April 2014 • Volume II, Issue 4 http://ibp.med.uth.tmc.edu/

Integrative Biology & Pharmacology Newsletter

A Random Walk in Pharmacology

Xiaodong Cheng, Ph.D.

Xiaodong Cheng, Ph.D.

Professor

When Dr. Xiaodong Cheng started his independent scientific career back in 1999, drug discovery was merely a concept and distant dream to him. Dr. Cheng was a bit puzzled, then, when he was hired onto the faculty of the department of Pharmacology and Toxicology, an area he had almost zero background in. Perhaps, it was meant to be. But in reality, it took more than a decade of hard-work, set-backs and serendipity for him to complete the transition from a simple-minded biochemist to a simple-minded pharmacologist.

Dr. Cheng has spent most of his scientific career studying a molecule called cAMP (a cyclic nucleotide) and its biological functions. cAMP was discovered by Dr. Earl Sutherland in 1953 as a "second messenger" transmitting the signal of hormones such as epinephrine (a.k.a adrenaline), which cannot cross the cell membrane into the cell. cAMP is important for many biological functions, particularly under stress conditions, such as the famous "fight-or-flight" response associated with adrenaline. Because chronic stress is often associated human diseases it is not surprising that many of the current therapeutics on the market target the cAMP pathway.

In 1998, a major breakthrough happened in the field of cAMP signaling studies. A new family of cAMP receptor proteins, called EPAC1 and EPAC2, were discovered. This was quite a surprise considering that the other cAMP receptor family (protein kinase A, PKA) was discovered more than three-decades earlier. It made sense then, for Dr. Cheng to pick EPAC as the main subject of research for his new laboratory in 1999. Of course, many other laboratories around the world also seized this exciting opportunity and rushed into the field. Dr. Cheng's group quickly found out that although activated by the same signal, cAMP, EPAC and PKA can mediate the opposite functions of cAMP in cells. The net outcome of cAMP signaling is dependent upon the dynamic abundance and distribution of intracellular Epac and PKA, providing a mechanism for a more precise and integrated control of the cAMP signaling pathways in a spatial and temporal manner. In addition, studies from his lab also suggest EPAC1 is quite a free spirited protein, as it wanders around all over the place in cells, an early indication that it is a multi-functional protein.

Dr. Cheng remembered the comment from a preeminent guest speaker during the early days of his EPAC study. "Knockout the protein out in mouse and if you don't see any phenotype, the protein is not important and I would move on to something else." At the time, it made perfect sense. So he patiently waited for others to show the effects of deleting the EPAC genes in mice as he had neither the resources nor the expertise to take on the project himself. Five years passed and no report of EPAC knockout studies surfaced. Finally in 2006, running out of patience and unable to pass up such an interesting opportunity, Dr. Cheng convinced Dr. Ju Chen, a leading expert in mouse genetics whom Dr. Cheng has known since his postdoctoral years at UCSD, to join forces to create the EPAC1 and 2 knockout mice. As fate would have it, both EPAC1 and 2 knockout mice, and even the double knockout mice, turned out to be perfectly normal. You could probably sense the disappointments both Drs. Chen and Cheng had then. After a period of disbelief, Dr. Cheng came to the realization that since cAMP is a stress signal, perhaps the mice looked normal because they lived in a nearly perfect laboratory environment. Indeed, finding the proper stress conditions was the key. In 2013, both Drs. Chen and Cheng published their first papers using the EPAC knockout mice to demonstrate that EPAC may play a role in heart disease or obesity, respectively.

Another major challenge of studying EPAC signaling is the lack of pharmacological agents to sort out the individual biological functions of EPAC and PKA, since both proteins are

Continued on page 3

Departmental News & Events

New Website

Lisa Byrd

Originally, it was thought that our new website would up and running by the end of January. That didn't happen. Now, rather than give you another date, let's just say we are presently waiting on the Office of Communications to fix a couple of glitches, prior to the roll out. We are currently in the last stages of editing. It is hoped that the web site will be ready for viewing before the end of April.

Once the web site is ready, faculty will need to visit the site and preview their information. Any changes or updates to be made will be taken care of as quickly as possible.

The new web site is attractive and user friendly. We will have the ability to see how many visitors to the site there are, which pages are most often viewed, and even what countries the visitors are in when they view it.

The process of updating the site has been simplified. Needless to say, this is the most attractive feature to some of us.

Notification will be sent when the site is published. Please be on the lookout for the email.

We hope you will be as excited by the new site and its updated appearance and functionality as we are.

*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

Please contact any of our dedicated staff for whatever assistance you may require:

Deborah Brougher
Sr. Contracts & Grants Specialist
Deborah.Brougher@uth.tmc.edu
713.500.6322

Lisa Byrd
Senior Administrative Coordinator
Lisa.Byrd@uth.tmc.edu
713.500.7508

Sandy Cegielski
Senior Administrative Coordinator
Sandy.Cegielski@uth.tmc.edu
713.500.7514

Cordelia Conley
Administrative Assistant III
Cordelia.P.Conley@uth.tmc.edu
713.500.7459

Anne Dybala
Administrative Services Officer III
Anne.L.Dybala@uth.tmc.edu
713.500.7502

Monica Gardner
Director, Management Operations
Monica.Gardner@uth.tmc.edu
713.500.7516

Trish McFarland
Coordinator I, Educational Programs
Patricia.McFarland@uth.tmc.edu
713.500.5470

Naomi Pinkney
Sr. Executive Assistant
Naomi.Pinkney@uth.tmc.edu
713.500.7547

Catrina Stevens
Administrative Coordinator
Catrina.M.Stevens@uth.tmc.edu
713.500.7536

UTHealth™
The University of Texas
Health Science Center at Houston
Medical School

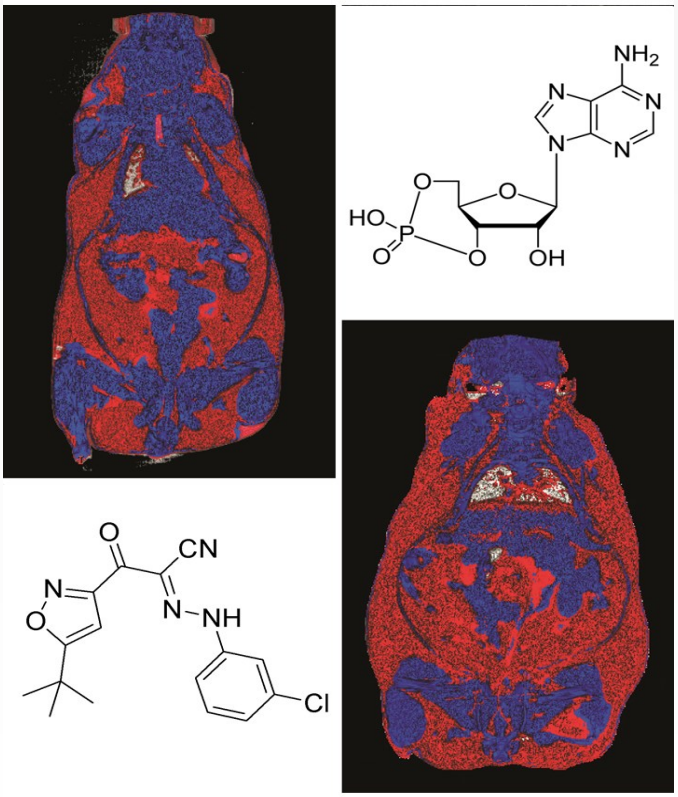
Continued from page 1

ubiquitously expressed and activated by the same signal, cAMP. One approach was to modify the cAMP molecule in hoping to generate cAMP analogs that have selectivity for EPAC or PKA. These designer cAMP analogs are useful when used as agonists. However, this approach has one major limitation for designing EPAC antagonists because cAMP analogs can bind and inhibit other cAMP-binding proteins in cells, in particular PDEs, which are enzymes critical for metabolizing and desensitizing cyclic nucleotide signaling. Inhibiting PDEs increases cyclic nucleotide concentration in cells and counters the effects of the antagonist. To circumvent the problem, Dr. Cheng decided to search for EPAC inhibitors from the chemical library with random chemical structures, a difficult task considering the vastness of the search space. Fortunately, pharmaceutical companies have perfected the art of high throughput screening (HTS), allowing researchers to sieve through thousands chemical compounds in a short period of time. Dr. Cheng's lab was able to find a cAMP analog that increases its fluorescent signal dramatically when bound to EPAC2 protein. This provided a quick and simple way to screen compounds competing for cAMP binding. With this robust HTS assay, Dr. Cheng's lab carefully screened a 15,000 compound library and was able to identify several chemicals with the ability to specifically inhibit EPAC proteins, but not PKA. Two compounds can even distinguish EPAC2 from EPAC1.

After more than one decade, the EPAC research field has matured. Recently, a plethora of studies using whole organisms have demonstrated the important role of EPAC proteins in cancer, obesity diabetes, chronic pain etc. With the genetic and pharmacological tools that they lab has built, Dr. Cheng's lab is in a great position to test the role of EPAC protein in various disease models and the therapeutic potential of EPAC specific inhibitors. For example, Dr. Cheng and his collaborators have recently shown that the deletion of EPAC1 in mice protects them from an ordinarily lethal dose of rickettsiae. Most importantly, a small-molecule EPAC inhibitor can prevent and suppress rickettsial infection. These results demonstrate EPAC1 is a potential target for the prevention and treatment of fatal rickettsioses.

Moving forward, Dr. Cheng's lab has developed a second generation HTS assay, which allows them to screen both EPAC agonists and antagonists in a single assay. Working with the NIH Chemical Genomics Center (NCGC) under the National Center for Advancing Translational Sciences (NCATS), parallel quantitative HTS screens of EPAC1 and EPAC2 against a library of more than 480,000 compounds was performed, and led to the discovery of multiple new hits with diverse chemical structures. These new chemical scaffolds will be invaluable for developing the next generation of EPAC inhibitors with improved pharmacological properties.

It has taken more than a decade of collective struggle and the perseverance of every past and current member of the Cheng lab, as well as many collaborators, to reach their current position. While the road ahead will be unfamiliar and even more difficult, Dr. Cheng is very excited about the future. He is particularly encouraged by the news that the NIH will fund a collaborative preclinical development project of EPAC inhibitors involving an industry partner in the Stanford Research Institute. Perhaps, ending up in a Pharmacology department was not just an accident at all. As a student of pharmacology, Dr. Cheng now thinks that a so-called "normal phenotype" for a knockout mouse model is the best phenotype that a pharmacologist can ask for, as it indicates the toxicity associated with inhibiting the target will be minimal and is ideal for developing therapeutics. The challenge, as well as the fun, is to ask the right questions and design the proper experiments to tease out the real functions of the target.



cAMP, an Epac inhibitor and CT-scans of Epac and wild-type knockout mice

Departmental News & Events

New Members of the Team

Zhelong Liu

Post-Doctoral Research Fellow

Dr. Li

Melissa Rodriguez-Tallant

Post-Doctoral Research Fellow

Dr. Schonbrunn

Ming Cai, M.D.

Visiting Scientist

Dr. Du

Bangxing Hong, Ph,D.

Instructor

Dr. Du

Tony Li-Geng

OTVS Student Trainee

Dr. Lichtenberger

Emily Huang

Pre-Baccalaureate Trainee

Dr. Lichtenberger

Alexis Bavencoffe

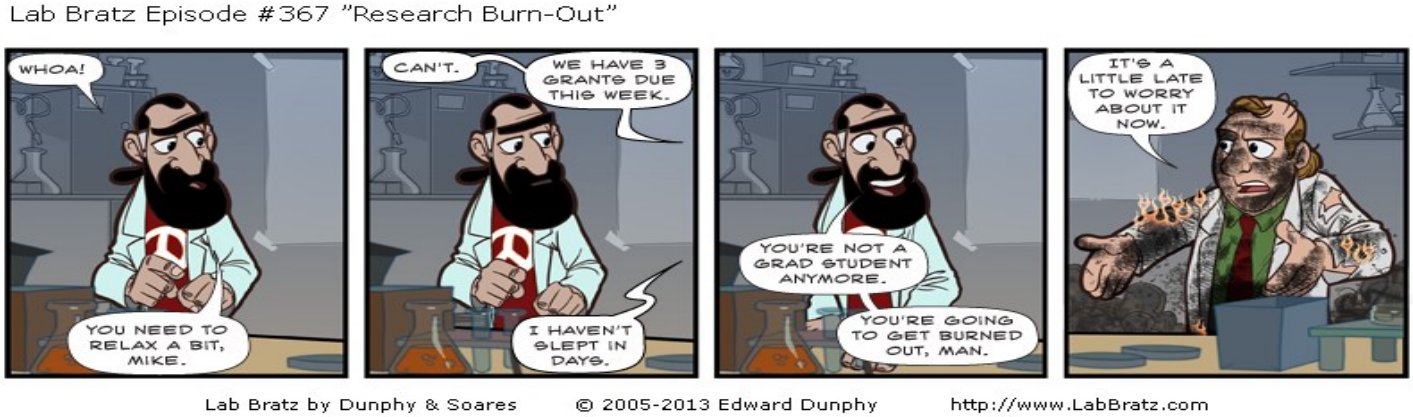
Post-Doctoral Research Fellow

Dr. Dessauer

Xiao Liang, M.D.

Visiting Scientist

Dr. Du



Did You Know?

Faculty Travel Restrictions on Outstanding Effort Cards

Faculty members who are paid from sponsored projects are required to certify effort cards in a timely manner to ensure compliance with HOOP 93 and federal regulations. At UTHealth, effort certification is completed semi-annually and a certification period of 30 days is provided for faculty to certify their effort for the prior six month reporting period. Travel restrictions will be imposed on individuals with outstanding effort cards effective February 21, 2014. The restrictions will include travel using any university funds or using university time.

These changes will assist in demonstrating to others that we are good stewards of public funds and the contributions to research made by UTHealth employees.

Thank you for your assistance with these new procedures.

For additional information, please refer to the **Effort Reporting Website** or contact the Effort Reporting Team at effort@uth.tmc.edu or 713-500-3392.

HOOP 75, Cost Sharing

HOOP 88, Buildings Pathways Use

HOOP 26, Achievement Award Program: Changes made to clarify responsible parties for achievement awards.

HOOP 30, Sick Leave: Revisions made to comply with new state law allowing employees to use sick leave to attend educational activities of their children. Also clarifies use of sick leave to care for child under employee's legal guardianship and for adoption of a child.

HOOP 55, Student Immunizations and Health Records: Revisions made to comply with new state law lowering the age requirement for the bacterial meningitis vaccine. Changes also made to match current vaccination form used by the university.

HOOP 85, University Closure for Emergency, Disaster or Severe Weather: Changes made to clarify procedures for official closure of the university.

HOOP 100, Worker's Compensation Insurance: Clarifies scope of the policy. Time period for reporting an injury to the university has been shortened to 7 calendar days. Clarifies that employees may be responsible for costs if health care provider is not part of UT System's IMO Health Care Network.

HOOP 125, Observers (now Visitors): Changes made to shorten policy and link to more detailed procedures on Office of Academic and Research Affairs website.

HOOP 147, Handling Legal Processes: Revisions made to clarify procedures for handling legal processes.

HOOP 185, Administrative Leave Revisions made to clarify scope and incorporate new definition of disability adopted by the most recent Texas Legislature (House Bill 489).

UT Health Discount Program

The UTHealth Discount Program continues to grow as 17 new participating businesses have been added to the list. Employees, students, residents and fellows can enjoy discounts on dining, entertainment, wellness and more by checking out the **UTHealth Discount Program website**.

Changes to HOOP Policy

The following policies were approved at the November 2013 and February 2014 University Executive Council meeting and have been updated in the HOOP. Please click on the links below to review the updated policies.

HOOP 23, Flexible Work Arrangements

HOOP 33, Reimbursement and Time Off for Job-Related Training and Education

HOOP 37, Sick Leave Pool Program

HOOP 69, Indirect Costs

Faculty Spotlight

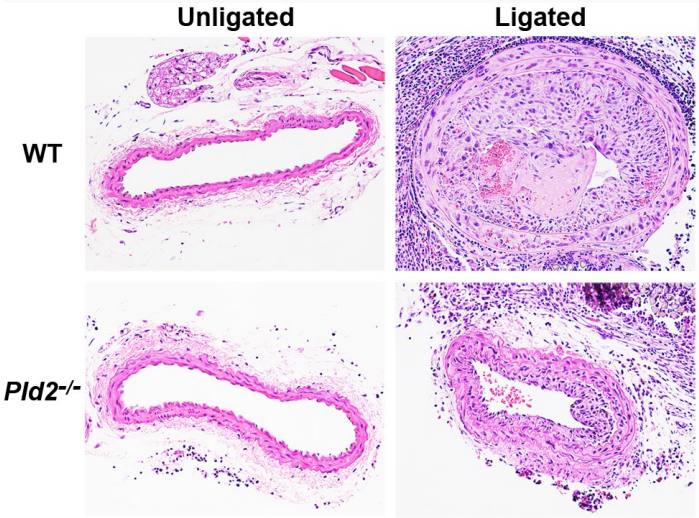
Guangwei Du, Ph.D.

Two Research Grants Awarded to the Du Laboratory

Guangwei Du, Ph.D.

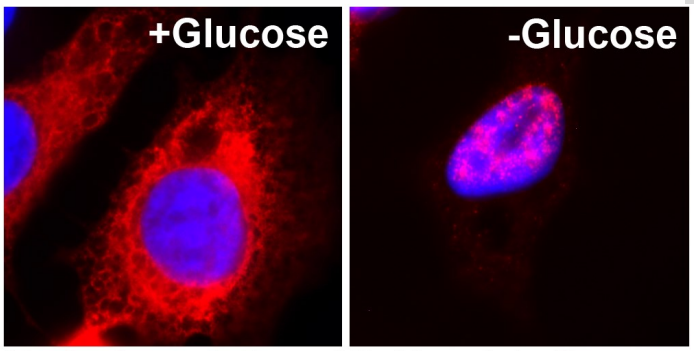
Dr. Guangwei Du's laboratory has recently received two grants for their research on vascular remodeling and cancer metabolism.

The first grant is a Research Project Grant (R01) from the National Heart, Lung, and Blood Institute to study ***the role of phospholipase D2 (PLD2)-regulated vascular smooth muscle cell migration in injury-induced vascular remodeling***. Injury-induced vascular remodeling is directly involved in several vascular diseases, such as atherosclerosis and post-angioplasty restenosis. The successful funding of this study was based on the strong preliminary data generated by Drs. Ziqing Wang and Feng Zhang, two postdoctoral fellows in the Du laboratory, who found that inhibition of PLD2 impeded vascular smooth muscle cell migration *in vitro* as well as neointima formation in injured arteries using a carotid artery ligation mouse model. In the next four years, Ziqing, and Drs. Xiao Liang and Ming Cai, two visiting scientists from China, will study the mechanisms through which PLD2 regulates vascular smooth cell migration and pathological vascular remodeling, and evaluate whether inhibition of PLD2 using small molecule inhibitors can block these processes, using a combination of techniques in biochemistry, molecular cell biology, and mouse models. These studies will identify novel molecular and cellular mechanisms that underlie vascular smooth muscle cell migration, and thus, may lead to the development of new therapeutic treatments for vascular diseases. Dr. Du and his team believe that pharmacological intervention of the PLD2 pathway may represent an efficient non-toxic therapy for vascular diseases, because removal of PLD2 in mice did not cause any adverse effect.



Inhibition of neointimal formation in *Pld2*$^{-/-}$ mice.

The second grant focuses on ***altered lipid metabolism in cancer cells***, and is funded by the Cancer Prevention Research Institute of Texas (CPRIT) CPRIT High-Impact/High-Risk Research Awards, which provide funding to explore the feasibility of high-risk projects that, if successful, will contribute major new insights into the etiology, diagnosis, treatment, or prevention of cancers. Because altered metabolism is one of the hallmarks of cancer, targeting the pathways controlling aberrant metabolism in cancer cells has been proposed as a strategy to improve current cancer therapies. Unfortunately, despite initial responses, many cancers become resistant to the current treatments and eventually relapse. Therefore, there is an urgent need to understand what makes cancer cell metabolism unique so we can design new therapies. Interestingly, two opposite activities of lipid metabolism, *de novo* lipid synthesis (anabolic) and fatty acid oxidation (catabolic), are both upregulated in cancer cells. An important question is how cancer cells sense their needs and respond by activating either anabolic or catabolic lipid pathways. To address this question, Maryia Lu, a graduate student, and Dr. Bangxing Hong, an instructor, will work together to investigate how the same protein, Lipin 1, controls either lipid synthesis or fatty acid oxidation, depending on the metabolic needs in cancer cells. Furthermore, they will test a new concept: the production of a lipid precursor by Lipin 1 not only controls the level of lipids, but also the synthesis of other macromolecules, such as proteins and nucleic acids. Knowledge gained from this study will help us to better understand the dependence of cancers on their altered metabolic requirements and to design new therapeutic strategies.



Nurient regulation of lipid metabolism.

Continued on page 7

The Du laboratory is focused on how lipid signaling and metabolism regulate some essential cellular functions, and how this knowledge can be used to design therapeutic strategies for human diseases. “We are very glad that our past accomplishments and future potential were recognized by NIH and CPRIT,” Dr. Du said. “With the support of these two grants, we look forward to some important discoveries in the following years.”

Ming Cai, M.D, Xiao Liang, M.D., Ziqing Wang, Ph.D., Maryia Lu, Bangxing Hong, Ph.D, Guangwei Du, Ph.D.

Jeff Frost, Ph.D.

Role of the RhoGEF Net 1 in Breast Cancer Cell Motility and Metastasis

A key problem in breast cancer research is to understand how and why breast cancer cells metastasize. Our research indicates that the Net1 protein may be an important regulator of this process. Net1 is a guanine nucleotide exchange factor for the RhoA subfamily of small G proteins. This means that it stimulates GTP binding by RhoA, which initiates cell signaling pathways that control actin cytoskeletal organization and cell motility. Net1 is unusual among RhoGEFs in that it cycles between the nucleus and plasma membrane. As RhoA must be activated at the plasma membrane to stimulate cell motility, this means that control of the subcellular localization of Net1 is a key mechanism that regulates its function. One of the goals of this R01 is to understand how Net1 localization in the cell is controlled.

Another goal of this R01 is to understand what Net1 may be doing in the nucleus. Our preliminary evidence indicates that Net1 is not targeted to the nucleus merely to hide it from RhoA. Instead, we believe that Net1 may act in the nucleus to control the expression of genes that are required for metastatic invasion. Cancer cells move through an extracellular matrix (ECM) by alternately squeezing between matrix fibers and using metalloproteinases to clear a path. We have published that the Net1A isoform is needed for cells to squeeze between fibers, and that without enough Net1A breast cancer cells will revert to the slower, protease-dependent form of movement. Importantly, it appears that Net1A may also work in the nucleus to actively suppress the expression of particular metalloproteinases that are critical to ECM invasion, and a key part of the proposed work is to understand how Net1A controls metalloproteinase transcription.

Lastly, in this work we will leverage our newly produced *Net1* knockout mouse model to understand how Net1 contributes to normal mammary gland development, as well as breast cancer tumorigenesis and metastasis *in vivo*. To understand its role in cancer, we will breed *Net1* knockout mice to different oncogene models, such as HER2, and monitor whether tumorigenesis or metastasis is affected. We will also apply what we have learned in our cell culture studies about Net1 to define which of its functions are most critical for breast cancer progression *in vivo*.

Publications

Tselnicker IF, Tsemakhovich V, Rishal I, Kahanovitch U, **Dessauer CW**, Dascal N. Dual regulation of G proteins and the G-protein-activated K^+ channels by lithium. *Proc Natl Acad Sci*. 2014 Mar 17. [Epub ahead of print]

Tao X, Mei F, Agrawal A, Peters CJ, Ksiazek TG, **Cheng X**, Tseng CT. Blocking of exchange proteins directly activated by cAMP leads to reduced replication of middle East respiratory syndrome coronavirus. *J Virol.* 2014 Apr; 88(7):3902-10.

Almahariq M, Mei FC, **Cheng X**. Cyclic AMP sensor **EPAC** proteins and energy homeostasis. *Trends Endocrinol Metab*. 2014 Feb;25(2):60-71.

Hocker HJ, Rambajal N, Gorfe AA. LIBSA–A method for the determination of ligand-binding preference to allosteric sites on receptor ensembles. *J Chem Inf Model.* 2014 Feb 24;54(2):530-8.

Prakash P, Gorfe AA, Overview of simulation studies on the enzymatic activity and conformational dynamics of the GTPase Ras. *Molecular Simulation.* 2014 Mar 19.

Mouw JK, Yui Y, Damiano L, Bainer RO, Lakins JN, Acerbi I, Ou G, Wijekoon AC, **Levental KR**, Gilbert PM, Chen Y-Y, Weaver VM. Tissue mechanics modulate microRNA-dependent PTEN expression to regulate malignant progression. *Nat Med.* 2014 Mar 16.

Kanakkanthara A, Eras J, Northcote PT, **Cabral F**, Miller, JH. Resistance to peloruside A and laulimalide: functional significance of acquired βI-tubulin mutations at sites important for drug-tubulin binding. *Curr Cancer Drug Targets.* 2014 Jan; 14(1):79-90.

Wu P, Wilmarth MA, Zhang F, **Du G**. miRNA and shRNA Expression Vectors Based on mRNA and miRNA Processing. *Methods. Mol. Biol.* 2013; 936:195-207.

Bohdanowicz M, Schlam D, Hermansson M, Rizzuti D, Fairn GD, Ueyama T, Somerharju P, **Du G**, Grinstein S. Phosphatidic acid is required for the constitutive ruffling and macropinocytosis of phagocytes. *Mol Biol Cell*. 2013 Jun; 24(11):1700-1712.

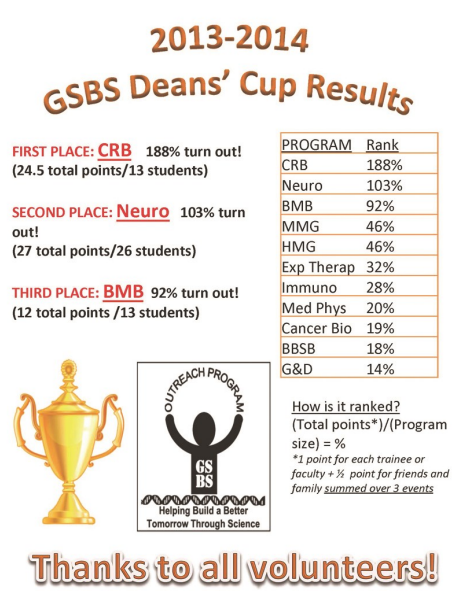
Schlam D, Bohdanowicz M, Chatgilialoglu A, Steinberg BE, Ueyama T, **Du G**, Grinstein S, Fairn GD. Diacylglycerol kinases terminate diacylglycerol signaling during the respiratory burst leading to heterogeneous phagosomal NADPH oxidase activation. *J Biol Chem*. 2013 Aug 9;288(32):23090-104.

Zhang F, Wang Z, Lu M, Yonekubo Y, Liang X, Zhang Y, Wu P, **Zhou Y**, Grinstein S, ***Hancock JF***, **Du G**. Temporal production of the signaling lipid phosphatidic acid by phospholipase D2 determines the output of ERK signaling in cancer cells. Mol Cell Biol. 2014 Jan; 34(1):84-95.

Feng X, Huang Y, Lu Y, Xiong J, Wong CO, Yang P, Xia J, Chen D, **Du G**, **Venkatachalam K**, Xia X, **Zhu MX**. Drosophila TRPML forms PI(3,5)P2-activated cation channels in both endolysosomes and plasma membrane. J Biol Chem. 2014 Feb 14;289(7):4262-72.

Annual Dean's Cup

Trish McFarland



Congratulations to the CRB Program on winning the 1st Annual Dean's Cup Award. Jeff Frost, Co-Director of CRB, accepted the trophy on behalf of the CRB at the GSBS Friday Afternoon Club gathering on Friday, March 7.

This award recognizes the CRB Program (students and faculty) along with their family and co-workers for their participation in local outreach/volunteer events. CRB won with 188% participation followed by Human Molecular Genetics with 103% and Neuroscience with 92%.

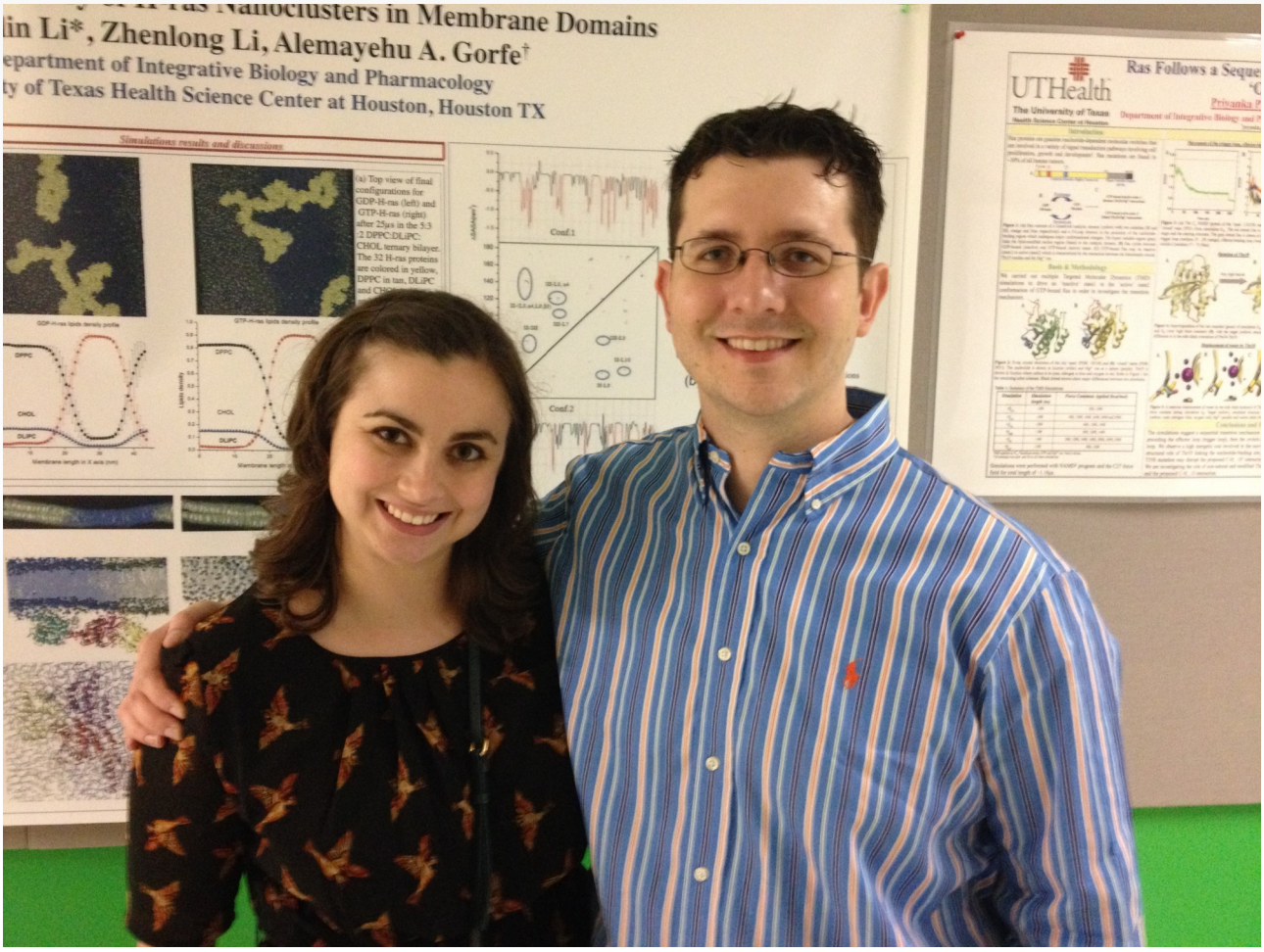
The GSBS Outreach Committee organized three events in which the programs could participate in this past year. The next event is scheduled for this summer. The trophy will be given to the Program who has the most volunteers (students, faculty, co-workers, family and friends) who participate at each event next year.

A big thanks for everyone who volunteered this past year.

Harrison Hocker Defends Ph.D. Dissertation

Harrison Hocker, Ph.D.

In February of 2014, Harrison Hocker successfully defended his Ph.D. dissertation entitled "Structured based prediction of drug -protein interactions at allosteric sites: applications to Ras". During his 5 years in the department Harrison published 2 first author journal articles in the Proceedings of the National Academy of Sciences USA and the Journal of Chemical Information and Modeling. In addition, he was an author on 4 other publications during his time in IBP. The majority of his efforts were directed towards developing novel computational methods to identify small molecule inhibitors of the Ras oncogene. The highlight of his work was the discovery that a natural medicinal product called Andrographolide inhibits the activation of this aberrant protein. The compound was used to demonstrate the requirement of key regulatory proteins, namely guanine exchange factors, for the maintenance of oncogenic Ras signaling. Soon after, Harrison published a complimentary paper, which utilized concepts derived from Fourier analysis and signal processing and developed a novel method for identifying drug cores by effectively analyzing patterns in the data. Harrison's graduation coincided with the graduation of his wife, Gabriela, from nearby UTMB; they are now moving to New Orleans where she will begin her residency in pediatric neurology at the LSU Health Science Center and Harrison is thinking about his next moves in the Industrial world. Harrison wanted to say thank you to everyone who made his time in the department a memorable and happy one!



Proposals & Awards

Anne Dybala & Deborah Brougher

Twenty proposals were submitted by the Department of Integrative Biology & Pharmacology in the first quarter of Fiscal Year 2014 by Drs. Berdeaux, Chang, Cheng, Denicourt, Grill, Hancock, Hu, Levental, Lichtenberger, O'Neil, Pochynyuk, Venkatachalam and Zhu.

One proposal was awarded this quarter along with one federal continuation. Faculty receiving awards include Drs. Loose and Venkatachalam. The success rate for awards is approximately 22%.

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

Proposals Submitted FY2014 2nd QTR

# Submitted	Federal	Federal Continuation	Private	State	Total
14	17,961,236.00				**17,961,236.00**
1		133,073.00			**133,073.00**
4			583,840.00		**583,840.00**
1				884,051.00	**884,051.00**
20	**17,961,236.00**	**133,073.00**	**583,840.00**	**884,051.00**	**19,562,200.00**

Awards Received FY2014 2nd QTR

# Rec'd	Federal	Federal Continuation	Private	State	Total
1	332,500.00				**332,500.00**
1		164,224.00			**164,224.00**
2	**332,500.00**	**164,224.00**	**-**	**-**	**496,724.00**

New Awards

Awards received during the **2nd** quarter of Fiscal Year **2014** include:

Kartick Venkatachalam, Ph.D. National Institute of Neurological Disorders and Stroke. *Alterations in synaptic growth and lipid-raft organization in a fly MLIV model.*

01 Feb 2014 – 31 Jan 2019

IBP Seminar Series

~Directed by Drs. Shane Cunha and Kartik Venkatachalam

April 7, 2014

Boyi Gan, Ph.D.
Department of Experimental Radiation Oncology
MD Anderson

Host: Guangwei Du, Ph.D.

April 14, 2014

Kevin Phillips, Ph.D.
Diabetes & Metabolic Disease Program
The Methodist Hospital Research Institute

Host: Rebecca Berdeaux, Ph.D.

April 21, 2014

Andrew Gladden, Ph.D.
Department of Genetics
MD Anderson

Host: Catherine Denicourt, Ph.D.

April 28, 2014

Matthias Buck, Ph.D.
Department of Pharmacology
Case Western Reserve University

Host: Alemayehu Gorfe, Ph.D.

May 5, 2014

Hoang Nguyen, Ph.D.
Department of Molecular and Cellular Biology
Baylor College of Medicine

Host: Shane Cunha

May 12, 2014

Shawn Burgess, Ph.D.
Developmental Genomics Section
National Human Genome Research Institute

Host: Ghislain Breton

May 19, 2014

Timo Rieg, M.D.
Department of Medicine
University of California San Diego

Host: Oleh Pochynyuk

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 or 713-500-7536.*

IBP Calendar of Events

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

April 8; May 13

CRB Meetings, 12-1 PM, Room 4.100

February 6; March 6; April 3

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

January 15, 22, 29; February 5, 12, 19, 26; March 5, 12, 19, 26; April 2, 9, 16, 23, 30

Journal Club, 3-5PM, MSB 4.100

January 23; February 6, 20; March 6, 20; April 3, 17

STG Seminar, 4-5 PM, MSB 4.100

January 15, 22, 29; February 5, 12, 19, 26; March 5, 12, 19, 26;

April 2, 9, 16, 23, 30

Dates to Remember:

April 17-18: CRB Retreat, Camp Allen, Navasota, TX

April 22: Earth Day

May 5: Cinco De Mayo

May 11: Mother's Day

May 17: Armed Forces Day

May 26: Memorial Day-*The University will be closed for Official Business*

June 1: Hurricane Season begins

June 14: Flag Day

June 15: Father's Day

July 4: Independence Day-*The University will be closed for Official Business*

The next newsletter will be published in early August 2014.

FACULTY—*Please send information on recent research awards, publications, honors, awards and your student's successes.*

STUDENTS—*Please send information regarding your achievements and the activities in which you are involved. This can include honors, awards, publications, master's defense, doctoral defense.*

STAFF—*Please send information regarding your achievements as well! Have you attended a training or a conference that you found helpful? Have you received an honor or an award?*

EDUCATION IN THE DEPARTMENT—*Faculty, please feel free to send a story about a class you teach and/or organize.*