DEPARTMENT OF

Molecular & Integrative Physiology

SCHOOL OF MOLECULAR & CELLULAR BIOLOGY

NOVEMBER 2017 NEWSLETTER

IN THIS ISSUE

ABOUT THE NEWSLETTER

The Molecular and Integrative Physiology Newsletter is an annual publication of the Department of Molecular and Integrative Physiology in the School of Molecular and Cellular Biology at the University of Illinois, Urbana-Champaign. The newsletter is written by MIP faculty and friends, and designed by MCB Communications.

Our alumni are important to us. We want to hear from you. Send us your latest news, and we'll include it in the next newsletter's MIP Family News. We also welcome articles and suggestions for future newsletters. Here's how to reach us:

Molecular & Integrative Physiology
University of Illinois at Urbana-Champaign
524 Burrill Hall
407 S. Goodwin
Urbana, IL 61801
email: mip-news@illinois.edu

GREETINGS FROM THE HEAD

Claudio Grosman

As Prof. Milan Bagchi transitioned into his new leadership role as the Director of the School of Molecular and Cellular Biology (MCB) this past August, I proudly received the torch to become the new Head of our Department. As I learn the intricacies of this new role, I reminisce to the summer of 2002 when I joined the Department of Molecular and Integrative Physiology (MIP) as an Assistant Professor. It has been a challenging, exciting and rewarding 15 years working in the area of ion-channel structure–function relationships utilizing molecular-biology, protein-engineering, electrophysiology, and structural-biology tools. I became a full Professor in 2013, and now, as a Head, I aspire to lead our Department into the future, venturing into new exciting areas while bolstering and retaining our well-recognized strengths.

This issue of the MIP Newsletter features an article by Assistant Professor Sayee Anakk and Professor Jongsook Kim Kemper on bile-acid signaling, a relatively unexplored area of intracellular signal transduction involving molecules whose role was once thought to be strictly confined to the solubilization of fat in the lumen of the intestine. We are also thrilled to highlight the promotion of three young faculty members (Hee Jung Chung, Eric Bolton, and Dan Llano) to Associate Professor with indefinite tenure, a most celebrated milestone. I am very proud of the younger generation of scientists our Department is fostering and am confident that their vision will guide us into a bright future.

This year's Newsletter also captures a glimpse of our Annual Departmental Retreat held in the beautiful Allerton Park in April 2017, which featured a talk by Emeritus Prof. Victor Ramirez and his former (1997–2002) PhD student Sean Smith, now Executive Director of Neuroscience at Merck. In addition, you can learn more about Prof. Bagchi's decisive new role, and find out about MIP's scientific contributions both in terms of papers and grants. More importantly, we highlight the success of our Departmental trainees — students and postdocs who are the tour de force of our scientific achievements.

However exciting these times are to conduct research, the funding climate has become very tough, thus posing a formidable barrier to scientific progress. Indeed, with NIH paylines slowly approaching the single digits in some Institutes, the scientific endeavor has become increasingly challenging. We hope that our alumni and friends will remain actively committed to our Department so as to buttress our continued growth and sustain our rank as one of the most prestigious places to do research and receive education in modern Physiology.

DIVERSE FUNCTIONS OF THE BILE ACID SIGNALING AND ITS SENSOR SHP

Updates from the Anakk and Kemper laboratories

Bile acids (BAs) are amphipathic steroid molecules that aid in digestion of dietary lipids, but also function as signaling molecules that profoundly impact metabolism and energy balance. Due to the detergent-like toxic properties of excess BAs, BA levels must be tightly controlled through feedback regulation of BA synthesis, transport, and metabolism. Deficiencies in these homeostatic responses result in abnormal accumulation of BAs in the liver, resulting in cholestatic liver injury, which can further progress to fibrosis, cirrhosis and cancer.

It is known that liver cirrhosis results in a heart disease called Cirrhotic Cardiomyopathy that is characterized by reduced heart function, rhythmic disturbances and electrophysiological abnormalities. Clinical statistics reveal that approximately 50% of adults and 70% of children diagnosed with cirrhosis develop this cardiac condition. However there are no feasible treatments for this disorder. Recent findings from the Anakk laboratory revealed the "Heart" of BA signaling in pathophysiology of this condition (Fig 1). While characterizing the Farnesoid X Receptor (FXR); Small heterodimer Partner (SHP) double knockout (DKO) mouse model for cholestasis (excess accumulation of bile acids), the Anakk Lab observed that DKO mice had enlarged heart size. Echocardiographic studies revealed 30% reduction in cardiac output in DKO mice. Additionally, DKO hearts displayed attenuated response to ß-adrenergic challenge. Consistent with these defects, BA overload coincided with a metabolic switch from fatty acid utilization to glucose oxidation. Utilizing ex vivo working heart model, it was found that WT hearts reversed the substrate preference from fat to glucose when exposed to pathological concentrations of BAs. Mechanistically, this metabolic switch was mapped to the down-regulation of *Pgc1α*, a key regulator of cardiac fatty acid oxidation. Further, increasing *Pgc1α* levels in cardiac cells was able to revert the metabolic switch mediated by BA. Interestingly, her lab demonstrated that reducing serum BA pools was sufficient to overcome the cardiac defect in the DKO hearts (Mathur, Desai, et al., Hepatology, 2017). The BA binding resin, cholestyramine used in the study is a FDA approved drug for treating hypercholesterolemia in the clinics. This is exciting and opens a venue for potential therapeutic option for patients with Cirrhotic Cardiomyopathy. Future clinical trials should validate these findings from mice to men!

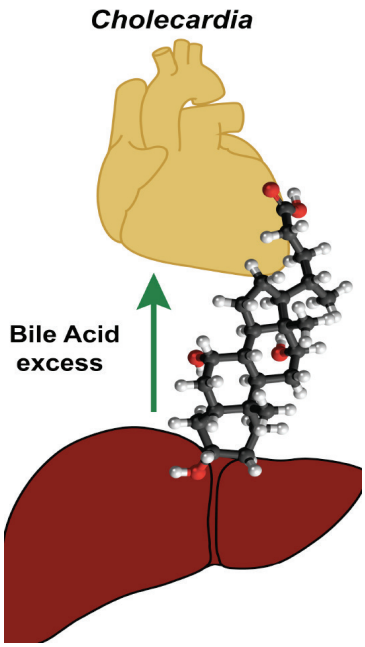


Fig 1. Bile acids expand their reach to the heart.

In another recent study, the Anakk lab identified a novel role for BA sensor- SHP in regulating fat accumulation in the liver (Akinrotimi, Riessen, et al., Hepatology, 2017). It has been known for a decade that BA signaling can protect against diet-induced obesity. To identify the downstream signals responsible for the beneficial metabolic phenotype, the Anakk lab examined the role for FXR and SHP that are both known to coordinate BA homeostasis. When they challenged DKO, liver- specific FXR knockout and liver-specific SHP Knockout mice with 45% high fat containing diet they found that double deletion of FXR and SHP was beneficial and it protected against diet-induced obesity. They attribute this metabolic protection to reduced fat accumulation in the liver and increased fat burning in the skeletal muscle of the DKO mice.

On the contrary, liver-specific deletion of FXR did not protect against diabetes or obesity whereas liver-specific SHP knockout (L-ShpKO) mice showed lower weight gain and modest improvement in glucose tolerance. Intriguingly, the liver-specific SHP knockout mice were resistant to developing fatty liver subsequent to the high fat diet regimen (Fig. 2). The Anakk lab mapped it to a reduction in key transcription factors, SREBP1c and PPARγ2 responsible for fat synthesis. Additionally they found that when SHP is specifically deleted in the liver, it results in significant down regulation of Fsp27ß (Fat specific Protein 27 ß) expression. It is known that Fsp27ß is required to maintain lipid droplet size and fat accumulation in the liver. These findings uncover that SHP is necessary to regulate fat synthesis and accumulation in the liver. In the United States, fatty liver disease is on the rise with a striking 25% incidence rate; therefore targeting SHP signaling in the liver may be offer new modality to treat this rising epidemic.

The Kemper lab has conducted a series of studies to elucidate a variety of molecular mechanisms regulated by SHP since 2004. They demonstrated that SHP represses expression of its target genes that are involved in hepatic BA production, including the cholesterol 7α hydroxylase (CYP7A1), in response to BA and FGF19 signaling, by recruiting gene-repressive histone modifying proteins, including HDAC histone deacetylases and LSD1 histone demethylase.

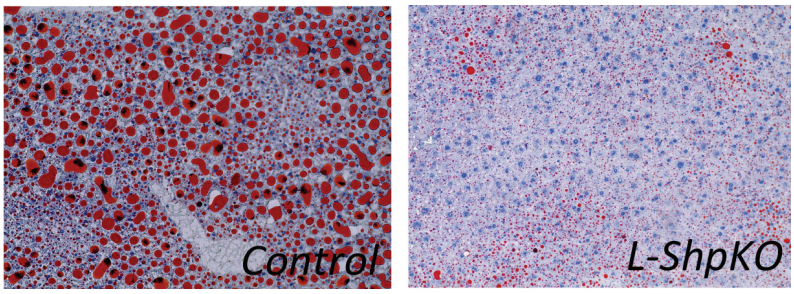


Fig 2. Oil Red O Staining of neutral lipids shows robust decrease in the hepatic fat depots in *L-ShpKO* than the control mice.

Endogenous ligands for SHP have not been identified, but the transcriptional function of SHP can be profoundly modulated by signal-induced post-translational modifications. In recent studies, the Kemper group discovered the functional importance of BA- and FGF19 signal-induced phosphorylation and SUMOylation of SHP in protein stability and nuclear localization of SHP, which greatly enhances its ability to repress BA synthetic genes (Miao et al., Genes Dev., 2009; Seok et al., J Biol Chem., 2013). Intriguingly, her lab recently showed that RanBP2, which is the largest filament of the nucleopore complex and an E3 SUMO ligase, has an unexpected function to mediate SUMOylation of SHP (Kim et al., Nat Commun., 2016). This SUMOylation is critical for nuclear transport and the gene repression function of SHP to maintain BA homeostasis (Fig 3).

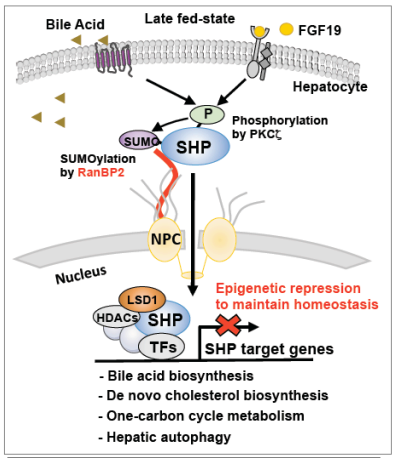


Fig. 3 Working model of SHP signaling on BA homeostasis and cholesterol biosynthesis.

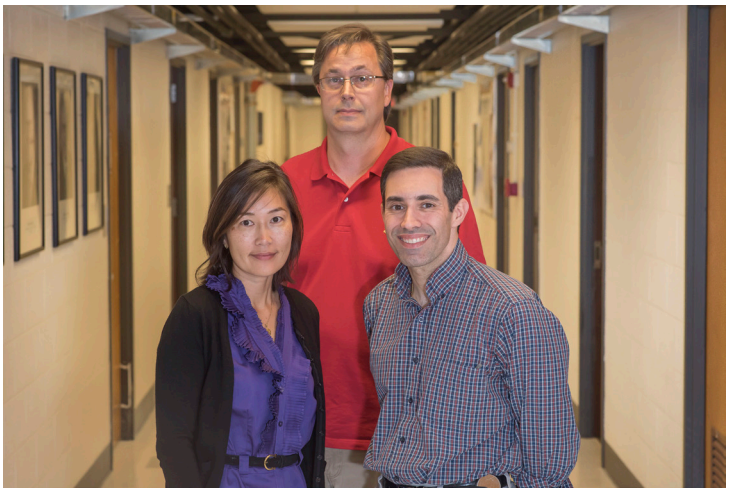
In recent global ChIP-seq studies, Dr. Kemper's lab identified genome-wide binding sites for SHP in mouse liver (Kim et al., Genome Biol., 2015), which confirmed the known repression function in BA synthesis, but also revealed previously unknown functions of SHP (Fig 3). These analyses led to the discovery that SHP inhibits de novo cholesterol biosynthesis, which complements the previously known inhibition by SHP of cholesterol conversion to BAs and prevents excessive accumulation of cholesterol in the liver. In addition, based on these genomic data, the Kemper lab further identified a novel function of SHP in the repression of hepatic autophagy in the nutrient-rich fed-state (Byun et al., EMBO J., 2017). Together with a previous finding reported by her group that the feeding-sensing nuclear receptor FXR transcriptionally inhibits hepatic autophagy gene networks (Seok et al., Nature, 2014), both FXR and SHP inhibit autophagy in a mutually dependent manner, but FXR acts early, while SHP acts relatively late after feeding, which effectively sustains postprandial inhibition of autophagy. Finally, the Kemper lab also identified SHP and AhR, also known as dioxin receptor, as postprandial physiological regulators of phosphatidylcholine and S-adenosylmethionine levels in the one carbon-cycle (Kim et al., under revision in Nat Commun.). Thus, SHP plays a critical role in maintaining metabolic homeostasis. In each case studied, levels of BA, cholesterol, autophagy, and one carbon-cycle metabolites must be tightly regulated under physiological conditions and abnormal levels are associated with metabolic disorders, neurodegenerative diseases, and cancer. Currently, her group also focuses on exploring new biological functions of intestinal SHP using global ChIP-seq and RNA-seq, intestinal organoids, and genetic mouse models.

Taken together, results from the Anakk and Kemper Laboratories emphasize on the relevance of understanding metabolic signaling. Despite the complexities and overlap with different pathways, it is appealing to postulate that modulating metabolites and their sensors locally may benefit a wide range of disorders from metabolic syndrome to cancers!

HEE JUNG CHUNG'S RESEARCH PROGRAM

Dr. Chung received her B.A. degree in Biochemistry and Chemistry from Cornell University. She then received her Ph.D. in Neuroscience under Dr. Richard L. Huganir from Johns Hopkins University and her postdoctoral training with Dr. Lily Y. Jan at the University of California San Francisco. Her graduate and postdoctoral studies discovered activity-dependent phosphorylation of glutamate receptors and potassium channels as a crucial mechanism for regulating synaptic plasticity.

After Dr. Chung established her own laboratory at UIUC in 2010, her research focused on understanding the pathogenic mechanisms underlying epilepsy, a common hyperexcitability brain disorder clinically characterized by chronic occurrence of seizures. Since ion channels are critical regulators of neuronal excitability, the major goals of her research have been to (1) understand the pathogenic mechanisms of epilepsy mutations of KCNQ/Kv7 potassium channels that potently inhibit repetitive and burst firing of action potentials, and (2) identify molecular mechanisms that persistently alter ion channels to cause hyperactivity during the development of acquired epilepsy.



Newly Tenured Associate Professors: Hee Jung Chung, Eric Bolton, Daniel Llano

Dr. Chung's research group discovered that epilepsy mutations disrupts function, expression, and localization KCNQ/Kv7 potassium channels, leading to neuronal hyperexcitability and degeneration. Her lab is currently charactering KCNQ/Kv7 epilepsy mutation knock-in mice to study epileptogenesis in vivo and use it for small molecule drug screening. Her research group also uncovered a number of genes whose protein products (including KCNQ/Kv7 channels) mediate persistent increase in neuronal excitability. Given that epilepsy is a comorbid condition for Alzheimer's disease, her lab is currently generating new mouse models to study neural plasticity in these diseases.

ERIC BOLTON'S RESEARCH PROGRAM

Eric Bolton received a B.S. magna cum laude in Biochemistry and Molecular Biology at the University of Wisconsin-Eau Claire and a Ph.D. in Molecular Biology and Genetics at the Johns Hopkins University-School of Medicine. He completed postdoctoral research with Dr. Keith Yamamoto at the University of California-San Francisco before joining the MIP faculty in 2010.

The major goals of his research have been to understand how the prostate gland develops and how disruption of hormone receptor signaling pathways early in prostate development leads to neoplasia. Prostate neoplasia is a leading cause of urologic disease among men. Indeed, most lower urinary tract symptoms in men over age 50 are due to benign prostatic hyperplasia, and prostate cancer is the second leading cause of cancer-related deaths in men. Genetic studies suggest that the androgen receptor (AR) plays a role in prostate neoplasia initiation. Androgens, like testosterone, and AR signaling are necessary for prostate development and homeostasis, and disruption of AR-mediated prostate development leads to prostate neoplasia by altering the gland's phenotype early in life. Due to the involvement of AR in prostate development and neoplasia, a thorough understanding of how AR controls cell proliferation and differentiation during prostate development will offer clues to central developmental events that reemerge in prostate neoplasia.

A fundamental question is how hormones, such as androgens and growth factors, regulate the development and growth of the prostate gland. Further, molecular crosstalk between such signaling pathways may direct prostate development and neoplasia. Bolton's laboratory utilizes an innovative organ culture system and complementary mouse models for prostate development and modern genomic and signaling pathway analyses to identify novel crosstalk between AR and growth factor signaling pathways that control cell proliferation and differentiation to orchestrate prostate development.

DAN LLANO'S RESEARCH PROGRAM

Daniel Llano obtained his BS, PhD and MD from the University of Illinois. He first became interested in scientific research while working in an MIP lab (that of Dr. Tony Waldrop) as an undergraduate Howard Hughes fellow. He obtained his PhD in MIP under the guidance of Dr. Albert Feng. After graduating, he completed neurology residency training at the Massachusetts General Hospital and Brigham and Women's Hospital and postdoctoral training at the University of Chicago. He joined the MIP faculty in 2010 and runs a laboratory at the Beckman Institute while seeing patients with cognitive disorders at Carle one day per week.

Dr. Llano's laboratory is focused on the organization of the mammalian auditory system and how it changes under certain pathological states, such as during tinnitus and during the aging process. He uses a range of electrophysiological, optical and anatomical tools to complete these studies. One major line of research in the laboratory involves how "top-down" modulation works. That is, how do our expectations and prior history of sound exposure affect how we process incoming sounds? Understanding these mechanisms will yield great insights into sensory perception and may shape how we develop neuroprosthetic devices in the future. Another line of research involves investigation of how aging affects the mammalian auditory system, and how these changes can be ameliorated by physical exercise. This latter project is being done in collaboration with Dr. Justin Rhodes at the Beckman Institute. To complete this project, the laboratory is developing novel methods to image the redox state of multiple brain structures simultaneously and will use these methods to measure the impact of physical exercise on aging-related changes in the brain.

THE 2017 MIP ANNUAL RETREAT

The 2017 MIP Annual Retreat returned for a second year in a row to the Allerton Park and Retreat Center in Monticello. Once again, we were treated to high quality oral and poster presentations highlighting the diverse and excellent work being conducted in MIP labs.

We were delighted to welcome back MIP alumnus Dr. Sean Smith and MIP professor emeritus Victor Ramirez to deliver two keynote lectures. This occasion was special as Dr. Smith obtained his PhD in Dr. Ramirez's lab. Dr. Smith, the Executive Director of Neuroscience at Merck & Co., presented "Increasing Protein O-GlcNAcylation as a Disease Modification Strategy for Tauopathies." He highlighted the importance of collaborations between academia and industry, and presented some of the work that Merck has done in developing new therapies for neurodegenerative diseases including Alzheimer's, Parkinson's, schizophrenia, and lesser known disorders, such as Progressive Supranuclear Palsy.

Dr. Ramirez delivered a very entertaining presentation, "My Experience at MIP." He stressed the importance of mentoring in his career, both as a mentee of two giants of neuroendocrinology, Dr. Samuel McCann and Dr. Charles Sawyer, and as a mentor to many graduate and undergraduate students in MIP. He expressed deep gratitude to the MIP department for welcoming him from Chile, and encouraged the current students to take full advantage of school and campus resources in their time here.

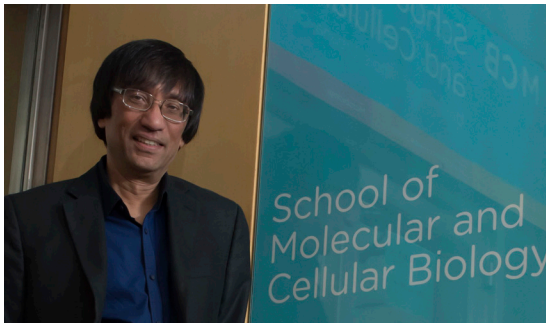
We were also able to enjoy a beautiful spring day and get some fresh air by competing in an egg race, scientific charades, bean bags, and the hula hoop pass. Connor Courtney (Christian Lab) and Hanna Erickson (Anakk Lab) received awards for the best oral presentations, and the poster awards went to Whitney Edwards (Raetzman Lab) and Bingtao Tang (Roy Lab). Hanna also won a contest to decide the next MIP t-shirt design, with a creative image that highlights several facets of MIP research in endocrinology, neurobiology, development, and disease.


Hanna Erickson (Anakk lab)


Bingtao Tang (Roy lab) and Jessica Saw (Wildman lab)


MIP Retreat Committee: Alison Hantak, Sisi He, Bhoomika Mathur, Dr. Anakk, Dr. E. Nelson, Dr. Bagchi, Matthew Biehl, Whitney Edwards, Hanna Erickson, Jessica Saw

MESSAGE FROM DR. MILAN BAGCHI, DIRECTOR OF THE SCHOOL OF MCB



As I transition from the headship of the Department of Molecular and Integrative Physiology (MIP) to assume my new role as the Director of the School of Molecular and Cellular Biology (MCB), I feel confident that the future of MIP is bright and secure. During my tenure as the department head (2012-17), I was able to recruit several talented young faculty members who I believe will achieve great distinction in their careers and carry the torch of this department, which has a rich history as one of the finest physiology departments in the nation, to an exciting future. The department is fortunate to have Professor Claudio Grosman as the new head in whose leadership we have great confidence.

Our current challenge is to keep building the MIP department in its traditional core areas of strength, such as neuroscience, endocrinology, and metabolism by recruiting top-notch scientists. With the establishment of the Carle-Illinois College of Medicine and an upswing in our collaborations with the engineering faculty on the campus, we envision developing a significant translational component in key research areas, such as molecular and systems neurobiology, metabolic health, cancer immunology, and genomic medicine. This will be a challenging task given the difficult economic times for the State of Illinois and the University, but we will keep on striving in that direction. As MCB Director, I will devote a large part of my energy to make sure that MIP and MCB function in unison to accomplish our goals for the future and attain the great heights that we are aiming for.

GRADUATE STUDENT AND POSTDOC AWARDS

Mariam Bonyadi-Camacho, a graduate student of Dr. Tom Anastasio, received an Avery Brundage Scholarship

Sangwon Byun, a postdoc of Dr. Jongsook Kim Kemper, received an American Heart Association Postdoctoral Fellowship, "Control of Bile Acid/Cholesterol Levels by Src Phosphorylation of FXR."

Hanna Erickson, a graduate student of Dr. Sayee Anakk, was elected Vice President of the American Physician Scientists Association

Sisi He, a graduate student of Dr. Erik Nelson, received a Carle-Illinois C*STAR Graduate Scholarship, "The Impact of Cholesterol on Ovarian Tumor Microenvironment and Cancer Progression

Alexandria Lesicko, a graduate student of Dr. Dan Llano, was awarded the C. Ladd Prosser Scientific Achievement Award in Neuroscience

Bhoomika Mathur, a graduate student of Dr. Sayee Anakk, was awarded the Presidential Poster of Distinction in the American Association for the Study of Liver Diseases meeting

Valeria Sanabria Guillen, a graduate student of Dr. Benita Katzenellenbogen, was appointed as a trainee in the NIH-supported Chemistry and Biology Interface Training Program

Courtney Sobieski, a postdoc of Dr. Catherine Christian, received a Beckman Institute Postdoctoral Fellowship

Georgiy Yudintsev, a graduate student of Dr. Dan Llano, was awarded the Robert Bilger Award for Excellence in Auditory Research

2017 GRADUATES AND THESIS

Kirsten Eckstrum: (Raetzman lab) Defining critical windows in pituitary development and elucidating sex differences in response to bisphenol- A

Dan Ryerson: (Kemper lab) Role of Src phosphorylation of FXR in bile acid regulation

Janelle Mapes: (Bagchi lab) Investigation of the role of CUZD1-STAT5 signaling in mammary gland development and breast cancer

Matthew Biehl: (Raetzman lab) Uncovering the role of notch signaling in development of hypothalamic nuclei in vivo and using in vitro microenvironments

Samuel Irving: (M. Gillette lab) The role of light signaling on astrocytic morphological plasticity in the adult male rat suprachiasmatic nucleus

Mathew Cherian: (D. Shapiro lab) Novel tools to identify estrogen regulated genes important for breast and ovarian cancer cell proliferation

ALUMNI UPDATES

Yash Gad (Anastasio lab) was recently promoted to Technology Lead of Health Analytics Innovation at W2O Group.

Tyler Harpole (Grosman lab) is now a Data Scientist at Polaris Industries.

Kathryn Jewett (Tsai lab) is now a Senior Fellow at University of Washington.

Janelle Mapes (Bagchi lab) has started as an Assistant Professor at Pacific Northwest University of Health Sciences.

Wei Wang (Bagchi lab) was recently promoted to Senior Research Scientist in Non-Clinical Safety Assessment of Eli Lilly and Company.

Yuechao Zhao (Katzenellenbogen lab and Bagchi lab), is now a Senior Scientist at MI Bioresearch.

FACULTY GRANTS NEWLY AWARDED IN 2017

Sayee Anakk, NIH R01, "Understanding mechanism(s) that regulate liver growth and function."

Hee Jung Chung, NIH R01 Co-I (Paul Selvin, lead PI), "Super Resolution Microscopy of Neuronal Synapses with Small Quantum Dots and Advanced Imaging Tools."

Dan Llano, NIH S10, "An upright multiphoton microscope for biomedical research applications."

Benita Katzenellenbogen, Breast Cancer Research Foundation, "Genomic Profiling of the Estrogen Hormonal Pathway for Breast Cancer Prevention and Treatment."

Benita Katzenellenbogen, Breast Cancer Research Foundation Co-I (John Katzenellenbogen, lead PI), "Antagonists for Metastatic Breast Cancers Driven by Estrogen Receptors with Activating Mutations."

Jongsook Kim Kemper, NIH R01, "Coordination of Gut-Liver Bile Acid Signaling by FXR."

Jongsook Kim Kemper, NIH R01, "Regulation of Cholesterol Catabolism by Bile Acids."

Erik Nelson, NIH R01 Co-I (Andrew Smith and Kelly Swanson Co-PIs), "Targeted Drug Delivery to Adipose Tissue Macrophages in Obesity."

Erik Nelson, Beckman Research Award Co-PI (with William Helferich and Nicki Engeseth), "Thermally Abused Frying Oils and Secondary Breast Cancer Metastasis."

Lori Raetzman, Campus Research Board, "Mechanisms of altered fetal hypothalamic development in response to gestational diabetes."

Derek Wildman, NIH U01 Co-PI (with Leon Mutesa, Stefan Jansen, and Monica Uddin), "Transgenerational Epigenomics of Trauma and PTSD in Rwanda."

Derek Wildman, Mayo Clinic Grand Challenge, "Computational Methods for Insight into Hypoplastic Left Heart Syndrome (HLHS)."

Derek Wildman, NIH R01 Co-PI (with Monica Uddin and Allison Aiello), "Epigenomic Predictors of PTSD and Traumatic Stress in an African American Cohort."

SELECTED MIP PAPERS NOVEMBER 2016 - OCTOBER 2017

Akinrotimi O, Riessen R, VanDuyne P, Park JE, Lee YK, Wong LJ, Zavacki AM, Schoonjans K, Anakk S. "Shp deletion prevents hepatic steatosis and when combined with Fxr loss protects against type 2 diabetes." Hepatology. June 2017

Anastasio TJ, Barreiro AK, Bronski JC. "A geometric method for eigenvalue problems with low-rank perturbations." Royal Society Open Science. September 2017

Armstrong DL, McGowen MR, Weckle A, Pantham P, Caravas J, Agnew D, Benirschke K, Savage-Rumbaugh S, Nevo E, Kim CJ, Wagner GP, Romero R, Wildman DE. "The core transcriptome of mammalian placentas and the divergence of expression with placental shape." Placenta. September 2017

Baculis BC, Weiss AC, Pang W, Jeong HG, Lee JH, Liu DC, Tsai NP, Chung HJ. "Prolonged seizure activity causes caspase dependent cleavage and dysfunction of G-protein activated inwardly rectifying potassium channels." Scientific Reports. September 2017

Baek AE, Yu YA, He S, Wardell SE, Chang CY, Kwon S, Pillai RV, McDowell HB, Thompson JW, Dubois LG, Sullivan PM, Kemper JK, Gunn MD, McDonnell DP, Nelson ER. "The cholesterol metabolite 27 hydroxycholesterol facilitates breast cancer metastasis through its actions on immune cells." Nature Communications. October 2017

Boddy AM, Harrison PW, Montgomery SH, Caravas JA, Raghanti MA, Phillips KA, Mundy NI, Wildman DE. "Evidence of a Conserved Molecular Response to Selection for Increased Brain Size in Primates." Genome Biology and Evolution. March 2017

Byun S, Kim YC, Zhang Y, Kong B, Guo G, Sadoshima J, Ma J, Kemper B, Kemper JK. "A postprandial FGF19-SHP-LSD1 regulatory axis mediates epigenetic repression of hepatic autophagy." EMBO Journal. June 2017

Choi SE, Kwon S, Seok S, Xiao Z, Lee KW, Kang Y, Li X, Shinoda K, Kajimura S, Kemper B, Kemper JK. "Obesity-Linked Phosphorylation of SIRT1 by Casein Kinase 2 Inhibits Its Nuclear Localization and Promotes Fatty Liver." Molecular Cellular Biology. July 2017

Eckstrum KS, Edwards W, Banerjee A, Wang W, Flaws JA, Katzenellenbogen JA, Kim SH, Raetzman LT. "Effects of exposure to the endocrine disrupting chemical bisphenol A during critical windows of murine pituitary development." Endocrinology. In press October 2017

Gonzalez-Gutierrez G, Wang Y, Cymes GD, Tajkhorshid E, Grosman C. "Chasing the open-state structure of pentameric ligand-gated ion channels." Journal of General Physiology. In press October 2017

Ibrahim BA, Wang H, Lesicko AMH, Bucci B, Paul K, Llano DA. "Effect of temperature on FAD and NADH-derived signals and neurometabolic coupling in the mouse auditory and motor cortex." Pflugers Archiv: European Journal of Physiology. August 2017

Jang SS, Jeong HG, Chung HJ. "Electroconvulsive seizures in rats and fractionation of their hippocampi to examine seizure-induced changes in postsynaptic density proteins." Journal of Visualized Experiments. August 2017

Kwon S, Seok S, Yau P, Li X, Kemper B, Kemper JK. "Obesity and aging diminish SIRT1-mediated deacetylation of SIRT3, leading to hyperacetylation and decreased activity and stability of SIRT3. Journal of Biological Chemistry. August 2017

Li J, Kim JS, Abejuela VA, Lamano JB, Klein NJ, Christian CA. "Disrupted female estrous cyclicity in the intrahippocampal kainic acid mouse model of temporal lobe epilepsy." Epilepsia Open. March 2017

Liu DC, Seimetz J, Lee KY, Kalsotra A, Chung HJ, Lu H, Tsai NP. "Mdm2 mediates FMRP- and Gp1 mGluR-dependent protein translation and neural network activity." Human Molecular Genetics. October 2017

Lu W, Katzenellenbogen BS. "Estrogen Receptor-ß Modulation of the ERα-p53 Loop Regulating Gene Expression, Proliferation, and Apoptosis in Breast Cancer." Hormones and Cancer. August 2017

Mapes J, Li Q, Kannan A, Anandan L, Laws M, Lydon JP, Bagchi IC, Bagchi MK. "CUZD1 is a critical mediator of the JAK/STAT5 signaling pathway that controls mammary gland development during pregnancy." PLoS Genetics. March 2017

Park HJ, Bolton EC. "RET-mediated glial cell line-derived neurotrophic factor signaling inhibits mouse prostate development." Development. June 2017

Weckle A, McGowen MR, Xing J, Chen C, Sterner KN, Hou ZC, Romero R, Wildman DE. "Ancestral resurrection of anthropoid estrogen receptor β demonstrates functional consequences of positive selection." Molecular Phylogenetics and Evolution. September 2017

Zhao Y, Laws MJ, Guillen VS, Ziegler Y, Min J, Sharma A, Kim SH, Chu D, Park BH, Oesterreich S, Mao C, Shapiro DJ, Nettles KW, Katzenellenbogen JA, Katzenellenbogen BS. "Structurally novel antiestrogens elicit differential responses from constitutively active mutant estrogen receptors in breast cancer cells and tumors." Cancer Research. September 2017

Zhu J, Lee KY, Jewett KA, Man HY, Chung HJ, Tsai NP. "Epilepsy-associated gene Nedd4-2 mediates neuronal activity and seizure susceptibility through AMPA receptors." PLoS Genetics. February 2017

MOLECULAR & INTEGRATIVE PHYSIOLOGY

University of Illinois at Urbana-Champaign

524 Burrill Hall
407 South Goodwin Avenue
Urbana, Illinois 61801