integrative Biology and Pharmacology  
University of Texas, Medical School, Houston  
**"TRPV4-Mediated Mechanosensitivity in the Distal Nephron. Implications for Polycystic kidney disease"**

Host: Roger O'Neil, Ph.D.



**September 15, 2014**

**Ross A. Poché, Ph.D.**  
Molecular Physiology and Biophysics  
Baylor College of Medicine  
**"Ronin Influences Retinal Proliferation through Transcriptional Regulation of Mitochondrial Function"**

Host: Ghislain Breton, Ph.D.



**September 22, 2014**

**Kenneth Hargraves, D.D.S., Ph.D.**  
Department of Endodontics  
University of Texas Health Science Center,  
San Antonio, Texas  
**"Iron in the Fire: The Role of Oxidized Lipids as TRPV1 Agonists in Inflammatory Pain"**

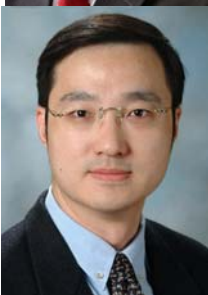
Host: Edgar T. Walters, Ph.D.



**September 29, 2014**

**Dongfang Liu, Ph.D.**  
Pathology and Immunology  
Baylor College of Medicine  
**"Immunological Synapse and Diseases"**

Host: Michael Zhu, Ph.D.



**October 6, 2014**

**Zhimin (James) Lu, M.D., Ph.D.**  
Neuro-Oncology  
MD Anderson Cancer Center  
**"The Warburg effect and beyond"**

Host: Guangwei Du, Ph.D.



**October 13, 2014**

**Kevin Pflieger, Ph.D.**  
Molecular Endocrinology and Pharmacology  
Harry Perkins Institute of Medical Research  
**Title of Talk:**

Host: Agnes Schonbrunn, Ph.D.



**October 20, 2014**

**Mong-Hong Lee, Ph.D.**  
Molecular and Cellular Oncology  
MD Anderson Cancer Center  
**Title of Talk:**

Host: Jeff Frost, Ph.D.



**October 27, 2014**

**Gregory Dussor, Ph.D.**  
Behavioral and Brain Sciences  
The University of Texas at Dallas  
**"Meningeal nociceptive signaling and the pathophysiology of migraine"**

Host: Edgar T. Walters, Ph.D.

**November 3, 2014**

**Richard Anderson, Ph.D.**  
Molecular and Cellular Pharmacology  
University of Wisconsin, Madison  
**Title of Talk:**

Host: Guangwei Du

**November 10, 2014**

**Alexandra Newton, Ph.D.**  
Pharmacology and Physiology  
University of California-San Diego  
**Title of Talk:**

Host: Cell and Regulatory Biology Students

**NOTE**

*Seminars are held on Mondays at 4:00 PM in MSB 2.135, unless otherwise noted. For information and questions, please contact Catrina Stevens at [catrina.m.stevens@uth.tmc.edu](mailto:catrina.m.stevens@uth.tmc.edu) or 713-500-7536.*

# IBP Seminar Series

~Directed by Drs. Shane Cunba and Kartik Venkatachalam



**November 17, 2014**

**Guillermina (Gigi) Lozano, Ph.D.**

Department of Genetics  
MD Anderson Cancer Center

**“Unraveling the p53 Tumor Suppressor Pathway”**

**Host:** Jeffrey Frost, Ph.D.

**December 1, 2014**

**Hugo Bellen**

Molecular and Human Genetics  
Baylor College of Medicine

**Title of Talk: TBA**

**Host:** Kartik Venkatachalam, Ph.D.

**December 8, 2014**

**Janos Peti-Peterdi**

Physiology, Biophysics and Medicine  
University of Southern California

**Title of Talk: TBA**

**Host:** Oleh Pochynyuk, Ph.D.

**December 15, 2014**

**Sarah Veatch**

Department of Biophysics  
University of Michigan

**Title of Talk: TBA**

**Host:** Ilya Levental, Ph.D.

**January 5, 2015**

**Robert Dantzer, D.V.M., Ph.D.**

Symptom Research, Division of Internal  
Medicine  
MD Anderson Cancer Center

**Title of Talk: TBA**

**Host:** Edgar T. Walters

**January 12, 2015**

**Catherine Collins**

Molecular, Cellular, and Developmental  
Biology  
University of Michigan

**Title of Talk: TBA**

**Host:** Kartik Venkatachalam

**January 26, 2015**

**Stephen Russ Price, Ph.D.**

Department of Nephrology  
Emory University School of Medicine

**Title of Talk: TBA**

**Host:** Yi-Ping Li, Ph.D.

**February 2, 2015**

**Kirill Martemyanov**

Molecular Signaling Section  
Scripps Research Institute, Florida

**Title of Talk: TBA**

**Host:** Carmen Dessauer, Ph.D.

**February 9, 2015**

**Anthony Means**

Molecular & Cellular Biology  
Baylor College of Medicine

**Title of Talk: TBA**

**Host:** Agnes Schonbrunn, Ph.D.

**February 23, 2015**

**Jurgen Wess**

Molecular Signaling Section  
National Institutes of Health

**Title of Talk: TBA**

**Host:** Rebecca Berdeaux, Ph.D.

**NOTE**

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# IBP Calendar of Events

## Meetings:

### **Administrative Staff Meetings, 2:30-3:30 PM, MSB 4.136**

October 14, December 9, February 10



### **CRB Meetings, 12-1 PM, Room 4.100**

September 4, October 2, November 6,  
December 4, January 1, February 5



### **Faculty Coffee/Tea, 10-11 AM, MSB 4.100**

September 3, 10, 17, 24; October 1, 8,  
15, 22, 29; November 5, 12, 19, 26;  
December 3, 10, 17



## Dates to Remember:

**September 11:** Patriot Day

**September 17:** Citizenship Day

**September 26:** Native American Day

**October 13:** Columbus Day

**October 16:** Boss's Day

**October 31:** Halloween

**November 2:** Daylight Savings Ends

**November 11:** Veterans Day

**November 27-28:** Thanksgiving Day-*The University will be closed for Official Business*

**December 7:** Pearl harbor Remembrance Day

**December 24-January 1:** Winter Holiday-*December 24, 25 & January 1-The University will be closed for Official Business; December 28-31 & January 2 are skeleton crew holidays*

**December 25:** Christmas Day-*The University will be closed for Official Business*

**December 31:** New Year's Eve-*The University will be closed for Official Business*

**January 1:** New Year's Day-*The University will be closed for Official Business*

**January 19:** Martin Luther King Day-*The University will be closed for Official Business*