School of Medicine
Department of Pharmacology and Chemical Biology
Annual Report 2014-2015
General Description
During the 2014-2015 academic year, the Department of Pharmacology & Chemical Biology continued to grow its strengths in discovery and education related to the practice of pharmacology. The discovery component of our departmental missions employs basic chemical principles in developing an understanding of cell signaling events, and then applies these insights in the creation of new therapeutic strategies.

This past year, the department made significant new advances in understanding fundamental mechanisms of cell and tissue communication, how these events impact on cell growth and function, the creation of new drugs to control these processes and, from this, the generation of new intellectual property (patents) that allows the commercialization of these discoveries. In spite of challenging times, the concerted effort of both faculty and staff in making and communicating seminal research advancements has been rewarded by a continued growth in extramural research support that now totals $16,287,155 annually.

The teaching missions of the department are a high priority, thus they both thrive and continue to evolve. Donald DeFranco, Ph.D., has been highly effective in his position as Vice Chair for Education, as has Patrick Pagano, Ph.D., Director of our Molecular Pharmacology Graduate Program. We have continued to restructure and revise the Ph.D. Program in Molecular Pharmacology to be more responsive to student needs and the rapidly evolving training requirements for minting a competitive pharmacologist in the contemporary job market. They are using multiple strategies to inculcate in our students new expertise spanning from synthetic organic chemistry to physiology. In this regard, the Molecular Pharmacology Graduate Program has refocused its areas of specialization to provide additional focus on molecular aspects of signal transduction, cell and organ systems pharmacology, cancer pharmacology and drug discovery. We are especially pleased with student reactions regarding our recent integration of combined clinical simulator- and murine-based “hands-on” training in organ physiology and pharmacology into our graduate curricula. Due to the interest of students outside of the Molecular Pharmacology Graduate Program, we are further expanding these organ physiology and pharmacology teaching missions. Important contributions and leadership have also been lent in graduate education by Guillermo Romero, Ph.D., Associate Director of Graduate Education and Daniel Altschuler, Ph.D., Co-Director of the Molecular Pharmacology course, in addition to our overall departmental faculty.

By new mechanisms that regularly evaluate both mentors and students, Drs. DeFranco, Pagano and Freeman have succeeded in reducing our average time for graduation of Ph.D. students to four, and sometimes less than four, years for exceptionally productive students. Departmental faculty are particularly proud of our current outstanding Molecular Pharmacology Ph.D. and M.D./Ph.D. students, who are contributing important new insight into fundamental cell signaling processes, drug actions and drug discovery.

Medical education has also thrived under Dr. DeFranco’s leadership. He is one of the most highly esteemed educators and researchers in the Department of Pharmacology & Chemical Biology and the School of Medicine. Dr. DeFranco, along with Stephan Tofovic, M.D., Ph.D., has dramatically improved the integration of pharmacology instruction into the organ-based, modular instructional approach currently given to Pitt medical students. Additionally, all of the Pharmacology & Chemical Biology faculty participate in the execution of multiple small-group and team-based workshops, focusing on clinically relevant case studies in pharmacology. With constant attention to designing course content, optimal teaching strategies and their execution, we strive to continuously evolve and fulfill our mission to educate medical students and physicians in the conceptual basis for drug selection and administration.

Pharmacology & Chemical Biology faculty are also dedicated to inspiring and growing the next generation of young scientists and physicians. Michael Palladino, Ph.D. continues to direct our highly acclaimed summer undergraduate research program, which attracts some of the brightest young, aspiring basic and physician-scientists from around the country. This program is in part funded by the American Society of Pharmacology and Experimental Therapeutics, as well as participating School of Medicine centers and departments. Our undergraduate research program supports the summer living and laboratory expenses of students who are interested in a broad range of research themes. These students are matched with a laboratory that fits their
general interests and by pursuing a research project become exposed to challenging new laboratory skills, classroom experiences and an opportunity to interact with our internationally renowned departmental investigators. This represents a significant investment of time and resources that pays untold future dividends.

The stellar research environment that exists in the Department of Pharmacology & Chemical Biology is reflected by the high-impact publications and abundant extramural support levels cited in this report. In order to grow and evolve this vibrant environment, new investments are continually being made in the research tools utilized by departmental investigators.

Career development and strong mentorship for junior faculty are critical elements for nurturing a healthy future for the department. A new faculty mentoring process was instigated in recent years, whereby the Chair and senior faculty regularly meet with non-tenured and associate professor-level faculty to discuss and strategize how best to pursue career progression and key research objectives.

In summary, the practice of Pharmacology & Chemical Biology is unique among basic sciences, as it embraces a broad range of expertise in our efforts to understand fundamental cell communication events, drug-target molecule reactions and, finally, the design, synthesis and therapeutic application of new drugs in patients. This latter effort requires strengths or critical collaborations in computational drug design, synthetic organic chemistry, biophysics, structural biology and organ physiology. Thus, Pharmacology & Chemical Biology maintains strong ties with a variety of basic science and clinical disciplines, while maintaining firm roots in fundamental elements of chemistry, cell and molecular biology, drug actions and drug metabolism. These precepts are exemplified by our premier educational and research missions that include the institution of new educational strategies, the creation of new centers of excellence, the recruitment of stellar new faculty, and the scientific and teaching contributions of our dedicated faculty. We thus invite you to explore in more detail our annual report and, if interested, to join us in fulfilling the departmental mission to excel in education, drug discovery and improved patient care.
Research and Other Scholarly Activities
Research Interests

Bruce A. Freeman, Ph.D
Professor and Chair
Ph.D., University of California, Riverside, 1978

The basic and clinical research activities of the Freeman Laboratory focus on the eukaryotic cell production, reactions and signal transduction properties of oxidizing and free radical inflammatory mediators (e.g., superoxide, hydrogen peroxide, nitric oxide (NO), peroxynitrite, nitrogen dioxide, oxidized/nitrated lipids). In particular, we are interested in the action of these species as both redox signaling mediators under basal conditions and as pathogenic agents in inflammatory diseases. Our observations regarding O₂ and NO-derived reactive species have lent new insight into redox-dependent cell signaling and have revealed new therapeutic strategies for treating acute inflammation, metabolic syndrome, respiratory disorders and cardiovascular diseases.

In the late 1980s, his group studied the cellular and subcellular organelle production of superoxide and hydrogen peroxide. Following the landmark description of endothelial-derived relaxing factor (EDRF) as the free radical NO, the Freeman laboratory pioneered the concept that the inflammatory and signal transduction mediator NO displays unique redox signaling actions following reaction with superoxide, oxidizing fatty acids and heme peroxidases. The “oxidative inactivation” of NO is a kinetically fast reaction, yielding “reactive nitrogen species” as products. This array of reactions of O₂-derived species with NO can serve to both impair and transduce NO signaling via non-cGMP dependent mechanisms.

There is now a rapidly expanding appreciation that NO-derived reactive species display distinct chemical reactivities and exert cell signaling actions beyond the activation of guanylate cyclase – e.g., via thiol oxidation, electrophilic addition and receptor-dependent reactions. This aspect of redox-related chemical biology is an area that the Freeman laboratory continues to investigate, with the intent of defining the linkages between reactive oxygen species and NO-dependent cell signaling mechanisms. From a translational research perspective, his group is addressing how these interactions impact cell and organ function, with particular directed towards metabolic, cardiovascular and pulmonary diseases.

Dr. Freeman’s laboratory observed that NO reacts with superoxide (O₂⁻) to yield the potent biological oxidizing and nitrating species peroxynitrite (ONOO⁻) and its conjugate acid, peroxynitrous acid (ONOOH). Groundbreaking observations were made in this area by Joe Beckman, PhD and Rafael Radi, MD, PhD. Their work showed that peroxynitrite is both a direct oxidant and, after homolytic scission of peroxynitrous acid, yields the potent oxidant hydroxyl radical (OH) and the oxidizing and nitrating species nitrogen dioxide (NO₂) (Fig. 1). Also, they identified thiols and carbon dioxide as the principal biological targets of peroxynitrite. It is now known that peroxynitrite accounts for many of the pathogenic actions previously ascribed to its precursors - superoxide (and its products) and NO. Work from many laboratories continues to affirm that peroxynitrite mediates redox cell signaling actions upon the oxidation or nitration of target molecules such as thiols, aromatic amino acids, nucleotides and unsaturated fatty acids – with downstream cell signaling events and reactions of peroxynitrite now appreciated to be a consequence of its potent and unique reactivities.

An observation from the Freeman laboratory, related to peroxynitrite biochemistry and pharmacology, has yielded new insight into biochemical and tissue responses to ischemia. Specifically, the CO₂ accumulation that occurs during impaired tissue perfusion and oxygen delivery displays potent pro-inflammatory properties. Observations made by Dr. Radi showed that carbon dioxide indirectly affects the reactivity of O₂-and NO, via its facile chemical reaction with the superoxide and NO reaction product, peroxynitrite. This reaction yields the potent oxidizing and nitrating species nitrosoperoxocarbonate (ONOOCO₂) that in turn yields secondary radical species (Fig. 2). John Lang, MD then discovered in an animal model of sepsis that there is a potent contribution of CO₂ to tissue redox signaling and inflammatory responses. For example, clinically-relevant
mechanical ventilation strategies performed on anesthetized rabbits reveals that mild hypercapnia amplifies inflammatory lung injury. Of interest, this also causes a CO2-dependent increase in iNOS gene/protein expression and NO/ONOO- production. The discovery that CO2 actively participates in oxidative inflammatory reactions has relevance to ICU-related care and organ transplantation.

Studies with wild type and MPO-/- mice undergoing an acute inflammatory response provided another important insight into the actions of MPO during NO signaling. Specifically, reactions catalyzed by MPO directly modulate vascular relaxation and inflammatory responses by regulating NO bioavailability. In addition to directly reacting with NO (a kinetically slow reaction), MPO predominantly alters vascular responsiveness by generating substrate radicals (such as tyrosyl radical and ascorbyl radical) that rapidly consume NO and abrogate its cGMP-dependent signaling capabilities. Thus, multiple reactions of MPO lead to biomolecule nitration and NO consumption.

Of important clinical relevance, Drs. Margaret Tarpey and Stephan Baldus have discovered that enzymatic reactions leading to the catalytic consumption of NO impair vascular function and are linked with increased risk for an adverse myocardial event (heart attack or death) in patients. The main perpetrators of oxidative NO consumption in the vasculature appear to be the reactive species derived from xanthine oxidoreductase (XO) and MPO, with both plasma XO and MPO levels elevated in patients with coronary artery disease. The work of others also suggests that a variety of NADPH oxidases act in a similar manner. As for MPO, XO readily binds to and enters the vessel wall, with this occurring to a much greater extent in patients with coronary artery disease (Fig. 5). Work by Dr. Baldus convincingly shows that both coronary blood flow and the risk for adverse myocardial events are strongly linked with plasma MPO levels in patients. More recently, it has been observed that XO also contributes to impaired coronary vasomotion in patients.

In coronary artery disease patients XO accumulates along the vessels wall and catalytically consumes NO. Dr. Freeman's group is presently actively investigating the pluripotent signaling actions of the NO-derived, nitrated unsaturated fatty acids formed during enzymatic and autocatalytic lipid oxygenation (Fig 6). Homero Rubbo, PhD discovered that NO potently inhibits fatty acid oxidation, via reactions that are >2000 times faster than similar events catalyzed by vitamin E. Dr. Rubbo also observed at the same time that NO-dependent reactions induce fatty acid nitration. Building on this observation, Valerie O’Donnell, PhD lent important structural and functional insight into these endogenously-present species, and how they can be formed biologically.

Nitro-fatty acids are present under physiological and pathological conditions in a broad range of species, including insects, fish (e.g., salmon), plants (e.g., olives) and humans. The consumption of diets promoting increased tissue and plasma nitro-fatty acid levels has recently been proposed to account for a significant element of the health benefits linked with Mediterranean and Japanese-like diets.

The use of high performance liquid chromatography techniques coupled with high accuracy mass spectrometry analysis has given precise structural and conformational characterization of these species. As a result, pure preparations of synthetic nitro-fatty acids are structurally identical to those found endogenously in humans at nM to sometimes mM concentrations.

These reaction mechanisms support the formation of nitrogen dioxide (NO2) which rapidly reacts with unsaturated double bonds in fatty acids, giving rise to the formation of nitro-fatty acid derivatives. These products contain a reactive center that interacts with critical protein targets and induces post-translational protein modifications. Somewhat akin to protein phosphorylation or dephosphorylation, nitro-fatty acids transiently alter the function of key target proteins to modulate the activity of critical gene expression programs, enzymatic activities and signaling network activities – thereby inducing characteristic anti-inflammatory, anti-fibrotic and cytoprotective actions.
Nitro-fatty acid metabolism and clearance - The formation of reversible adducts between target proteins and nitro-fatty acids results from the “soft” electrophilic nature of nitro-fatty acids. This chemical property is also the basis for nitro-fatty acid reaction with glutathione (GSH), which helps regulate nitro-fatty acid levels. Products of GSH and nitro-fatty acid reaction are exported from the cell to the circulation, where these adducts are filtered by the kidneys and excreted in urine, where nitro-fatty acid levels can be easily detected by mass spectrometry analysis. Nitro-fatty acids are also irreversibly inactivated by the enzyme prostaglandin reductase-1. Finally, like all fatty acids, nitro-fatty acids are metabolized by the mitochondrial energy-generating process termed beta-oxidation. These pathways all modulate the activity and half-life of both endogenous and exogenous nitro-fatty acids. Importantly, all critical protein adducts, gene expression events and metabolic products can be monitored in accessible tissue compartments, the plasma and urine of humans and animal models, thereby providing real-time insight into the metabolism and actions of nitro-fatty acids.

Nitro-fatty acids are adaptive signaling mediators

Biological systems are endowed with an arsenal of sentinel proteins that constantly sense and react to changes in the cellular environment, in order to ensure the continued function of critical life-sustaining processes over a wide range of pathophysiological scenarios. In this regard, the detection of an invading pathogen or a toxin is met by the activation of pro-inflammatory cascades aimed at neutralizing the threat. The excessive or chronic activation of these physiological pathways can, if left unchecked, result in further damage to organs and tissues and the development of disease.

Cells rely on the generation of adaptive signaling mediators that dynamically modulate the extent and duration of inflammatory and metabolic responses to external insults. Nitro-fatty acids were discovered to be a novel family of adaptive signaling mediators that pleiotropically down-regulate inflammation, activate protective cellular responses and induce endogenous production of antioxidant defenses in cells. Specifically, nitro-fatty acids reversibly react with electrophile-susceptible transcription factors and protein thiols to modulate gene expression and enzyme activities via the post-translational modification of functionally-significant proteins.

This ability of nitro-fatty acids to reversibly adduct proteins is at the root of their potent signaling actions and apparent lack of toxicity. For example, the pro-inflammatory actions of bacterial lipopolysaccharides (LPS) are inhibited by nitro-fatty acid adduction of the NFkB p65 subunit that undergoes DNA binding and mediates the stimulation of pro-inflammatory gene expression. Additionally, nitro-fatty acid reaction with critical cysteines in Keap1 results in the nuclear translocation of the transcription factor Nrf2 leading to increased generation of antioxidant and cytoprotective proteins by the cell. Accumulating evidence from multiple international research groups support that these cytoprotective activities of nitro-fatty acids can be harnessed for the development of effective therapies for the treatment of inflammatory and metabolic diseases. Notably, the concentrations of nitro-fatty acids that exert potent protective actions in murine models of disease (obesity-induced diabetes, restenosis, atherosclerosis, inflammatory bowel disease, pulmonary hypertension, acute and chronic renal injury, etc.) are in the 5-25 nM range, well within a pharmacologically attainable and safe range in humans.

Imad Al Ghouleh, Ph.D.
Research Instructor
Ph.D. (Experimental Medicine), McGill University, 2009

Daniel Altschuler, Ph.D.
Associate Professor
Ph.D. (Biology), University of Buenos Aires, Argentina, 1989
Dr. Altschuler's laboratory studies mechanisms of signal transduction by the second messenger cAMP in cell proliferation. cAMP-dependent protein kinase (PKA) and Exchange protein activated by cAMP (Epac) represent the main effectors of cAMP action. Both pathways converge at the level of the small GTPase Rap1b, via its Epac-mediated activation and PKA-mediated phosphorylation. The role of Rap1 activation (Epac) and
phosphorylation (PKA) coordinating the early rate-limiting events in cAMP-dependent cell proliferation are studied using a multidisciplinary approach including molecular and cellular biology techniques in vitro, as well as in vivo validation using transgenic/knock in technologies in endocrine tumor models.

**Palaniappa Arjunan, Ph.D.**
*Research Instructor*  
*Ph.D., Indian Institute of Science, Bangalore, India, 1985*

Dr. Arjunan determines the structure of macromolecules of biological interest, and then analyses structure-function relationships. He primarily uses X-ray crystallography to accomplish this.

Dr. Arjunan's current research includes the high resolution three-dimensional structure determination of thiamin diphosphate (ThDP)-dependent enzymes, the yeast pyruvate decarboxylase (PDC) and pyruvate dehydrogenase multienzyme complex (PDHc) from Escherichia coli. The refined structure is then used to address long-standing issues regarding the structure and function of thiamin diphosphate-dependent enzymes. The structure determination also includes the structure of PDHc E1 in complex with a covalently bound reaction intermediate analogue. Other interests are: a) the crystal structural analysis of native and mutant ThDP-dependent enzymes, either alone or in complexes with substrates, inhibitors, activators or with other related enzymes and b) development of techniques for the determination and analysis of macromolecular crystal structure.

**Jonathan Beckel, Ph.D.**
*Research Instructor*  
*Ph.D. (Molecular Pharmacology), University of Pittsburgh, 2009*

Dr. Beckel’s research focuses on the control of the urinary bladder; specifically how the bladder epithelium (also called the urothelium) participates in the sensory limb of the micturition reflex. The ultimate goal of this research is to understand how the urothelium and bladder afferent nerves communicate with each other to transmit sensory information about the bladder to the central nervous system and how changes in this communication play a role in the increased urgency and/or pain often felt by patients suffering from conditions such as overactive bladder (OAB) or Interstitial Cystitis/Painful Bladder Syndrome (IC/PBS). Recent research has focused on the role of purinergic signaling mechanisms in bladder pathology and the release of ATP from urothelial cells through 1) pannexin channels or 2) vesicular release from secretory lysosomes.

**Dr. Alessandro Bisello**  
*Associate Professor*  
*Laurea (Chemistry), University of Padova, Italy, 1992*

The general scientific theme in the laboratory is to define the role of accessory/scaffolding proteins (such as caveolin and EBP50/NHERF1) in the regulation of cellular and tissue functions. Our efforts focus on two specific areas:

1) **Role of EBP50/NHERF-1 on vascular remodeling.** The Ezrin-Radixin-Moesin Binding Phosphoprotein of 50 kDa (EBP50), also known as NHERF-1 is a PDZ domain-containing scaffolding protein. Our studies show that EBP50 is expressed at low levels in healthy vessels but is up-regulated following arterial injury. EBP50 contributes to the proliferation of vascular smooth muscle cells (VSMC). The temporal expression of EBP50 following arterial injury and its ability to regulate specific cell cycle proteins and signaling receptors suggest that this adaptor protein plays a key role in the integrated response of VSMC to injury. Current studies aim at determining the role of EBP50 on vascular remodeling.
2) Cellular regulation of the glucagon-like peptide 1 (GLP-1R) receptor and its role in regulating beta cell function, proliferation and survival. One of the most promising therapeutic targets for the treatment of type 2 diabetes is the glucagon-like peptide 1 receptor (GLP-1R). The well documented ability of GLP-1R agonists, either GLP-1 itself or exendin-4, to stimulate glucose-dependent insulin secretion and increase beta cell proliferation and survival led to the approval of exendin-4 for the treatment of type 2 diabetes. Our studies show that the GLP-1R interacts with caveolin-1 and this is necessary for the trafficking of the GLP-1R to the cell membrane and directs its localization to lipid rafts. The central hypothesis of this project is that the interaction between GLP-1R and caveolin-1 and its localization in lipid rafts is a fundamental mechanism controlling both the insulinotropic and the proliferative actions of GLP-1 and exendin-4.

Dinara Bulgari, Ph.D.
Research Assistant Professor
Ph.D., Kazan State Medical University, Russia, 1993

Dr. Bulgari (Shakiryanova) studies the mechanisms of dense-core vesicles transport to nerve terminals and role of presynaptic signaling in regulation of neuropeptide release. She uses combination of genetic, electrophysiology and imaging techniques to study the native intact synapses in Drosophila model system, which feature large synaptic boutons that are amenable to study by light microscopy. Neuropeptides are packaged in large dense-core vesicles and act as local co-transmitters. They influence development, behavior, mood, pain perception, sleep and circadian rhythms, inflammation, appetite. But despite these important functions little is known about the cell physiology underlying their release. These studies will continue to transform the understanding of neuropeptide release and signaling in the nerve terminal, which are fundamental to understanding the operation of the nervous system under physiological and pathological conditions.

Eugenia Cifuentes-Pagano, Ph.D.
Research Instructor
Ph.D., State University of New York at Stony Brook, 1994

Dr. Cifuentes-Pagano’s research interests focus on the understanding of the molecular mechanisms of action of novel NADPH oxidase isoforms and their regulation in the vasculature. The phagocyte NADPH oxidase (or respiratory burst oxidase) is a well-characterized reactive oxygen species (ROS)-generating system that catalyzes the one-electron reduction of oxygen to O2-, the precursor to a variety of other reactive oxygen species. The NADPH oxidase paradigm is a multi-subunit enzyme complex that includes two membrane-spanning subunits, p22-phox and nox2, and three cytoplasmic subunits, p40-phox, p47-phox and p67-phox. Our laboratory was the first to discover a nox2-based oxidase in the vasculature and to develop specific inhibitors targeting this robust source of ROS. Since that initial discovery, various isoforms of NADPH oxidase have been described which differ from the nox2 system in unique modifications of their nox-subunit amino acid sequence as well as the cytoplasmic components that they require. Besides their structural differences, the various isoforms present differential tissue and cellular distribution. The multi-level complexity of this family of proteins provides an opportunity to develop new tools to dissect the role of each of the isoforms in vascular function and pathology.

Donald DeFranco, Ph.D.
Professor & Vice Chair, Medical Education
Ph.D., Yale University, 1981

Glucocorticoid Action in the Developing Brain

The life threatening, emotional and economic burdens of premature birth (~12% of pregnancies) have been greatly alleviated by antenatal treatment with synthetic glucocorticoids (sGCs). Antenatal sGCs accelerate tissue development reducing respiratory distress syndrome and intraventricular hemorrhage in premature infants, but they can affect developmental processes in the brain and trigger adverse behavioral and metabolic
outcomes later in life. While the well-established beneficial neonatal outcomes of antenatal GCs support their continued use, we are utilizing mechanistic analysis of novel GC pathways in the developing brain (particularly in the hypothalamus and cerebral cortex) to identify new biomarkers and drug targets for male and/or female fetuses at high risk for adverse, neurological outcomes of GCs or resistant to their beneficial effects on the developing cerebral vasculature. Our approach includes molecular studies, genome wide assessment of sGC target genes, behavioral analyses in mice and histological analysis in knock-in mouse models as well as primary human neural stem cell cultures. Of direct clinical relevance are our proposed studies with cells derived from human fetuses that were exposed to antenatal GCs, which could identify novel real time biomarkers that predict outcome in children exposed to sGCs in utero.

Biological effects of GCs are mediated by the glucocorticoid receptor (GR), a member of the nuclear receptor super-family of transcription factors. While primarily studied for its role in the nucleus, GR is also localized in the plasma membrane where it can rapidly mobilize a variety of cytoplasmic signaling pathways. We have identified unique genomic (i.e. nuclear) and nongenomic (i.e. cytoplasmic) GR signaling pathways that are activated in response to antenatal GCs in the developing brain and cultured embryonic neural stem cells. For example, novel gender specific molecular targets of the genomic GR pathway are being identified by genome-wide RNA-Seq and ChiP-Seq approaches while work continues to investigate the biological impact of nongenomic GC effects on gap junction communication and synchronous, spontaneous Ca$^{2+}$ transients in coupled neural stem cells. Furthermore, we continue to explore the impact of crosstalk between nongenomic and genomic GC pathways, which we have found to regulate site-specific GR phosphorylation and recruitment of the receptor to distinct subsets of target genes.

**Estrogen Action in Benign and Malignant Prostatic Disease**

Prostatic inflammation is a common feature of symptomatic benign prostatic hyperplasia (BPH) and may alter epithelial cell proliferation and tissue homeostasis in BPH through cytokine induction of proinflammatory signaling mediators such as cyclooxygenase-2 (Cox-2). Current therapies for symptomatic benign prostatic hyperplasia (BPH), an androgen receptor (AR) driven, inflammatory disorder affecting elderly men, include 5α-reductase (5AR) inhibitors (i.e. dutasteride and finasteride) to block the conversion of testosterone to the more potent AR ligand dihydrotestosterone (DHT). Since DHT is the precursor for estrogen receptor β (ERβ) ligands, 5AR inhibitors could potentially limit ERβ activation, which maintains prostate tissue homeostasis. We have uncovered signaling pathways in BPH-derived prostate epithelial cells (BPH-1) that are influenced by 5AR inhibition. The induction of apoptosis and repression of the cell-adhesion protein E-cadherin by the 5AR inhibitor, dutasteride, requires both ERβ and Transforming growth factor-beta (TGF-β). Dutasteride also induces COX-2 expression, which functions in a negative-feedback loop in TGFβ and ERβ signaling pathways as evidenced by the potentiation of apoptosis induced by dutasteride or finasteride upon pharmacological inhibition or ablation of COX-2. Concurrently, COX-2 positively impacts ERβ action through its effect on the expression of a number of steroidogenic enzymes in the ERβ-ligand metabolic pathway. Therefore, effective combination pharmacotherapies, which have included non-steroidal anti-inflammatory drugs, must take into account biochemical pathways affected by 5AR inhibition and opposing effects of COX-2 on the tissue protective action of ERβ.

Our laboratory seeks to identify novel gene signatures that can be utilized as biomarkers for symptomatic BPH patients to predict response to NSAIDs or to direct therapeutic development towards a drugable target in BPH, ERβ. We utilize 3D cultures of human prostate epithelial cells (PrECs) to provide mechanistic insights into the putative loss of epithelial integrity in BPH. Finally, the novel, unique genetic changes (i.e. gene signatures) associated with inflammatory mediators (i.e. Cox-2) or responsive to NSAIDs in cultured human PrECs are being assessed in patient samples, including from a clinical trial that utilizes combined 5AR and NSAID treatment. Thus, these studies will directly test the utility of identified gene signatures for predicting BPH patient responses to NSAIDs and stimulate the development of new drugs that target ERβ and/or enhance the therapeutic efficacy of NSAIDs.
W. Chet de Groat, Ph.D.
Distinguished Professor
Ph.D., University of Pennsylvania Medical School, 1965

Dr. de Groat is interested in the autonomic nervous system and the neural regulation of pelvic visceral functions. Current studies focus on the reflex control of the urogenital tract and the mechanisms underlying transmission at central and peripheral autonomic synapses. These experiments are designed to examine (1) the neurotransmitters in the reflex pathways, (2) neuroplasticity during postnatal development or following neural injury, (3) the neural pathways responsible for the detection of visceral pain, and (4) the actions of drugs used to treat urogenital dysfunction. Experiments are conducted on a variety of preparations ranging from intact animals to isolated tissues, like spinal cord slices and dissociated neurons.

Julie Eiseman, Ph.D.
Professor
Ph.D., Cornell University Medical College, 1980

Research in the Eiseman laboratory is directed at the preclinical evaluation of potential anti-cancer agents. Studies include the determination of the maximum tolerated dose, pharmacokinetics, pharmacodynamics and efficacy. The laboratory is also interested in non-invasively measuring compounds with absorbance spectra in the long visible range.

Specific studies include the pharmacokinetics and efficacy of the pyrimidine compounds, fluorodeoxycytidine (FdCyd) and gemcitabine (dFdCyd) in combination with a cytidine deaminase inhibitor, tetrahydouridinidine in CD2F1 mice and SCID mice with human pancreatic cancer xenografts.

The pharmacokinetics and efficacy of tubulin interactive agents including docetaxel, paclitaxel and 6-epidictyostatin are also under investigation. Studies with docetaxel have examined the interaction with 9-nitrocamptothecin in an ovarian cancer xenograft (SK-OV3) and a physiological based pharmacokinetic model was developed to describe the disposition of docetaxel. This model will be evaluated for its usefulness in predicting patient docetaxel pharmacokinetics.

Dr. Eiseman is interested in understanding the mechanisms involved during photodynamic therapy with Pc 4 and other phototherapeutic agents and use elastic scattering spectrometry to measure changes in drug concentrations and hemoglobin saturation during and following photodynamic therapy. For these studies, we measure the concentrations of the drug and hemoglobin non-invasively as well as through destructive methods such as HPLC and LC/MS-MS.

Other agents investigated include a wide range of potential cancer chemotherapeutics including DB-67, CKD-602, 2,2-dimethylbutyrate, DA-3003-1, Zebularine, 17-allyl aminogeldanamycin and 17-dimethylamino-geldanamycin.

Keri Fogle, Ph.D.
Research Instructor
Ph.D., Columbia University, 2007

The ATP61 Drosophila model of mitochondrial encephalomyopathies shares common features with human diseases, including Maternally Inherited Leigh Syndrome (MILS) and Neuropathy, Ataxia, and Retinitis Pigmentosa (NARP). Some of the hallmarks of these diseases include neurodegeneration and seizure-like activity that is often unresponsive to commonly-used anti-epileptic drugs.
Dr. Fogle is using whole-cell patch clamp electrophysiology of the intact fly brain to explore the molecular mechanisms which underlie the neurobiological symptoms in the ATP61 model, specifically the membrane channel complexes which may couple metabolic disruption to neuronal dysfunction and hyperexcitability, and thus represent targets for novel therapeutic interventions.

**Peter Friedman, Ph.D.**  
*Professor & Vice Chair, Academics*  
*Ph.D., SUNY Upstate Medical Center, 1975*

Studies in Dr. Friedman’s laboratory focus on spatiotemporal regulation of protein-protein interactions governing GPCR signaling and function. We are especially interested in the parathyroid hormone receptor (PTHR), which controls extracellular mineral ion homeostasis and bone turnover. Key advances have been made in understanding cell-specific PTHR signaling, trafficking, and post-translational modifications.

Recent observations indicate that PTHR activation, desensitization and endocytosis are mediated through distinct structural states that derive from specific interactions between ligand and receptor. Agonist- or antagonist-occupied receptor states induce discrete conformations with accessibility to intracellular receptor domains. The differential or inducible involvement of these domains in coupling to G proteins may represent a molecular basis for ligand-selective responses not only for the PTHR, but also for other G protein-coupled receptors, and are novel drug targets.

Current work is directed at elucidating the molecular and structural mechanisms of how cytoplasmic PDZ proteins such as NHERF1 legislate cell-, ligand-, and stage-specific receptor trafficking. The resulting information will be valuable in understanding mineral ion homeostasis under normal conditions, as well as disordered calcium balance in renal failure, hyperparathyroidism, or osteoporosis.

**William Furey, Ph.D.**  
*Professor*  
*Ph.D., The State University of New Jersey, 1977*

Dr. Furey’s research involves the structure determination and analysis of large biological molecules and complexes by x-ray crystallography, and correlating the results with known functions. The work currently focuses on thiamin (vitamin B1) dependent enzymes and cell cycle regulating enzymes, as well as crystallographic methods development. Results of these studies could lead to development of therapeutic agents directed against pathogenic organisms, and anti-cancer drugs.

The pyruvate dehydrogenase multienzyme complex (PDHC, MW 4.7 million Daltons, 60 protein subunits & 60 active sites for the E. coli version) is present in most organisms and is critical for carbohydrate metabolism where it converts pyruvate, the product of glycolysis, to acetyl-CoA via a complicated process of substrate channeling within the confines of the complex. Structural analyses of the complex and its three major enzymatic components E1 (24 copies), E2 (24 copies), & E3 (12 copies) are underway, and Dr. Furey has already determined high resolution crystal structures for some of the components and reaction intermediates from the E. coli version.

The E1 components are rate determining and require thiamin diphosphate as a cofactor, but must interact with a flexible segment [lipoyl domain (LD) and associated lipoamide side chain] on an E2 to transfer the first reaction product, an acetyl group, to the E2 active site. The acetyl group is then transferred to co-enzyme A within the E2 active site, and the product acetyl-CoA is released. The E2 bound lipoamide group (now reduced) then moves to an E3 (FAD dependent) active site, where it is oxidized to restore the initial conditions. Binding of the flexible segment to E1 and E3 subunits is mediated by additional binding to a peripheral subunit-binding domain (PSBD), shown bound to E1.
Mechanistic details regarding the catalytic reactions in each active site are sought, as well as identifying structural aspects critical for assembly of the individual components to form the complete multienzyme complex. Specific mutations in some of the components are associated with hereditary diseases in humans, and detailed analyses of the structure-function relationships may suggest development of plausible therapeutic agents to counter the effects of the mutations. Additionally, given the critical nature of this system in overall energy production for cellular function, development of inhibitors binding at any of the catalytic sites, or at sites disrupting protein-protein assembly, may considerably weaken or kill the organism. Lack of appreciable sequence homology between PDHc’s from humans and pathogenic bacteria therefore suggests that effective, pathogen specific antibacterial agents may be developed.

Early expression or over expression of Cdc25 proteins can cause the cell to prematurely progress leading to oncogenic effects, making these enzymes exciting targets for anti-cancer drug development. As part of a collaborative effort with Dr. John Lazo’s group, several potent inhibitors of Cdc25 proteins have been discovered, and structural analysis of their complexes with the enzymes are underway to reveal both where and how these inhibitors function. Dr. Furey has crystallized the catalytic domain of Cdc25b and determined its high-resolution structure. His group is currently co-crystallizing the catalytic domain with several inhibitors, as a step towards development of effective anti-cancer agents via structure-based drug design procedures.

In collaboration with the Hauptman-Woodward Institute for Medical Research, Dr. Furey's group is developing new computational methods for solving macromolecular crystal structures by automated techniques. This work involves creating and developing a software package BnP, which is a merging of the PHASES package developed in the Furey lab, and the SnB package developed in Buffalo. A simple, graphical user interface is developed to enable automatic creation of an interpretable electron density map starting from observed x-ray diffraction data, with only a few mouse clicks and text field entries required. This will invoke automatic scaling of data, determination of heavy atom/anomalous scatterer sites, refinement and validation of sites, calculation of protein phases, phase refinement, and phase improvement via solvent flattening/negative density truncation. A few more mouse clicks enable automated building of a complete or nearly complete model by interfacing with other externally developed software. The idea is to make it simple for novices to determine good quality crystal structures, while enhancing the productivity of more sophisticated users as well.

Ferruccio Galbiati, Ph.D.
Professor
Ph.D., University of Milan, 1996

Most cells cannot divide indefinitely due to a process termed cellular senescence. Because cancer cells need to escape cellular senescence in order to proliferate and eventually form tumors, it is well accepted that cellular senescence is a powerful tumor suppressive mechanism. In addition, since several molecular changes that are observed in senescent cells occur in somatic cells during the aging process, investigating the molecular mechanisms underlying cellular senescence will also allow us to better understand the more complicated aging process. Thus, molecules that regulate cellular senescence represent potential therapeutic targets for the prevention/treatment of cancer as well as the fight against aging.

Our work is directed at unraveling the role of caveolin-1 as a novel mediator of cellular senescence. Caveolin-1 is the structural protein component of caveolae, invaginations of the plasma membrane involved in signal transduction. Caveolin-1 acts as a scaffolding protein to concentrate, organize, and functionally modulate signaling molecules within caveolar membranes.

Dr. Galbiati’s laboratory was the first to demonstrate that caveolin-1 plays a pivotal role in oxidative stress-induced premature senescence. We found that oxidative stress upregulates caveolin-1 protein expression through the p38 MAPK/Sp1-mediated activation of the caveolin-1 gene promoter. We also demonstrated that upregulation of caveolin-1 protein expression promotes premature senescence through activation of the
p53/p21Waf1/Cip1 pathway by acting as a regulator of Mdm2, PP2A-C, TrxR1 and Nrf2. Moreover, we found that caveolin-1-mediated premature senescence regulates cell transformation and contributes to cigarette smoke-induced pulmonary emphysema, directly linking caveolin-1’s function to age-related diseases.

Taken together, these data indicate that caveolin-1 plays a central role in the signaling events that lead to cellular senescence. We are currently investigating, at the molecular level, the signaling pathways that link caveolin-1 function to oxidative stress-induced premature senescence. These investigations will contribute to elucidate the molecular mechanisms underlying aging and cancerous cell transformation.

**Eun-Ryeong Hahm, Ph.D.**
*Research Instructor*
*Ph.D., Seoul National University, Seoul, Korea, 2003*

**Ryan Hartmaier, Ph.D.**
*Research Instructor*
*Ph.D., Baylor College of Medicine, 2010*

Dr. Hartmaier’s research focuses on the discovery and understanding of genetic changes acquired during breast cancer metastasis. Under the mentoring of Dr. Adrian Lee, Dr. Hartmaier has applied many Next-Generation Sequencing (NGS) technologies to paired primary and metastatic tumors (from the same patient). This includes: whole genome paired-end sequencing, exome sequencing, RNA sequencing, and large-insert whole genome mate-pair sequencing. Analysis of this data allows the identification of somatic mutations that occur during breast cancer tumorigenesis, metastasis, and drug resistance.

Through many studies, including The Cancer Genome Atlas (TCGA), we know that primary tumors, in general, have acquired an extreme number of mutations during tumorigenesis. The overwhelming majority of these mutations are not ‘driver mutations’ that the tumor relies on for its carcinogenic phenotype. During metastasis, we and others have shown, the primary tumor enters an ‘evolutionary bottleneck’ and the majority of this genetic diversity is lost. Thus, by studying paired primary and metastatic tumors we can effectively find the genetic events driving metastasis and drug resistance.

Paired-end sequencing (PES) is the most common NGS strategy and is extremely powerful at detecting single nucleotide variants (SNVs) or small insertions and deletions (INDELs). PES can be used to detect structural variations (SVs) as well but this is mostly limited to ‘unbalanced events’ such as amplifications or deletions. For ‘balanced events’ such as inversions and translocations, detection by PES can be difficult or impossible, particularly when the SV is flanked by a repetitive element. Mate-pair sequencing (MPS), however, is specifically designed to identify SVs even when present in repetitive regions. Below is a theoretical example comparing the ability of PES and MPS to detect an inversion flanked by low-copy repeat (LCR) regions.

Combining these different sequencing strategies on paired primary-metastasis tissues allows a comprehensive understanding of the genetic events that occur during metastasis and drug resistance.

**Jing Hu, Ph.D.**
*Visiting Associate Professor*
*Ph.D., Karolinska Institute, Sweden, 1997*

The main focus of Dr. Hu’s research is to understand how posttranslational modifications—particularly by ubiquitin-related modifiers such as SUMO—of cancer-related factors, regulate cellular process in cancer biology and treatment. Through our research we hope to provide a novel angle of understand why chemotherapy often fails. Our goal is to identify novel molecular targets or events that have potential to
guide the clinical development of new means to inhibit tumor progression and chemoresistance.

One of her research aims is to investigate how HDAC2 (Histone deacetylase 2) promotes tumorigenesis through enhancing substrate sumoylation. HDAC2 is a key regulator of oncogenic processes and is elevated in several human cancers, but how HDAC2 functions to promote carcinogenesis remains elusive. A commonly known feature of HDAC is to remove the acetyl group from an acetylated lysine and, consequently, our view of HDAC has for many years been solely from the deacetylase perspective. Intriguingly, we have found that HDAC2 possesses a deacetylase-independent sumoylation-promoting activity. To date her lab has identified two sumoylation substrates of HDAC2, including eukaryotic initiation factor 4E (eIF4E).

eIF4E is an mRNA cap-binding factor. As a potent oncogene, eIF4E is found elevated in many human cancer including colorectal cancer (CRC). High levels of eIF4E contribute to carcinogenesis by stimulating protein synthesis of cancer-related genes—genes related to growth, proliferation and apoptosis. We find that sumoylation activates eIF4E-dependent mRNA translation and is required for eIF4E’s anti-apoptotic and oncogenic properties. We further show that HDAC2 promotes sumoylation of eIF4E, resulting in activation of protein translation of a subset eIF4E-target genes. These findings raise the possibility that HDAC2 promotes tumorigenesis through upregulating sumoylation of eIF4E. We are currently validating the functional role of HDAC2 sumoylation activity in intestinal tumorigenesis and dissecting the molecular basis underlying it.

She is also interested in uncovering the HDAC mechanism responsible for the limited efficacy of histone deacetylase inhibitor (HDACi) treatment in solid tumors. HDACi is a promising new class of anticancer drugs. Two HDACis—Vorinostat (SAHA) and Romidepsin—are licensed by the United States FDA for the treatment of advanced cutaneous T-cell lymphoma. Despite its low clinical efficacy as a single agent, HDACi shows promise in combination therapy in lung cancer patients suggesting that the full therapeutic potential of HDACis will probably be best realized through a combination with other anticancer agents. Currently there are about 10 ongoing clinical trials testing HDACis for colon cancer (ClinicalTrials.gov); therefore it is critical to identify resistance mechanisms that can lead to strategies that increase HDACi therapeutic potential in CRC. We are investigating HDACi resistance from a previously unexplored angle—HDAC2-mediated sumoylation. We hope the information gained from this study could lead to identification of new drug combinations and more rationally designed future trials.

Yi Huang, Ph.D.
Assistant Professor
Ph.D., Medical University of South Carolina, Charleston, SC, 2001

Dr. Huang’s research interests focus on the investigation of epigenetic regulation of gene expression in breast cancer. There is a growing body of evidence to suggest that changes in the activity of chromatin-modifying enzymes contribute to the uncontrolled cell proliferation and tumorigenesis. Importantly, epigenetic changes, unlike mutations or loss of chromosomes, are reversible that provides a rational mechanism for applying small molecule drugs as personalized therapeutics to target these changes in cancer. Our main research objective is to define in depth the mechanisms and biological consequences of functional interplay between chromatin-modifying enzymes in breast cancer development. We are also interested in identifying novel, small molecule reagents that act as selective inhibitors of important chromatin-modifying enzymes to target more specifically the small regions of chromatin and the subset of genes that are associated with most prominent alterations in the breast cancer genome. Our recent work demonstrated that activities of histone lysine-specific demethylase 1 (LSD1) and histone deacetylases (HDACs) are functionally linked in breast cancer, especially in triple negative breast cancer (TNBC). LSD1 inhibitor in combination with HDAC inhibitor displays superior synergy in blocking growth and metastasis of TNBC cells. We are investigating the precise mechanisms underlying orchestrated LSD1 and HDAC crosstalk in breast cancer and determining how the dysregulated interaction of histone-modifying enzymes leads to aberrant gene silencing and aggressive phenotype of TNBC. We are also
studying if targeting LSD1/HDAC crosstalk by novel inhibitors are more efficacious in hindering TNBC growth than current strategies and thus represent a novel targeted therapy for this devastating disease.

Another focus of the lab is to determine the role of polyamine biosynthesis pathway in mediating the activity of estrogen receptor signaling in breast cancer. Since the growth of ER positive breast cancer largely relies on the action of estrogen, antagonizing ER or its ligands is one of the most important strategies for breast cancer treatment and prevention. Our recent study demonstrated that inhibition of a key polyamine biosynthesis enzyme, ornithine decarboxylase (ODC), diminishes ERα expression that leads to the loss of expression or function of several important ERα target or partner genes including PR, NF-kB and cyclin D1. Loss of ODC disrupts the binding of Sp1 and its newly identified co-factors (Pokemon, PARP-1, myc, etc.) to ERα minimal promoter element. Clinically, patients that initially respond to anti-estrogen endocrine therapy will gradually develop resistance. This constitutes a major clinical challenge in breast cancer therapy and prevention. Therefore, development of more effective estrogen receptor modulators is necessary for improving the therapeutic efficacy of breast cancer. We are investigating how ODC mediates the expression and activity of ERα and elucidating the potential role of ODC in endocrine resistance development in breast cancer.

Edwin Jackson, Ph.D.
Professor
Ph.D., University of Texas at Dallas, 1979

Purine Pharmacology: Adenosine is an endogenous purine that regulates most physiological systems. We are investigating (using a variety of molecular, analytical, cellular and physiological tools and using several strains of genetically modified animals, as well as conducting studies in patients): 1) the production of adenosine from 3’,5’-cAMP and 2’,3’-cAMP (the cAMP-adenosine pathways); 2) the modulation of adenosine levels by guanosine; 3) the roles of adenosine in regulating the sympathetic nervous system, heart, vascular system, kidneys, bladder, brain and immune system; 4) the effects of adenosine on cardiac fibroblasts, vascular smooth muscle cells, vascular endothelial cells, glomerular mesangial cells, renal epithelial cells, T cells and B cells; 5) the role of exosomes in adenosine biochemistry; 6) how to modulate the adenosine system with drugs to treat cardiovascular and renal diseases, traumatic brain injury, cancer and HIV infected patients.

Cardiovascular and Renal Pharmacology: Our recent studies indicate that NPY1-36 (a peptide released from sympathetic nerves) and PYY1-36 (a peptide released from the intestines) trigger proliferation of and extracellular matrix production by preglomerular vascular smooth muscle cells (PGVSMCs) and glomerular mesangial cells (GMCs) in kidneys from genetically-hypertensive animals, a phenomenon mediated via Y1 receptors and that involves signaling by RACK1 (receptor for activated C kinase 1). Dipeptidyl peptidase IV (DPPIV) metabolizes NPY1-36 and PYY1-36 (Y1 receptor agonists) to NPY3-36 and PYY3-36 (inactive at Y1 receptors). We are investigating whether a new class of antidiabetic drugs (DPPIV inhibitors) may adversely affect the kidneys of hypertensive subjects by preventing the conversion of PYY1-36 and NPY1-36 to less active metabolites and thereby promoting inappropriate cell proliferation and extracellular matrix production.

Tija Jacob, Ph.D.
Assistant Professor
Ph.D., University of California, Berkeley, 2002

How does the neurotransmitter GABA produce myriad forms of inhibition in the central nervous system (CNS), restraining and shaping electrical activity to prevent anxiety, agitation, seizures, chronic pain and sleep disturbance? The majority of fast synaptic inhibition in the CNS is mediated by GABA type A neurotransmitter receptors (GABA<sub>A</sub>Rs) which are Ca<sup>2+</sup> selective ligand-gated ion channels composed of 5 subunits (from up to 17 different subunits), with differential expression across brain regions, cell types and subcellular localization.
The Jacob lab’s broad goal is to understand the impact of dynamically regulated GABA_A_R surface levels and distribution in normal development and pathological conditions. The lab uses a combination of molecular, biochemical, cell biological and live-imaging approaches. GABA_A_R are the sites of action of many clinically important drugs, including the benzodiazepines (BZ), which are front line treatments for anxiety, insomnia, schizophrenia and epilepsy. The Jacob lab is investigating modulation of GABA_A_R trafficking and synaptic inhibition by BZ and other GABAergic agents.

Another area of research in the lab focuses on the role of GABAergic signaling in CNS development and plasticity. The majority of excitatory synapses in the brain are located at the end of dendritic spines, small protrusions from neuronal processes, with neighboring GABAergic synapses predominantly located on dendritic shafts. We have shown that higher GABA_A_R surface levels leads to more inhibitory synapses, enhanced inhibitory synaptic transmission and a deficit in mature dendritic spines. Alterations in the excitatory/inhibitory ratio of neuronal signaling, abnormal spine morphology and mutations in GABA_A_R subunits are associated with many neurological disorders including autism and other neurodevelopmental disorders. The Jacob lab is investigating the contribution of GABAergic inhibition to dendritic spine morphology, movement and plasticity. These studies aim to improve understanding of how GABAergic dysfunction contributes to human neurodevelopmental disorders including autism.

Yu Jiang, Ph.D.
Associate Professor
Ph.D., Yale University, 1995

Dr. Jiang’s laboratory is interested in intracellular signaling pathways governing cell growth and metabolism. The laboratory’s current research projects concern the signaling mechanism of the mammalian target of rapamycin (mTOR). mTOR is a protein ser/thr kinase that plays a key role in translation, autophagy and mitochondrial biogenesis. Its activity is regulated by signals of various origins, including nutrient, growth factor, energy and stress. The laboratory has previously identified FKBP38 that acts as an inhibitor of mTOR. Three projects centering on the role of FKBP38 in mTOR regulation are on-going. The first project concerns the activity of mTOR in mitochondrial function. We have recently found that FKBP38 is involved in recruitment of mTOR to mitochondria. The project investigates the role of the mitochondrial localized mTOR in mitochondrial function and cell senescence. The second project aims at the mechanism of FKBP38 in apoptosis regulation. FKBP38 has been shown to interact with the anti-apoptotic proteins, Bcl-2 and Bcl-xL. The project is to determine whether nutrient, growth factor and oxygen levels control the anti-apoptotic activity of Bcl-2 and Bcl-xL through FKBP38. The third project focuses on the role of primary cilium in mTOR regulation. Primary cilium is a vital cellular organelle that functions as a signaling hub in many eukaryotic cells. mTOR has been recently found to be a key effector of primary cilium-mediated signaling. This project explores the mechanisms through which primary cilium controls mTOR activity.

Thomas Kensler, Ph.D.
Professor
Ph.D., Massachusetts Institute of Technology 1976

Research interests in Dr. Kensler’s laboratory focus on the biochemical and molecular mechanisms involved in the induction of cancer by chemicals to serve as a basis for the prevention, interruption or reversal of these processes in man. One of the major mechanisms of chemical protection against carcinogenesis, mutagenesis and other forms of toxicity mediated by carcinogens is the induction of enzymes involved in their metabolism, particularly enzymes such as glutathione S-transferases, UDP-glucuronosyl transferases and NAD(P)H:quinone reductase that facilitate the detoxication and elimination of carcinogens. Furthermore, animal studies indicate that induction of these cytoprotective enzymes is a sufficient condition for obtaining chemoprevention and can be achieved in many target tissues by administering any of a diverse array of naturally-occurring and synthetic chemical agents. Our work utilizes animal and cell culture models to elucidate mechanisms of inhibition of
aflatoxin hepatocarcinogenesis by dithiolethiones such as oltipraz, isothiocyanates such as sulforaphane and triterpenoids such as CDDO-Im. While induction of glutathione S-transferases clearly play an important role in chemoprevention of aflatoxin hepatocarcinogenesis, ongoing studies are seeking to identify additional genes induced by these agents. The Keap1-Nrf2 signaling pathway is activated by these classes of chemopreventive agents and leads to increased expression of genes that attenuate oxidative stress and inflammation among other actions. Their contributions to protection against carcinogenesis are under investigation.

A practical goal of his research has been to develop the tools to test the hypothesis that enzyme induction is a useful strategy for chemoprevention in humans. Hepatocellular carcinoma is the leading cause of cancer death in parts of Asia and Africa and may relate to hepatitis B virus infection and aflatoxin ingestion. Longitudinal surveys and prospective case-control studies in Qidong, P.R. China demonstrate consistent exposure of individuals in this region to aflatoxins and indicate a prime role for aflatoxin in the etiology of liver cancer, respectively. As a consequence, we have conducted clinical chemoprevention trials of oltipraz and other agents in Qidong. The initial randomized, placebo-controlled intervention of oltipraz demonstrated an increased excretion of aflatoxin-mercapturic acid, a derivative of the aflatoxin-glutathione conjugate, in the urine of participants receiving oltipraz. This study highlights the general feasibility of inducing Nrf2-regulated enzymes in humans. Follow-up trials are evaluating more effective agents and are assessing whether protective alterations in aflatoxin metabolism can be sustained for extended periods of time and whether diminished incidence of liver cancer can be achieved in this high-risk population.

Nicholas Khoo, Ph.D.
Research Assistant Professor
Ph.D., University of Iowa, 2003

Dr. Khoo investigates the basic molecular mechanisms underlying the development of metabolic syndrome and the role of electrophilic lipids, particularly nitro-fatty acids (NO2-FAs) in preventing this pathogenesis. His specific research projects include:

1) Determination of the molecular mechanism(s) responsible for the anti-inflammatory cell signaling actions of electrophilic NO2-FAs resulting in insulin sensitivity.

Obesity induces chronic inflammatory responses that are characterized by abnormal cytokine production, increased reactive oxygen species (ROS) generation and activation of inflammatory signaling pathways. Preliminary studies demonstrate these inflammatory conditions induce the oxidation and nitration of fatty acids to electrophilic products, specifically NO2-FA derivatives, that serve as potent anti-inflammatory cell signaling mediators. These electrophilic NO2-FA species potently bind peroxisome proliferator-activated receptors (PPARs), inhibit NF-kB activity and induce heme oxygenase (HO)-1. The treatment of NO2-FAs results in improved glucose homeostasis in mouse models of obesity and diabetes. The electrophilic nature of these NO2-FA signaling molecules and their anti-inflammatory properties are being examined using cultured mammalian cells as well as mouse models of obesity and diabetes. Additionally, a mass spectrometer approach is being used to characterize the formation of NO2-FA derivatives in insulin responsive tissues from murine models of obesity and diabetes (ob/ob, db/db or high fat diet). This is being complemented by similar approaches in cell culture.

Identify molecular signaling pathways modulated by NO2-FA treatment in mice subjected to a high-fat diet. Currently, three putative pathways for the anti-inflammatory actions of electrophilic NO2-FAs are being examined. The signaling pathways of all three PPAR isotypes, NF-kB and HO-1 are being explored in insulin-responsive tissues (adipose, liver and muscle).

Define mechanistic roles of all three PPAR isotypes, NF-kB and HO-1 in cultured cells. 2-FA-induced HO1 expression.
2) Determination of the impact of NO2-FA derivatives on ROS and oxidative stress in insulin-responsive cultured cells and tissues of mouse models of obesity and diabetes. While oxidative stress and ROS are emerging as key culprits in the pathogenesis of obesity-induced insulin resistance, the sources of ROS remain unclear. Emerging studies suggest a link between mitochondrial dysfunction, insulin resistance and diabetic complications, suggesting that mitochondrially derived ROS could play a role in pathogenesis. How does this increase in ROS result in oxidative stress? Is there a decrease in antioxidant enzyme expression and activity in insulin-responsive tissues? These questions are currently being addressed by utilizing cutting edge techniques to detect ROS levels and antioxidant enzyme activity/expression in the cell culture and mouse models of obesity and diabetes described above.

3) The PPAR conundrum- Identification of novel PPAR agonists. The activation of PPARs has been shown to regulate glucose and lipid metabolism. These receptors are molecular targets for a number of marketed drugs. The hypolipidemic fibrates activate the isotype PPARα whereas PPARγ is the molecular target of thiazolidinedione (TZD) class of antidiabetic drugs. The activation of PPARγ-dependent downstream signaling has shown to improve insulin sensitivity. Rosiglitazone works as an insulin sensitizer by activating PPARγ and its downstream signaling pathways. Unfortunately, concerns about the severe adverse side effects have drastically limited the use of rosiglitazone despite excellent glycemic control in patients with diabetes. Thus, the development of new therapeutic strategies, such as dual PPARα/γ activators or selective PPARγ partial agonists, that retain their antidiabetic efficacy without adverse side effects are appealing, such as NO2-FAs.

In summary, these research interests will generate insights into mechanisms leading to obesity and its associated myriad of health problems and/or diseases such as diabetes, atherosclerosis and other cardiovascular complications, which will hopefully elucidate novel preventative and therapeutic strategies. The potential for electrophilic NO2-FA mediated therapy to prevent obesity-induced type 2 diabetes complications, without the known secondary effects exerted by TZDs, is currently being studied.

Joan M. Lakoski, Ph.D.
Professor
Ph.D., University of Iowa, 1981

Elucidating the cellular and molecular neuroparmacology of the aging brain is the focus of the Lakoski laboratory. Using multidisciplinary approaches to investigate biogenic amine receptor expression and function, both normal and pathological aging processes are being investigated in young, middle-aged and senescent small animal models. We are investigating the roles of the steroid hormones estrogen and corticosterone on serotonin receptors, their receptor-effector coupling to G-proteins and related signaling transduction cascades, including the 5-HT1A and 5-HT2A receptor subtypes, and the serotonin neurotransporter (SERT) in discrete brain regions including cortical, hippocampal and midbrain regions; radioligand binding techniques, receptor autoradiography and functional neurochemical assays are among the technical approaches used to study the impact of the circulating hormone environment on the aging serotonergic neuronal system.

Related ongoing studies are utilizing in vivo microPET image analysis techniques to elucidate SERT expression and function with respect to aging and hormone treatment. In addition, the impact of selective neurotoxic insults to the dopamine-containing neuronal system is being investigated using behavioral, neurochemical and molecular approaches to better understand how this neurotransmitter system responds and recovers from neuronal injury across the lifespan.

Our goal is to contribute new information to understand the biology of central nervous system aging, including normal and neurodegenerative processes, in neurotransmitter systems established as key components in cognitive declines, mood disorders, and stress-related disorders common in the elderly. Ultimately, our aim is to improve the quality of life with advancing age by pharmacological interventions to
delay the onset of neuronal decline and/or enhance endogenous repair mechanisms of the biogenic amine neurotransmitter systems.

**Jack Lancaster, Ph.D.**  
*Professor*  
*Ph.D., University of Tennessee Center for the Health Sciences, 1974*

Dr. Lancaster’s present research interests are in the chemical and physical foundations of the biological actions of reactive oxygen and nitrogen species. His most recent project is delineating the cellular functions of dinitrosyliron complexes (DNIC), which show a characteristic signal using electron paramagnetic resonance (EPR) spectroscopy and have been observed in tissues since the 1960’s in a huge variety of pathophysiological conditions. These species contain one iron with two molecules of bound nitric oxide (NO) but the complete molecular structures of these species are essentially unknown, as are possible biological functions. We recently reported data suggesting the cellular origin of the iron and also evidence for two cellular functions, formation of protein nitrosothiols and also protection against cellular injury as a result of hypoxia-induced iron mobilization and consequent oxidative stress (Li et al. J. Biol. Chem. 2014, in press).

**Adrian Lee, Ph.D.**  
*Professor*  
*Ph.D., University of Surrey, Guildford, Surrey, England, 1993*

Dr. Lee is investigating the endocrine regulation of mammary gland development and progression to mammary cancer. Specifically we are interested in interaction between steroid hormones (estrogen and progesterone) with the growth hormone (GH)/insulin-like growth factor-I (IGF-I) axis. These endocrine hormones are all critical for normal mammary development, but have also all been implicated in risk for breast cancer and in breast cancer progression.

They have shown that IGF-IR and IRSs are hormonally regulated in breast cancer, and we have now found that they are also developmentally and hormonally regulated during normal mammary gland development. We have found in breast cancer cell lines that estrogen can sensitize cells to insulin-like growth factor (IGF) stimulation by increasing expression of many of the IGF signaling components such as the IGF-IR and its downstream signaling intermediates IRS-1 and IRS-2.

They have also found that overexpression of IGF-IR, IRS-1, or IRS-2 causes transformation of mammary epithelial cells in culture, combined with epithelial to mesenchymal transition. We have also created transgenic mice that overexpress IGF-IR, IRS-1 or IRS-2 in the mammary gland and all mice develop mammary tumors. Mammary tumors in these mice show multiple cell lineages and expansion of putative mammary stem/progenitor cells. We are currently investigating how these pathways impact upon stem/progenitor cell renewal and cell fate determination.

**Edwin Levitan, Ph.D.**  
*Professor and Vice Chair, Research*  
*Ph.D., Brandeis University, 1986*

The Levitan lab studies biochemical and electrical signaling that controls neuronal and cardiac function with live cell imaging, electrophysiology and molecular biology. Current projects include in vivo imaging of green fluorescent protein (GFP) constructs in transgenic Drosophila nerve cells and serotonin in mammalian brain slices to determine how patterned electrical activity and synapses control transmitter release. We are also studying remodeling of rhythmic electrical activity in the heart and midbrain dopamine neurons by therapeutically important hormones and drugs. Most recently, multiphoton microscopy is being used to image vesicular accumulation and release of a psychiatric drug in the brain.
Tatyana Mamonova, Ph.D.
Research Instructor
Ph.D., Kazakh National Academy of Sciences, Kazakh Scientific Research Institute of Catalysis and Electrochemistry, Almaty, Kazakhstan, 1995

Dr. Mamonova research focuses on molecular modeling of the interactions of the adapter protein EBP50/NHERF1 with its target ligands, including parathyroid hormone receptor and type II sodium-dependent phosphate co-transporters. The goal of these studies will contribute to our understanding and prediction of conformational reorganization and the structure-function relationships in NHERF1 proteins associated with the ligand binding. The results will provide insights into developing drugs for selective therapeutic applications.

Carola Neumann, M.D.
Visiting Associate Professor
M.D., Ludwig-Maximilian’s University Medical School, Munich, Germany, 1997

Roderick O’Sullivan, Ph.D.
Assistant Professor
Ph.D., Institute for Molecular Pathology, Vienna, Austria, 2006

The O’Sullivan lab at the Hillman Cancer Center conducts research into proteins that alter the structural and epigenetic functions of human telomeres. Telomeres are structures at the ends of chromosomes – the integrity of telomeres is an important factor in maintaining genome stability to prevent cancer and accelerated aging. Current efforts in the lab relate to deciphering the relationship between the regulation between chromatin structure and telomere function in the Alternative Lengthening of Telomeres pathway.

Steffi Oesterreich, Ph.D.
Professor
Ph.D., Humboldt University, Max-Delbrück Center for Molecular Medicine, Berlin, Germany, 1992

The Oesterreich lab includes technicians, graduate students and postdoctoral fellows who are trained in a multi-disciplinary research environment to work in basic, translational, and clinical aspects of breast cancer research. Specifically, our research projects focus on the role of co-regulator proteins in estrogen response in breast cancer. Estrogen mediates its potent mitogenic effects through the estrogen receptor (ER), which has been a successful target for endocrine therapy in breast cancer. Despite the success of such treatment, de novo or acquired resistance remains a major problem. A better understanding of how ER works is critical for the development of more efficient therapies, and better prediction for who should receive which form of endocrine therapy.

Over the last years, many dogmas in hormone response have changed, which has opened many exciting novel research areas. Examples are estrogen-mediated repression of gene transcription, and the role of co-repressors in this process, the close connection between estrogen signaling and epigenetic regulation of gene transcription, and the role of regulatory elements which are located far outside the promoter of the estrogen regulated genes, and which might even be on other chromosomes. We are studying these processes using state-of-the-art molecular and cellular techniques, mouse models, and clinical specimens. The ultimate goal of Dr. Oesterreich's research is to use this knowledge for improved diagnosis and endocrine treatment of breast cancer patients.

Patrick J. Pagano, Ph.D.
Professor & Vice Chair, Graduate Education
Ph.D., New York Medical College, 1991
Dr. Pagano’s research focuses on the modulatory role of the adventitia in vascular function and structure under both physiological and pathophysiological conditions. Dr. Pagano’s laboratory was among the first to identify a non-phagocytic NADPH oxidase in the vascular wall, demonstrating a critical role for essential subunit p67phox in its activity. He subsequently cloned vascular p67phox and illustrated its potent activation at the mRNA and protein level in response to the potent pro-hypertensive hormone angiotensin II. Stemming from these early discoveries, Dr. Pagano was the first to develop specific cell- and tissue-permeant peptidic and adenoviral inhibitor of NADPH oxidase, which is widely considered the most specific NADPH oxidase inhibitor available. These and his other more recently developed inhibitors of novel isoforms of NADPH oxidase are expected to provide a platform for the development of new therapies aimed at treating hypertension and other cardiovascular diseases. Moreover, Dr. Pagano is broadly recognized for his pioneering work examining the role of adventitia-derived reactive oxygen species (ROS) and, in particular, superoxide anion and hydrogen peroxide in the modulation of vascular tone, inflammation, and remodeling.

Michael Palladino, Ph.D.
Professor
Ph.D., University of Connecticut, 2000

The Palladino lab uses Drosophila (the fruit fly) as a genetic model system, as well as mice and human cell culture to study progressive neurological and neuromuscular disorders. We are currently focusing on elucidating the mechanism by which mutations affecting Na/K ATPase, triose phosphate isomerase (TPI), and ATP6 function result in RDP (rapid-onset dystonia parkinsonism), glycolytic enzymopathy, and mitochondrial encephalomyopathy, respectively. Our research program is directed toward four main goals: 1) discovering and characterizing novel pathways that cause progressive disease, 2) understanding the physiological, cellular and molecular dysfunction that causes dysfunction in vivo, 3) understanding the molecular basis of progressive diseases, and 4) using our animal model system to identify novel therapeutic approaches.

Guillermo Romero, Ph.D.
Associate Professor
Ph.D., University of Virginia, 1980

G-protein coupled receptors (GPCR) are the largest family of cell surface receptors found in mammalian organisms. These receptors are a major target for drug development. Dr. Romero is interested in the dynamics and traffic of GPCR, with special emphasis on the parathyroid hormone receptor type 1 (PTH1R). His approach is based on the use and development of novel optical techniques to study membrane proteins and their interactions with other cellular components in live cells.

Dr. Romero’s research focuses on two main areas: a) the role of the PDZ proteins sodium-hydrogen exchange regulatory factor (NHERF1) and Disheveled-2 in the regulation of the dynamics and traffic of GPCR; and b) the role of phospholipase D in the regulation of receptor traffic and function.

Dr. Romero’s approach is based primarily on the analysis of the physical properties of molecules of interest in live cells, using advanced optical techniques such as confocal microscopy, fluorescence recovery after photobleaching (FRAP), total internal reflection microscopy (TIRFM), image correlation spectroscopy (ICS), quantum dots, and others. Using these techniques, Dr. Romero has developed novel methods to examine protein-protein interactions in the temporal domain. For example, he has recently demonstrated that the PTH1R is tethered to the cytoskeleton and accumulates in the vicinity of subjacent actin stress fibers, forming bundles that are highly dynamic structures, moving along these bundles much more rapidly than between them.
Because of his interest in traffic, Dr. Romero is actively pursuing new approaches to the study of endocytic processes. To this effect, he recently developed novel methodologies for the purification and preparation of endosomes based on the use of magnetic nanoparticles attached to peptide ligands. In this technique, peptide ligands, such as insulin or epidermal growth factor, are adsorbed to colloidal iron nanoparticles (5-10 nm in diameter) and applied to the external surfaces of cells. These particles are sufficiently small to be internalized via the standard endocytic pathway and, because of the ferromagnetic properties of the colloidal iron, allow for a simple and rapid isolation of the endocytic vesicles containing the particle. He is using this novel technology to study the role of specific proteins in the endocytic pathway in a time-resolved manner.

James Roppolo, Ph.D.
Research Assistant Professor
Ph.D., University of Michigan, 1970

Dr. Roppolo’s research is concerned with the autonomic nervous system’s control of bladder activity in normal animals and those with central nervous system injuries. A variety of techniques are used to examine, at the level of the lumbosacral spinal cord and brainstem, the various neuronal processes that occur in maintaining normal excretory function.

These methods include: (1) anatomical techniques (HRP tracing and immunohisto-chemical techniques) to determine the location of neurons and possible neuropeptides involved in these processes, (2) neurophysiological techniques (evoked potentials, intracellular and extracellular single neuron recordings) to determine the types of neuronal interactions that occur in this system, (3) neuropharmacological techniques (systemic and iontophoretic application of drugs), (4) behavioral techniques and microstimulation of the of the lumbosacral spinal cord.

Francisco Schopfer, Ph.D.
Research Associate Professor
Ph.D., University of Buenos Aires, Argentina, 2001

Dr. Schopfer’s research is focused on the understanding of the biological effects of electrophilic fatty acids. In particular, he studies the mechanism by which nitrated fatty acid activate and signal through peroxisome proliferator-activated receptor gamma (PPARγ). This receptor is the target of currently used antidiabetic drugs (thiazolidinediones). The activation of the receptor regulates fat and glucose metabolism, resulting in an overall decrease of glucose levels to normal values in patients with type II diabetes. The targeting of this receptor by nitrated fatty acids results in a decrease of the glucose levels to normal values like thiazolidinediones, but without the known secondary effects exerted by thiazolidinediones. In addition to the intrinsic therapeutic value of nitrated fatty acid, they will aid in the understanding of the biological mechanism involved in PPARγ activation, leading to improved designs of anti-diabetic drugs targeting the PPARγ receptor.

The role of the PPARγ receptor in diabetes has been well established. Nonetheless, the role of endogenous signaling molecules on the activation of PPARγ is still unclear and under debate. Nitrated fatty acids are endogenously formed and bind to PPARγ with high affinity rivaling Rosiglitazone (thiazolidinediones), resulting in receptor activation. In addition, nitrated fatty acids covalently modify a critical cysteine (cys285) in the ligand binding pocket of PPARγ, promoting a particular conformational change that results in partial receptor activation. This partial activation results in the expression of a particular subset of genes under PPARγ regulation and a biological outcome that differs from the one obtained when activating the receptor with Rosiglitazone. Dr. Schopfer’s work focuses on understanding the mechanism of this selective activation and how it avoids the side effect presented upon full activation by agonist like Rosiglitazone.

Electrophilic fatty acids are constantly formed as fatty acid breakdown products during oxidative stress and as signaling messengers by enzymatic or non enzymatic pathways. Dr. Schopfer studies the formation of
biologically relevant electrophiles, in particular nitrated fatty acids, and their signaling mechanisms. The study involves the detection and characterization of novel electrophiles formed during inflammation. Once the molecules are characterized, a chemical synthesis approach is used to generate enough quantities for biological experiments.

Electrophiles induce an important cellular response that includes the induction of phase II genes. This will in turn set up a more protective environment against damaging electrophilic molecules. A key player in the initiation of this biological response is the Keap 1/Nrf 2 couple. Keap 1 is usually bound to Nrf 2 in the cytoplasm. Upon formation of electrophiles, Keap 1, which contains several highly reactive cysteine, is targeted, dissociates from Nrf2 and is routed to degradation by the proteosome. These lead to Nrf2 nuclear translocation and activation of phase II genes. In particular, we study the mechanism by which different biologically relevant electrophiles target KEAP 1 and activate Nrf 2 responses. In addition, a more general proteomic approach is use to evaluate and characterize different electrophilic cellular protein targets. Once critical targets are identified using a mass spectrometry approach, a functional study of the modification is performed to determine the relevance and its cellular effects.

Sruti Shiva, Ph.D.
Associate Professor
Ph.D., University of Alabama at Birmingham, 2004

Dr. Shiva’s research focuses broadly on understanding the mechanisms by which mitochondrial function is regulated, particularly by reactive oxygen and nitrogen species and the contribution of these mechanisms to cardiovascular health and disease pathogenesis. Using a wide spectrum of techniques ranging from the biochemical study of isolated mitochondria to whole animal models and measurement of bioenergetic function in human blood cells, the Shiva lab is currently engaged in a number of active projects along four major scientific themes:

1 – Utilization of platelets to measure human mitochondrial function in health and disease. Mitochondrial dysfunction plays a role in the pathogenesis of numerous diseases, however data on human mitochondrial function is lacking due to the lack of non-invasive methodology to obtain sufficient quantities of live mitochondria. Platelets contain fully functional mitochondria and we have optimized and validated methodology (utilizing Seahorse XF analysis) to measure human platelet bioenergetics. We have used this technique to show that platelets from different patient cohorts have differing bioenergetic profiles. We are currently comparing platelet bioenergetics in a number of different patient populations to determine whether platelet bioenergetics can be utilized as a biomarker of disease and a measure of disease progression.

2 – The role of platelet mitochondria in hemolytic disease pathogenesis. Sickle cell disease is characterized by severe hemolysis, which has been linked to platelet activation. We recently showed that hemolytic components directly inhibit mitochondrial oxidative phosphorylation leading to the production of oxidant which stimulates platelet activation. Current work in the lab aims to determine the role of this hemolysis-dependent altered mitochondrial function in sickle cell disease as well as expand these studies to other diseases with a component of hemolysis, including sepsis and pre-eclampsia.

3- The mitochondrion as a physiological target for nitrite. Nitrite (NO$_2^-$) is a signaling molecule that mediates a number of biological actions including protection after ischemia/reperfusion, reversal of symptoms of the metabolic syndrome and increased exercise efficiency. However, the molecular mechanisms underlying these actions are unknown. Dr. Shiva’s work demonstrates that nitrite-dependent modulation of mitochondrial function underlies some of nitrite’s protective actions. For example, nitrite-dependent post-translational modification of mitochondrial complex I is crucial for protection after ischemia/reperfusion and the nitrite-dependent stimulation of mitochondrial fusion regulates nitrite mediated preconditioning and adipocyte glucose uptake. Ongoing studies in the lab are investigating the mechanisms by which nitrite modulates mitochondrial efficiency as well as its regulation of mitochondrial kinase signaling.
4 – Myoglobin as a regulator of mitochondrial function. The monomeric heme protein myoglobin, expressed in cardiac and skeletal muscle, has long been known to facilitate oxygen delivery to mitochondria in hypoxic conditions. However, recent studies have shown that myoglobin is expressed in tissues beyond the heart and muscle and has functions beyond oxygen binding and transport. For example, we showed that in hypoxia, myoglobin enzymatically reduces nitrite to nitric oxide, a potent inhibitor of mitochondrial respiration. Our lab is currently investigating how reactions of nitrite, nitric oxide and oxygen with myoglobin regulate mitochondrial function to modulate biological processes such as injury after ischemia/reperfusion, smooth muscle cell proliferation and cancer cell proliferation.

Shivendra Singh, Ph.D.
Professor & UPMC Chair in Cancer Prevention
Ph.D., Banaras Hindu University, India, 1984

Cancer chemoprevention is a relatively new but rapidly emerging sub-discipline in oncology and signifies the use of natural or synthetic agents to reverse or delay the process of carcinogenesis. Long latency of most epithelial cancers presents a large window of opportunity for intervention to prevent or slow disease progression. The research interests of the Singh laboratory include molecular characterization of novel cancer chemopreventive agents and rational design of mechanism-driven combination chemoprevention regimens. Cellular and transgenic animal models are used to screen potential cancer chemopreventive constituents from dietary and medicinal plants. Cutting edge cellular, molecular biological, Omics (metabolomics and proteomics), structural biology, and imaging techniques (MRI and bioluminescence) are used to (a) determine the mechanism of action of promising cancer chemopreventive agents, (b) monitor effects on cancer progression, and (c) identify biomarkers predictive of tissue exposure and possibly response. Some of the agents under active investigation in the Singh laboratory include: cruciferous vegetable-derived isothiocyanates, garlic-derived organosulides, and medicinal plant constituent withaferin A. As an example, recent published work from the Singh laboratory indicates suppression of glycolysis in mammary cancer prevention by withaferin A in a transgenic mouse model (JNCI, In Press, 2013). Likewise, complementary cellular and molecular biological, targeted proteomics, and molecular modeling techniques were used to identify beta-tubulin as a novel target of cancer cell growth arrest by withaferin A (WA).

Robert Sobol, Ph.D.
Associate Professor
Ph.D., Temple University, 1991

DNA damage is implicated as playing a causal role in numerous disease processes. Hence, it is suggested that DNA repair proteins, which maintain the integrity of the nuclear and mitochondrial genomes, play a critical role in reducing the onset of multiple disease phenotypes. Conversely, the requirement for DNA repair and genome maintenance in response to radiation and genotoxic chemotherapeutics implicates DNA repair proteins as prime targets for improving response to currently available anti-cancer regimens. Further, cancer-specific DNA repair defects offer novel approaches for tumor-selective therapy. It is now expected that all cancer cells will be found to be defective in some aspect of DNA repair encoded by one of the 150 different proteins that catalyze DNA repair. To help in our understanding and treatment of cancer, geneticists and molecular biologists must explore the detailed consequences of an alteration in each of these repair pathways. It is our expectation that a detailed genetic and mechanistic understanding of the cellular phenotypes associated with specific DNA repair deficiencies will offer the opportunity to identify novel drug targets, optimize and validate new small molecule inhibitors of DNA repair and provide a mechanistic underpinning for the development of tumor selective therapeutic strategies.

In Dr. Sobol’s lab, they have identified two compensatory pathways that respond to defects in DNA repair mediated by the base excision repair pathway, including proteins involved in the synthesis and degradation of
poly-ADP-ribose (PAR) and those involved in the biosynthesis of NAD$^+$. In particular, we study the convergent roles of DNA Repair, PAR metabolism and NAD$^+$ biosynthesis in response to chemotherapy.

Novel approaches to enhance tumor cell cytotoxicity of alkylating agents: Glioblastoma is the most commonly diagnosed brain malignancy and a major cause of cancer related death in the United States. Limited success in the treatment of glioblastoma has been demonstrated with the alkylating agent Temozolomide (TMZ). The overall goals of this project are to discover strategies to circumvent resistance to TMZ and enhance the cytotoxicity and efficacy of this alkylating agent. The base excision repair (BER) pathway provides significant resistance to TMZ by repairing damaged bases but some of the activity of TMZ is due to the accumulation of cytotoxic BER intermediates that result from incomplete or failed repair (termed BER Failure). As the rate-limiting enzyme in BER, DNA polymerase β (Polβ) is important to facilitate repair and to maintain cell survival following DNA damage. Therefore, inhibition of Polβ will enhance TMZ response. We posit that BER mediated by Polβ is a regulated process that signals BER failure through poly(ADP)ribose (PAR) synthesis and NAD$^+$/ATP depletion by a process that requires activation of PARP1 & PARP2 and is regulated by the enzyme PARG. Specifically, we hypothesize that TMZ efficacy can be increased in glioma cells by increasing post-translational modification and inhibition of Polβ. BER failure-induced cell death results from energy (NAD$^+$ & ATP) depletion due to elevated PAR synthesis mediated by the PARP1/PARP2 BER sensor complex, suggesting that the response to TMZ can be enhanced via increased PAR synthesis or further depletion of cellular NAD$^+$ synthesis and/or deregulation of the BER enzyme PARG. Overall, we are testing the hypothesis that the BER pathway is a determinant of resistance to TMZ and therefore selective targeting of the BER pathway will significantly enhance TMZ efficacy.

Development and characterization of isogenic DNA Repair deficient human cell lines: To extend our analysis beyond base excision repair, we have embarked on a large-scale project for the development, characterization and transcriptome analysis of isogenic human cell lines deficient in all known DNA repair genes (>150) in three unique cell backgrounds (glioma, breast cancer and neuronal). These include genes involved in Base Excision Repair, Direct Reversal of Damage, Mismatch Excision Repair, Nucleotide Excision Repair, Homologous Recombination, Non-homologous End-Joining, the modulation of nucleotide pools, DNA polymerases, editing and processing nucleases, the Rad6 pathway, Chromatin Structure, DNA Repair genes defective in diseases and conserved DNA Damage Response genes and Fanconi Anemia/DNA crosslink repair. Further, each will be analyzed for alterations in PAR metabolism and NAD$^+$ biosynthesis. With the expectation that DNA repair capacity impacts basic cellular functions both spontaneously and in response to genotoxic stress, alters the transcriptional and epigenetic landscape and dictates the cellular response to stress, the development of a complete panel of isogenic DNA repair deficient cell lines across multiple backgrounds will be a valuable platform for gene and drug discovery, validation of inhibitor specificity and the identification of response biomarkers and novel targets for gene/drug synthetic-lethality combinations.

Ongoing and future projects also include the identification of novel DNA Repair targets for improved chemotherapy response, testing and evaluation of novel DNA repair inhibitors, tumor tissue analysis for defects in expression of enzymes critical for chemotherapy response and evaluating the potential of NAD$^+$ biosynthesis inhibitors in the development and testing of tumor specific, synthetically lethal, chemotherapy combinations.

Laura Stabile, Ph.D.
Research Associate Professor
Ph.D., West Virginia University, 1999

Dr. Stabile is interested in the role of growth factors and hormones in the development of human lung cancer. The hepatocyte growth factor (HGF)/c-Met pathway and the estrogen pathway both play key roles in the development and progression of lung cancer and represent attractive targeted pathways for drug development. Lung cancer kills more Americans every year than any other type of cancer, and the 5-year survival rate is only
Lung cancer patients are typically diagnosed at a late stage and have very few effective therapeutic options. Thus, new targeted strategies are essential to make an impact on this disease.

c-Met is a receptor tyrosine kinase whose activation by HGF can lead to transformation (conversion of a normal cell into a malignant cell) and tumorigenicity (growth of tumors) in a variety of human tissues. Since c-Met and HGF are frequently overexpressed in lung cancer and there is a strong correlation between overexpression and decreased patient survival, the HGF/c-Met signaling pathway is a potential target for tumor control. Primary projects in this area of interest include: 1) studying the development and inhibition of lung carcinogenesis in a novel transgenic mouse model that overexpresses HGF in the airways 2) preclinical development of therapeutic drugs that target this pathway using a variety of techniques such as neutralizing antibodies to HGF, c-Met small molecule inhibitors, c-Met guanidinium-peptide nucleic acid antisense technology and 3) understanding the mechanism of signaling interactions between c-Met and the epidermal growth factor receptor (EGFR) pathway.

Dr. Stabile has successfully developed a murine model that mimics the overproduction of HGF found in human lung tumors and has shown that a single human HGF neutralizing antibody, L2G7, has profound inhibitory effects on development of lung tumors in this transgenic mouse model. Furthermore, lung tumors with K-ras mutation are resistant to blockage of the HGF pathway using L2G7. In addition, we have recently demonstrated the importance of induction of the cyclooxygenase 2 (COX-2)/prostaglandin E2 (PGE2) pathway and subsequent activation of EGFR by HGF in lung cancer cells. Figure 2 describes the signaling pathway of HGF that we are studying and areas of therapeutic intervention. Another aspect of research involves the estrogen pathway in lung cancer. Lung cancer is becoming increasingly common in women and in the U.S. accounts for more female deaths annually than breast cancer and all other gynecological cancers combined. Epidemiological studies show that male-female differences exist in the presentation of lung cancer. These observations suggest the role of estrogens in lung carcinogenesis. Primary projects in this area of interest include: 1) understanding both genomic and non-genomic effects of estrogen in the lung 2) elucidating cross-talk pathways between estrogen and the EGFR and VEGF pathways in the lung 3) understanding the differences in estrogen signaling in lung cancer patients who actively smoked versus those who never smoked and 4) preclinical development of therapeutic drugs that target this pathway such as estrogen antagonists and aromatase inhibitors.

Dr. Stabile has demonstrated that estrogen receptors are expressed in both normal lung as well as lung tumor cells and that estrogen promotes the growth of lung tumor cells. The growth stimulation is significantly inhibited in vitro and in vivo with the pure estrogen receptor antagonist, ICI 182,780 (Faslodex, fulvestrant).

In addition, Dr. Stabile has demonstrated that the estrogen receptor pathway can cross-talk with the EGFR pathway and targeting both pathways simultaneously using clinically relevant agents show enhanced anti-tumor effects compared to targeting either pathway alone. This drug combination is currently being tested in clinical trials. Dr. Stabile is currently interested in elucidating the role of the newly discovered estrogen receptor, GPR30, which is thought to be responsible for some of the non-genomic actions of estrogen.

The overall goal for both areas of interest are to test different mechanisms by which these pathways control other growth-promoting proteins in the lung and test both available and novel drugs as single agent or combination therapies using novel animal models of lung cancer to determine how to inhibit these pathways most effectively. Optimal preclinical drugs will ultimately be translated to patient clinical trials.
The overarching goal of the Straub laboratory is to investigate novel redox-controlled cell signaling mechanisms that regulate endothelial and smooth muscle cell biology and cell-cell communication in the microcirculation. Our investigations focus on two important pathways: 1) the mechanisms by which endothelial cell expressed hemoglobin a regulates nitric oxide signaling in the blood vessel wall and 2) the mechanisms by which the intracellular nicotinamide phosphoribosyltransferase (NAMPT) pathway controls basic redox signaling functions in vascular cells.

**Mechanisms of hemoglobin a-regulated nitric oxide signaling in endothelial cells.**
Nitric oxide (NO) signaling regulates arterial vascular reactivity in the microcirculation to control peripheral vascular resistance and thus blood pressure. Recently, it was reported for the first time that hemoglobin a is expressed in arterial endothelial cells (ECs), where it is in complex with endothelial nitric oxide synthase (eNOS) (Straub et al., *Nature* 2012). It was demonstrated that endothelial hemoglobin a is enriched at the myoendothelial junction, the point where endothelial cells and smooth muscle cells make contact in resistance arteries and arterioles, where it regulates the effect of NO signaling on vascular reactivity. Mechanistically, hemoglobin a heme iron in the Fe$^{3+}$ state permits active NO signaling, and this signaling is shut off when hemoglobin a is reduced to the Fe$^{2+}$ state by endothelial cytochrome B5 reductase 3 (CytB5R3). These data reveal a novel paradigm by which the regulation of intracellular hemoglobin oxidation controls NOS signaling in non-erythroid cells. This paradigm may be relevant to a broad range of other somatic cells discovered to express hemoglobin (i.e. neurons, renal mesangial cells, macrophages, sympathetic nerves, hepatocytes, alveolar epithelial cells, and endometrial cells) and also known to express NOS isoforms. Our studies will be a direct outgrowth of this work, where we will focus on the molecular, cellular, and *in vivo* contribution of somatic hemoglobins and CytB5Rs as it pertains to vascular physiology and disease.

**NAD regulation and the NAMPT pathway in vascular physiology and disease**
Nicotinamide adenine dinucleotide (NAD) is a fundamentally important molecule critical for many redox reactions in biology. Interestingly, the regulation of NAD$^+$ and NAD(H) is dependent on cell type, which is governed by multiple mechanisms. One mechanism that regulates NAD$^+$ levels is NAMPT, also known as pre-B cell colony factor (PBEF) or visfatin. Existing both as an extracellular and intracellular protein, NAMPT is a rate-limiting enzyme in the NAD$^+$ biosynthesis pathway and is vital for embryonic development since homozygous knockout mice are embryonic lethal (Revollo *et al.*, 2007). The extracellular form of NAMPT stimulates both NAD$^+$ and non-NAD$^+$ signaling pathways in vascular cells, while intracellular NAD$^+$ generated by NAMPT has been shown to regulate vascular cell longevity and protection against ischemia/reperfusion in the heart through a surtutin-1 dependent pathway (van der Veer *et al.* 2007, Hsu *et al.*, 2009). Although it has been established that intracellular NAMPT can regulate NAD$^+$ levels, the downstream signaling pathways relying on this enzyme with regards to resistance arterial tone regulation remains elusive. Our goal is to better understand the role(s) of this enzyme in the microcirculation and in vascular biology in general.

**Kristal Tucker, Ph.D.**
*Research Instructor*
*Ph.D., Florida State University, 2010*

Midbrain dopamine neurons exhibit slow intrinsic pacemaker activity. Synaptic input modifies this baseline activity to produce the irregular and bursts of activity that are important for movement and motivated behaviors. Dr. Tucker uses current-, voltage-, and dynamic-clamp electrophysiology in brain slices to investigate the role of different voltage-gated ion channels in these patterned electrical events. Multiphoton live cell imaging and electrophysiology in midbrain slices is then used to visualize the effects of patterned electrical activity and psychiatric drugs on vesicular accumulation and release of intrinsically fluorescent drugs and neurotransmitters.
Ben Van Houten, Ph.D.

Professor

Ph.D., Oak Ridge Graduate School of Biomedical Sciences, University of Tennessee, 1984

Dr. Van Houten’s laboratory studies the formation and repair of DNA damage in nuclear and mitochondrial genomes. We are particularly interested in the structure and function of proteins that mediate nucleotide excision repair and the role of oxidative stress in human disease.

Faulty DNA repair can promote mutations, aging, cancer and cell death. The process by which protein components of repair detect damaged or modified bases within DNA is an important but poorly understood type of protein-DNA interaction. The cell contains a series of pathways designed to protect its DNA from environmental and endogenous damage. One of the most remarkable aspects of nucleotide excision repair (NER) is that it can remove a wide range of DNA lesions that differ in chemistry and structure. During bacterial NER UvrA, UvrB and UvrC proteins work together to identify and remove DNA damage.

The UvrA and UvrB proteins are believed to recognize damage-induced distortion in the DNA helix rather than the lesion per se. However, detailed studies of the kinetics, thermodynamics and structures of the Uvr proteins have been limited due to their instability. To overcome this problem, UvrA, UvrB and UvrC from the thermophilic bacteria Bacillus caldotenax and Thermotoga maritima were recently cloned and overexpressed. The proteins maintain optimal activity at 65°C and are amenable to both structural and biophysical studies. The group is collaborating with Bob London’s NMR group at NIEHS for an analysis of the dynamics of UvrB upon ligand binding using NMR techniques. The group is also collaborating with Caroline Kisker, Ph.D. and her group, in Wurtzburg, Germany, on solving protein and protein-DNA structures by X-ray crystallography. We have recently solved a co-crystal structure of UvrB bound to DNA.

These complexes are being visualized on DNA using atomic force microscopy, performed Hong Wang, Ph.D., and single-molecule techniques using quantum dot labeling, performed by Neil Kad, Ph.D. at the University of Essex. These tools, combined with site-directed mutagenesis and biochemical analyses, allow for structure-function studies of the UvrA, UvrB and UvrC proteins, and form a basis for understanding the fundamental molecular processes of NER. The long-term goal is to have a complete understanding of how structural perturbations induced by specific DNA lesions are detected and removed by the NER machinery at the atomic level. Most recently we have begun to extend our studies to similar proteins found in humans.

Mitochondria represent an important target of reactive oxygen and mitochondrial DNA (mtDNA) appears to be an early and sensitive marker of this stress. Many human diseases are associated with reactive oxygen including cancer, heart disease and neurodegenerative diseases. Mitochondria are essential organelles for generating ATP during oxidative phosphorylation. The mtDNA encodes 13 polypeptides, 11 involved in electron transport and two serving as subunits of ATP synthase. Damage to mtDNA is repaired, but prolonged oxidant treatment results in persistent mtDNA damage, loss of mitochondrial function and apoptosis. These observations suggest that mtDNA damage is important in the toxicity induced by reactive oxygen species (ROS) such as superoxide, hydrogen peroxide and the hydroxyl radical. Our group is testing the hypothesis that ROS generated in the mitochondria results in mtDNA damage causing a vicious cycle of damage: mtDNA damage causes a decrease in transcription and loss of essential mitochondrial proteins, causing an inhibition of electron transport and subsequent release in more ROS. This process causes further mitochondrial decline and many degenerative diseases associated with aging. We have developed a very sensitive DNA damage assay based on quantitative PCR that allows us to examine damage to nuclear and mitochondrial DNA from as little as 100 microliters of human blood. We are currently examining the role of mtDNA damage and repair in several human diseases including cancer and Friedreich’s ataxia.
Jean-Pierre Vilardaga, Ph.D.
Professor
Ph.D., Free University of Brussels, Belgium, 1996

The Vilardaga laboratory is directed at understanding molecular mechanisms of G protein-coupled receptor (GPCR) signaling and trafficking – two key processes in biological signaling in general and, more specifically, in pharmacology and drug research. Adrenergic and peptide receptors, which transmit signals, respectively for small neurotransmitters (such as noradrenaline and dopamine) and larger peptide hormones (vasopressin, parathyroid hormone, parathyroid hormone related peptide), are two well characterized distinct subtypes of GPCRs that serve as useful models for analyzing GPCR mechanisms. The objective of this line of research is to elucidate the general principles of signal transduction from the extracellular ligand binding event to intracellular signaling cascades, which are involved in systems as diverse as neurotransmitter and hormonal signaling.

Optical approaches (e.g. FRET, TIRF microscopy) are used to monitor the activation/deactivation steps along the signaling cascades of GPCRs in live cells. This approach revealed fundamental mechanisms of GPCRs signaling and trafficking in live cells for neurotransmitter and peptide hormones such as the PTH, which were published in 2003-2007 in Nature Biotech, Nature Methods, Nature Chemical Biology, the Journal of Biological Chemistry and PNAS.

Recently Vilardaga laboratory also discovered the new concept that persistent cAMP production mediated by parathyroid hormone receptor endocytosis may mediate potent catabolic signaling actions via PTH (PNAS 2008, Nature Chem Biol 2009). This prolonged cAMP production from intracellular compartments further indicates that the traditional concept that cAMP production triggered by GPCRs originates exclusively at the cell membrane must be revised. The main focus of my current research aims at determining the origin of the prolonged signaling by GPCRs and its termination. These events and consequent signaling patterns are quite novel and important for cellular signaling.

Dario Vitturi, Ph.D.
Research Instructor
Ph.D., University of Alabama at Birmingham, 2010

Dr. Vitturi’s research focuses on the mechanisms by which biological systems harness the generation of free radicals and other reactive species to modulate cell signaling and maintain physiological homeostasis. Dr. Vitturi has been involved with the study of nitrite as a physiologically relevant precursor for nitric oxide (NO) formation that becomes activated under conditions of hypoxia and/or metabolic acidosis via reaction with metalloproteins. Nitrite has been shown to be a promising therapeutic intervention for many conditions associated with uncontrolled inflammation, decreased NO availability and ischemia-reperfusion events. Importantly, NO generation is just one aspect of the biological reactivity of nitrite, with this molecule also acting as a precursor for the formation of secondary species such as nitrated fatty acids, S-nitrosothiols and N-nitrosamines, all of which are capable of mediating specific cellular responses. In this regard, Dr. Vitturi’s current research interests encompass the study of biochemical pathways leading to the formation of these secondary species, the elucidation of their allied biological actions and ultimately the development of clinically viable interventions for their modulation under pathological conditions.

Daniela Volonte, Ph.D.
Research Assistant Professor
Ph.D., University of Milan, Italy, 1996

Tumor development is initiated by a multiplicity of genetic abnormalities. Tumor cells need to escape barriers that limit uncontrolled cell proliferation. One of these barriers is represented by cellular senescence. Cancer cells need to overcome this obstacle to produce a clinically relevant tumor mass. For these reasons, cellular
senescence represents a natural tumor suppressor mechanism. Thus, molecules that regulate cellular senescence are potential therapeutic targets for the treatment of cancer and the fight against aging.

Caveolae are invaginations of the plasma membrane enriched in cholesterol. Caveolin-1, the structural protein component of caveolar membranes, acts as a scaffolding protein to concentrate and functionally regulate signaling molecules.

In recent years, several independent lines of evidence have emerged suggesting that caveolin-1 may function as a "tumor suppressor protein" in mammalian cells. For example, caveolin-1 protein expression has been shown to be absent in several transformed cell lines derived from human mammary carcinomas, including MCF-7. In addition, caveolin-1 mRNA and protein expression are lost or reduced during cell transformation by activated oncogenes, such as v-Abl and H-ras (G12V); caveolae are absent from these cell lines. In addition, the human caveolin-1 gene is localized to a suspected tumor suppressor locus (D7S522; 7q31.1), a known fragile site (FRA7G) that is deleted in many types of cancer.

Oxidative stress is a known inducer of cellular senescence. We have shown that up-regulation of caveolin-1 is required for oxidative stress–induced cellular senescence in fibroblasts. To unravel the molecular mechanisms underlying oxidative stress-induced up-regulation of caveolin-1 in senescent cells, Dr. Volonte has shown that oxidants stimulate the activity of the caveolin-1 promoter reporter gene construct in fibroblasts. She has identified Sp1 binding to two GC-boxes as the core mechanism of oxidative stress–triggered caveolin-1 transactivation. In addition, through signaling studies she has shown p38 mitogen-activated protein kinase (MAPK) as the upstream regulator of Sp1-mediated activation of the caveolin-1 promoter following oxidative stress. For the first time Dr. Volonte has delineated the molecular mechanisms that modulate caveolin-1 gene transcription upon oxidative stress bringing new insights into the redox control of cellular senescence in both normal and cancer cells.

Thus, cellular senescence may represent one of the molecular mechanisms through which caveolin-1 acts as a tumor suppressor protein. Current efforts are aimed at identifying the signaling molecules which link caveolin-1’s function to cellular senescence.

**Nobunao Wakabayashi, Ph.D.**
*Research Assistant Professor*
*Ph.D., Tohoku University Graduate School, 1959*

Loss of cellular homeostasis through exposures to endogenous (e.g., inflammation) and exogenous (e.g., carcinogens) stresses contributes to many diseases including carcinogenesis. The Keap1-Nrf2-ARE signaling pathway evokes an adaptive response to these stresses that serves to enhance cell survival. Through gene expression analyses of the signaling cascade linked to Nrf2, using both *Keap1*- and *Nrf2*-disrupted mice, it appears that multiple signaling pathways intersect with Nrf2 signaling. Our research goals are to elucidate novel signaling crosstalk based on genes bearing functional ARE (Nrf2-sMaf recognition enhancer element) in the promoter of target genes and the underlying mechanistic roles of the Keap1-Nrf2 system in protecting against chronic degenerative diseases *in vivo*. Currently, we are focusing on the role of Nrf2 signaling in tissue repair and regeneration.

**Q. Jane Wang, Ph.D.**
*Associate Professor & Vice Chair, Regulatory Affairs*
*Ph.D., Creighton University, 1995*

Dr. Wang’s lab conducts basic and translational research on oncogenic protein kinases in the area of cancer biology. The main focus is on diacylglycerol (DAG)-targeted protein kinases, particularly protein kinase D (PKD) and protein kinase C (PKC) family kinases. DAG is a key second messenger generated in cells in
response to the activation of receptor tyrosine kinases and G protein-coupled receptors. It acts to disseminate vital signals from the cell surface to the cell interior. PKD and PKC are primary targets of DAG that regulate many fundamental cellular processes such as cell growth, survival, apoptosis, motility, and protein trafficking. In particular, PKD is at the center of the DAG signaling network, integrating signals from both DAG and PKC. In recent years, PKD has emerged as a promising therapeutic target for several diseases including cancer and heart disease. Deregulated PKD expression and activity have been demonstrated in a variety of pathological conditions, suggesting an active contribution to disease initiation and progression.

Dr. Wang’s laboratory seeks to understand both the basic structure of PKD and how different structural domains in PKD interact to regulate its activity and function. We are particularly interested in the C1 domain, the high affinity DAG binding domain shared among all DAG receptors. Our previous studies identified PKD as a high affinity receptor for DAG and defined the structure-activity requirements for the binding of the PKD C1 domain to DAG and phorbol esters. This work has provided the basis for ligand-specific regulation of PKD isoforms. Our long-term goal is to investigate the intra- and intermolecular regulatory mechanisms controlling PKD activity and assess their impact on the function of PKD in intact cells. Ultimately, we seek to solve the 3D structure of this protein.

Growing evidence has implicated PKD in the pathogenesis of cancer. Using prostate cancer as a model, we will determine the functional relevance and signaling mechanisms of PKD in cancer. We will elucidate the signaling pathways both upstream and downstream of PKD and identify novel PKD substrates relevant to cancer promotion. In previous studies, we have identified PKD3 as a downstream target of the oncogenic kinase PKCe. We found that PKD3 acts through a hyperactive PKCe/PKD3 pathway to modulate the activity of ERK1/2 and Akt signaling, contributing to the growth and survival of prostate cancer cells. Another active area of research in our lab is to determine the in vivo relevance of PKD signaling to cancer. We use a variety of animal models including mouse xenograft models and transgenic/knock-out mice to determine the role of PKD in prostate cancer initiation, progression, and metastasis. In addition, we also study the cross-regulation between PKD and androgen receptor signaling and its implication in prostate cancer. Our studies have now expanded to other types of solid tumors such as head and neck cancer and pancreatic cancer. In the long run, we seek to validate PKD as a therapeutic target for cancer therapy.

In this major research endeavor, we seek to develop potent and selective small molecule inhibitors of PKD as pharmacological tools for dissecting PKD-mediated biological processes and as potential drugs for clinical application. This is an important study in the translation of PKD-targeted therapeutics into the clinic. In collaboration with our colleagues at the University of Pittsburgh Drug Discovery Institute and the Department of Chemistry, we have identified the first potent and selective small molecule PKD inhibitor, CID755673, and subsequently developed a class of unique ATP-noncompetitive inhibitors based on this parental compound. These inhibitors are cell-active and exhibit nanomolar potencies, with high specificity for PKD. Future studies are focused on further developing this series of inhibitors for in vivo and clinical application, and continuing our efforts in new drug discovery.

Stacy Gelhaus Wendell, Ph.D.
Research Assistant Professor
Ph.D., University of Maryland Baltimore County, Baltimore, MD, 2005

Nitrate and oxidized metabolites of O-3 and O-6 fatty acids, such as docosahexaenoic acid (DHA), eicosapentaenoic acid (EPA), docosapentaenoic acid (DPA) arachidonic acid (AA) and linoleic acid (LA), are potent signaling mediators involved in inflammation and resolution, amongst a variety of other regulatory pathways. These oxidized metabolites are produced by reactive oxygen and nitrogen species as well as through enzymatic pathways including cyclooxygenase (COX), lipoxygenase (LO), and cytochrome P450s (CYP450). Dr. Wendell’s research is specifically focused on understanding the anti-inflammatory mechanisms of a specific
subgroup of these metabolites; the electrophilic fatty acids. Examples of electrophilic fatty acids include nitrated fatty acids such as nitro-oleic and nitro-linoleic acid or oxidized lipids that contain an α,β-unsaturated ketone or epoxide. Electrophilic fatty acid metabolites can exert their effects through the modulation of transcriptional regulatory proteins. Many transcriptional regulatory proteins contain a nucleophilic amino acid residue, such as a cysteine or histidine, to which the electrophilic moiety of the fatty acid can form a reversible Michael addition. Many of these interactions have been described with the nitrated fatty acids, particularly nitro-oleic acid. In terms of cellular signaling, the Freeman lab has described several protein targets of posttranslational nitroalkylation modification, resulting in activation of pro-MMP9, PPARγ and the antioxidant pathway Keap1/Nrf2 and the inhibition of the pro-inflammatory transcription factor NF-kB.

The current focus of Dr. Wendell’s research is on the mechanism of these electrophilic fatty acids in asthma. Asthma is a complicated disease that much like cancer is comprised of numerous disease states and phenotypes. In many ways it is an umbrella of respiratory diseases that share some similar phenotypes such as airway hyper-responsiveness, increased mucus secretion, increased smooth muscle contraction and decreased airflow. In the most severe of cases, airway remodeling and corticosteroid resistance are not uncommon. It affects over 30 million worldwide and is an economic burden with therapeutic costs upwards of $19 billion dollars per year. While the number of asthmatics in Westernized countries seems to be plateauing, the worldwide number of asthmatics is projected to hit over 100 million by 2025, mostly in low/middle economically developing countries.

Dr. Wendell is looking at the formation of electrophilic fatty acids in transformed stable cell lines while collaborating with clinicians to study the formation and signaling of electrophilic fatty acids in mild to moderate asthmatic subjects. In this study, subjects undergo a baseline bronchoscopy after which they are placed randomly in one of three groups, control, aspirin, or indomethacin. The thought here is that indomethacin will completely inhibit cyclooxygenase activity; therefore, shunting metabolism to other pathways including lipoxygenase or CYP450. Aspirin will inactivate COX-1, but acetylate COX-2 at S516, thus altering enzymatic activity and the stereochemistry of product formation. Following 5 days of treatment, subjects return for a second bronchoscopy. At each bronchoscopy, blood, urine, bronchoalveolar lavage, and bronchial brushings are taken. The epithelial cells from the brushings can be cultured at the air liquid interface for mechanistic studies and identification of key electrophilic fatty acids. To reach these end goals, the lab utilizes molecular biology and analytical techniques, primarily mass spectrometry, (triple quadrupole and ion trap) to elucidate the structures of novel electrophilic species, accurately detect and quantify key electrophilic fatty acid oxidation products in biological matrices, and establish mechanisms of action in asthma and other lung and airway diseases. Furthermore, the implications of electrophilic fatty acid formation and signaling under inflammatory conditions and the ability of electrophilic fatty acids to decrease airway hyperresponsiveness are being investigated in a house dust mite allergen murine model of asthma.

**Steven Wendell, Ph.D.**
*Research Assistant Professor*
*Ph.D., University of Minnesota*

**Steven Woodcock, Ph.D.**
*Research Instructor*
*Ph.D., University of Oregon*

The biological convergence of reactive oxygen species, nitrate/nitrite and unsaturated lipids produce a variety of nitrated and oxidized lipids, which as redox signaling mediators have been observed to have pluripotent activity *in vivo*.

Dr. Woodcock’s research interests include the synthesis of novel nitrated and oxidized lipids, mechanisms of nitration, and the reactivity of nitroalkenes. We use a combination of synthetic and physical organic chemistry
along with biochemistry to help identify and study these species, and to study their production and interactions with a variety of electron-rich biological molecules. These studies support the emerging potential of nitrated lipids as therapeutic agents.

In addition we participate in collaborations with other labs to pair our synthetic interests with aspects of nitrated lipid activity in novel arenas, as well as to provide molecular probes for various redox signaling studies. Recent projects have included isotopically labelled standards, phospholipids, nucleic acid-steroid conjugates, and fluorescent and affinity-conjugates as chemical probes.

Kunhong (Kevin) Xiao, M.D., Ph.D.
Associate Professor
Ph.D., Oklahoma State University, 2003

Dr. Xiao’s research focuses on the molecular mechanisms of GPCR signaling and pharmacology. GPCRs, the largest family of cell surface receptors, are crucial to human health and present a core target of modern medicine. These receptors regulate virtually all known physiological/pathophysiological processes in humans. The clinical significance of GPCRs relies on the fact that these receptors account for about 50% of all prescription drugs, with annual worldwide pharmaceutical sales in excess of $700 billion. In recent years, the discovery of β-arrestin (β-arr)-mediated signaling has led to a paradigm shift in GPCR biology and pharmacology. The newly discovered nature of two parallel pathways (G protein- vs. β-arr-mediated) signaling downstream of GPCRs makes it possible to activate selectively one pathway over another (termed biased agonism or functional selectivity). This concept of biased agonism provides the potential opportunity to develop an entirely new class of drugs—biased ligands, which can activate selectively a specific pathway, and thus trigger only particular effects at the physiological level (e.g., desired therapeutic outcomes and diminished side effects). Therefore, to understand the molecular mechanisms of biased agonism is of great significance.

During the past 10 years, Dr. Xiao elucidated the molecular mechanisms of biased agonism from different perspectives—receptor level regulation, signaling networks, and structural basis (Figure 1). Using cutting-edge and high-throughput MS-based proteomics, in combination with systems, chemical and structural biology, Dr. Xiao studied protein function, macromolecular interaction, and post-translational modifications downstream of β-arrs and GPCRs. His work provided a global view of GPCR signaling and a better understanding of the molecular mechanisms of β-arr biased agonism of GPCRs (Figure 2). The findings from these studies have opened up new avenues for the rational design of more efficient and specific drugs as an approach to individualized medicine.

Dr. Xiao also is interested in delineating the structure-function relations of multi-protein complexes important for GPCR signaling and pharmacology. As our understanding of GPCR signaling evolves from simple, one-way pathways to more complicated networks, investigating protein complexes has become the keystone of GPCR research. One of the most direct ways to flesh out the function of these protein complexes is to study their structure. However, the structural characterization, achieved by standard methods such as X-ray crystallography or NMR of receptor protein complexes poses numerous challenges.

In recent years, Dr. Xiao developed multiple MS-based structural strategies, for example, an orthogonal approach combining hydrogen-deuterium-exchange/MS (HDXMS), cross-linking/MS (CXMS) and disulfide trapping, to reveal information regarding the overall architecture of protein complexes, detailed information of the interfaces, and structural dynamics during complex formation (Figure 3). The information offers new insight into the mechanistic-structural-functional relationships of protein complexes and provides a potential platform for developing novel therapeutic interventions.

Dr. Xiao is actively engaged in the development of new MS-based, high-throughput proteomics and systems biology technologies for therapeutic target identification and biomarker discovery. The ultimate goal of this line
of research is to 1) identify novel molecules and pathways that have the potential to become therapeutic targets for the treatment of diseases; 2) identify novel biomarkers and more sensitive methods for disease early stage detection and diagnosis.

**Li Yang, Ph.D.**  
*Research Instructor*  
*Ph.D., University of Nebraska, 2010*

- Elucidating the mechanism of endogenous estrogen and xenoestrogens metabolism in cancer initiating.
- Developing mass spectrometric analytical methods for identifying and measuring metabolomic profile of small molecules from cell models, animal models and human specimens.
- Discovering potential early biomarkers of cancer.
- Developing chemoprevention strategies by using nutritional antioxidant and phytochemicals to modulate metabolic profile.

**Cheng Zhang, Ph.D.**  
*Assistant Professor*  
*Ph.D., University of Science and Technology of China, Hefei, P.R. China, 2008*

Dr. Zhang’s group focuses on the study of model G protein-coupled receptors (GPCRs) to elucidate the molecular mechanisms of receptor signaling and to advance our understanding of their pharmacology. GPCRs are a family of cell surface receptors with over 700 members. They transduce signals from extracellular signaling molecules, including hormones and neurotransmitters, to intracellular effectors in order to mediate and regulate a broad spectrum of physiological and pathological processes. GPCRs have been heavily investigated in the pharmaceutical industry, and they constitute 30-40% of current drug targets. Yet the mechanistic details of GPCR signal transduction across the cell membrane are largely poorly understood, in part due to the extraordinary complexity of receptor conformational states associated with different ligands and different signaling outputs. His lab is trying to explore the molecular mechanisms underlying the signal transduction of certain GPCRs through combinatorial approaches, including structural biology approaches, spectroscopic tools, and molecular pharmacology approaches.

Current efforts are directed at elucidating the atomic structures of several GPCRs that function in inflammation and calcium metabolism, in complex with their ligands as currently used drugs or potential drug candidates. These structures will reveal the molecular basis of the action of the ligands, as well as the structural elements of receptors that are involved in signal transduction. This information will guide further biophysical and computational studies performed in my lab, or through collaborations, to explore the dynamics and conformational versatility of these receptors and guide structure-based drug design for the development of drugs with improved pharmacological properties. Long-term goals also include the structural characterization of these receptors in complex with their signaling effectors and the characterization of the different pharmacological behaviors of these receptors when coupled with different signaling effectors. The resulting information will greatly advance our understanding of GPCR signaling and GPCR molecular pharmacology, and will be quite valuable for designing new pharmaceuticals used in the treatment and management of inflammatory diseases, bone diseases and cancer.

**Lin Zhang, Ph.D.**  
*Professor*  
*Ph.D., University of Southern California, 1995*

The immediate goal of Dr. Zhang’s research is to understand how anticancer drugs kill cancer cells, and more importantly, why they fail so often. In the long term, we will attempt to use this knowledge to identify novel molecular targets and treatment strategies to improve cancer chemotherapy and chemoprevention.
Cell death in anticancer therapies
Our research program has centered on several molecules that control discrete steps of programmed cell death. The first one, PUMA, is a downstream target of the tumor suppressor p53 and a BH3-only Bcl-2 family protein. PUMA is required for DNA damage-induced and p53-dependent apoptosis, and also plays a key role in apoptosis induced by several targeted anticancer drugs. The second one, SMAC, is a mitochondrial apoptogenic protein and a caspase activator. SMAC helps to execute apoptosis induced by anticancer drugs via a mitochondrial feedback loop. Regulators of non-apoptotic cell death, such as the autophagy inducer Beclin 1 and the necrosis regulator RIPK3, have also been studied. Through analyses of these molecules and their associated protein networks, we try to gain deep understanding on how cell death is initiated and executed in human cancer cells, why some cancer cells are not sensitive to anticancer drugs, and what can be done to restore their sensitivity.

Oncogenic stem cells as the target of cancer chemoprevention
Prevention of human cancer through the use of chemical agents such as non-steroidal anti-inflammatory drugs (NSAIDs) has emerged as a promising strategy to reduce morbidity and mortality of cancer. Our recent studies showed that intestinal stem cells that have acquired oncogenic alterations are targeted by NSAIDs in chemoprevention of colon cancer. We are investigating how NSAIDs trigger apoptosis in such oncogenic stem cells, and if induction of apoptosis is critical for the chemopreventive effects of NSAIDs. We will also determine if apoptosis regulators can be used as markers to predict outcomes of chemoprevention of cancer patients, and if manipulation of apoptosis regulators can be used to improve the chemopreventive effects of NSAIDs.

Manipulation of cell death regulators
To target PUMA, we have developed a high-throughput screening system for identifying small molecules that can activate PUMA in p53-deficient cancer cells. In collaboration with the Pittsburgh Drug Discovery Institute, we will screen compound libraries to identify novel PUMA inducers. We have also identified and characterized small molecules that mimic the functional domains of PUMA and SMAC. Efforts are undertaken to apply these small molecules to chemotherapy and chemoprevention.
Daniel Altschuler, Ph.D.
Associate Professor
External Reviewer
  Argentine Society for Science and Technology
Ad Hoc Reviewer:
  Nature Cell Biology
  Journal of Biological Chemistry
  Proceedings of the National Academy of Sciences USA
  Molecular and Cellular Biology
  Endocrinology
  Molecular Endocrinology
  Molecular Pharmacology
  Journal of Cell Biology
  American Journal of Pathology
  Journal of Cell Science
  The Journal of Pharmacology and Experimental Therapeutics
  Molecular Biology of the Cell

Jonathan Beckel, Ph.D.
Research Instructor
Ad Hoc Reviewer:
  Journal of Urology
  Journal of Neuroscience
  American Journal of Physiology
    Renal Physiology
    Regulatory, Integrative and Comparative Physiology
    Endocrinology and Metabolism
  Neurourology and Urodynamics
  Journal of Neurophysiology
  Journal of Comparative Neurology
  Journal of Physiology
  Faculty of 1000 – Medicine Reports
  Medical Hypotheses
  Biomed Research International

Dr. Alessandro Bisello
Associate Professor
Grant Reviewer:
  AHA Basic Cell, Cell Structure and Survival 4 Committee Member
  AHA Basic Cell, Cell Structure and Survival 2 Committee Member

Donald DeFranco, Ph.D.
Professor
Study Section:
  Biomedical Research and Training (BRT-A) Study Section, NIGMS
Invited Member:
    National Board of Medical Examiners, USMLE Pharmacology and Biochemistry Test Material Development Committee

Editorial Board:
    Steroids
    Nuclear Receptor Signaling

Editor-in-Chief:
    Molecular Endocrinology

Ad Hoc Grant Reviewer:
    ZRG1 EMNR A Special Emphasis Panel, NIH
    American Cancer Society
    March of Dimes Birth Defects Foundation
    National Science Foundation

Ad Hoc Reviewer:
    Molecular Endocrinology
    Molecular and Cellular Biology
    Nature Communication
    Neuroscience Letters
    Nuclear Receptor Signaling
    Oncogene
    Proceedings of the National Academy of Sciences USA
    Steroids

W. Chet de Groat, Ph.D.
Distinguished Professor

Editorial Board:
    Journal of Pharmacology and Experimental Therapeutics
    Journal of the Autonomic Nervous System
    Life Sciences
    Journal of Experimental Pharmacology (honorary)

Editorial Consultant:
    Brain Research
    Journal of Neurophysiology
    Science
    Journal of Urology
    Journal Comparative Neurology
    European Journal of Pharmacology
    Experimental Neurology

Executive Committee, International Society of Autonomic Neuroscience
External Advisory Board, UCLA Center for Neurovisceral Sciences and Women’s Health
Member, Dana Alliance for Brain Initiatives
Faculty, 1000 Biology, Head of Section for the Genito-Urinary and Reproductive Pharmacology
Member, NIH External Treatment Advisory Committee for the Interstitial Cystitis Clinical Trials Group
Member, National Institute of Biomedical Imaging and Bioengineering (NIBIB) Consortium for Addressing Paralysis through Spinal Stimulation Technologies
Member, Vivonda Inc. Medical Advisory Board

Julie Eiseman, Ph.D.
Professor

Grant Reviewer:
    Veteran’s Administration Merit Review (central and local)
Study Section:
    NCI, Cancer Drug Development and Therapeutics (CDDT) Study Section, STIR/SBIR
Reviewer:
    Cancer Chemotherapy and Pharmacology
    Oncology
    Research Communications in Molecular Pathology and Pharmacology
    Clinical Cancer Research
    Journal of Pharmacology and Experimental Therapeutics
    Molecular Pharmacology
    Journal of AOAC International
    Cancer Research
    Journal of Photochemistry and Photobiology
    Molecular Cancer Therapeutics
    Antimicrobial Agents and Chemotherapy
Editorial Board:
    Cancer Chemotherapy and Pharmacology

Peter Friedman, Ph.D.
Professor
Reviewer:
    National Science Foundation, Panel on Cellular and Molecular Biology
    National Science Foundation, Panel on Regulatory Biology
    Endocrine Society, Chair for Pathophysiologic Consequences of Aberrant Heterotrimeric G Protein Regulation
Awards Committee:
    American Physiological Society, Chair, 2005-2007
Study Section:
    National Institutes of Health, Molecular and Cellular Endocrinology Study Section
    National Institutes of Health, NIDDK, GRB-J M5 R24 Study Section
Consultant:
    FDA, novel parathyroid hormone compounds
Invited Reviewer:
    Journal of Biological Chemistry
    Journal of Cell Biology
    Nature Chemical Biology
    Nature Reviews Rheumatology
    PLoS One
    Bone
    Biochemistry
    Journal of Bone and Mineral Research
    American Journal of Physiology
Editorial Board:
    European Journal of Molecular Biology
    Journal of Biological Chemistry
    PLoS One

William Furey, Ph.D.
Professor
Ad Hoc Reviewer:
    Journal of Molecular Biology

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Biochemistry
Nature Structure Biology
Acta Crystallographica
Proceedings of the National Academy of Sciences USA
Proteins
Journal of Biological Chemistry
Protein Science

Grant Reviews:
VA Merit Review Proposals

**Ferruccio Galbiati, Ph.D.**
*Professor*

Editorial Board:
- h Bioscience
- The Journal of Biological Chemistry

Ad Hoc Reviewer:
- FASEB Journal
- Aging Cell
- Cancer Research
- Molecular Cancer Research
- Molecular Pharmacology
- Molecular Endocrinology
- PLoS ONE
- Free Radical Biology & Medicine
- American Journal of Physiology
- Journal of Cell Science
- Journal of Neurochemistry
- Human Genetics
- Molecular Carcinogenesis
- European Journal of Cancer
- Journal of Applied Physiology
- Experimental Cell Research
- Journal of Molecular Medicine
- Proteomics
- Journal of Cellular and Molecular Medicine
- Carcinogenesis
- Cell Research

Ad Hoc Grant Reviewer:
- National Institute of Health, Cellular Mechanisms in Aging and Development (CMAD) Study Section

**Ryan Hartmaier, Ph.D.**
*Research Instructor*

Ad Hoc Reviewer:
- BMC Cancer
- Breast Cancer Research and Treatment
- PLoS One

Grant Reviewer:
- Pennsylvania Breast Cancer Coalition

**Jing Hu, Ph.D.**
*Visiting Associate Professor*
Ad Hoc Reviewer:
Molecular Carcinogenesis
British Journal of Cancer
Experimental Cell Research
Biochemical Pharmacology
Current Drug Metabolism
Journal of Cancer Research and Clinical Oncology
Cellular and Molecular Life Science
Molecular Nutrition and Food Research
Journal of Biological Chemistry
Oncogene
Pharmacology & Therapeutics
Biochemical Pharmacology
Neoplasia

Editorial Board:
American Journal of Cancer Biology

Ad Hoc Grant Reviewer:
Florida Department of Health: James & Esther King Biomedical Research Program and the Bankhead-Coley Cancer Research Program

Study Section:
USAMRMR/CDMRP Study Section, Department of Defense Breast Cancer Research Program

Yi Huang, M.D.
Assistant Professor
Ad Hoc Reviewer:
Amino Acids
Molecular Carcinogenesis
Journal of National Cancer Institute
Breast Cancer Research and Treatment
Breast Cancer Research
BMC Cancer
Breast Cancer Research
BBA – Molecular Cell Research
The Journal of Investigative Dermatology
Cancer Research
Carcinogenesis
Cell Death and Differentiation
Clinical Cancer Research
Clinical & Experimental Metastasis
Cancer Letters
Cell Biochemistry and Biophysics
Frontiers of Epigenomics
Hormones and Cancer
Life Science
Molecular and Cellular Endocrinology
Neoplasia
Oncotarget
Reproductive Biology and Endocrinology
Scientific Report
The Journal of Investigative Dermatology
Translational Oncology

Editorial Board:
Frontiers in Epigenomics
Cancer and Clinical Research

Grant Reviewer:
Israel Science Foundation

Panel Reviewer:
US Army/DOD Breast Cancer Research Program, Molecular Genetics & Biology

Edwin Jackson, Ph.D.
Professor
NIH Study Section, Permanent Member:
Hypertension and Microcirculation

Grant Reviewer:
P30 O'Brien Kidney Center Pilot Projects
NIH Special Emphasis Panel (Chair and Reviewer)

Editorial Board:
Hypertension
Journal of Pharmacology & Experimental Therapeutics
Clinical and Experimental Hypertension
American Journal of Physiology - Renal Physiology
American Journal of Physiology - Regulatory, Integrative and Comparative Physiology
American Journal of Physiology – Heart and Circulatory Physiology
Cardiovascular Therapeutics

Journal Refereeing:
American Journal of Physiology Cell Physiology
American Journal of Physiology Heart and Circulatory Physiology
American Journal of Physiology Regulatory, Integrative and Comparative Physiology
American Journal of Physiology Renal Physiology
Biochemical Pharmacology
British Journal of Pharmacology
Cardiovascular Research
Circulation
Circulation Research
Clinical Cancer Research
Clinical and Experimental Hypertension
Clinical Pharmacology and Therapeutics
Hypertension
Journal of Cardiovascular Pharmacology
Journal of Clinical Investigation
Journal of Pharmacology and Experimental Therapeutics
Kidney International
Molecular Pharmacology
Nephrology Dialysis Transplantation
PLOS ONE
PNAS
Tija Jacob, Ph.D.
Assistant Professor
Journal Refereeing:
   Neuroscience
   Molecular Neurobiology
Editorial Board:
   Frontiers in Molecular Neuroscience (Review Editor)

Yu Jiang, Ph.D.
Associate Professor
Study Section:
   NIH NCI-Transition to Independence (K Grant), ad hoc reviewer
Editorial Board:
   World Journal of Biological Chemistry
Ad Hoc Reviewer:
   Annals of the Rheumatic Diseases
   Autophagy
   BBA
   Breast Cancer Research and Treatment
   Cancer Microenvironment
   Cancer Research
   Current Molecular Pharmacology
   Drug Discovery Today
   EMBO Journal
   FEBS Letter
   FEM Yeast
   Gene & Development
   Journal of Biological Chemistry
   Journal of Biological Sciences
   Molecular Biology of Cell
   Molecular Biology Reports
   Molecular Carcinogenesis
   Molecular Cell Biology
   Molecular Endocrinology
   Molecular Microbiology
   Molecular Pharmacology
   Nutrition Research
   Oncogene
   Oncotarget
   PNAS
   Reproductive Sciences
   Science
   Structure
   Yeast
Ad Hoc Grant Review:
   Medical Research Council, England
   National Science Centre, Poland
   Department of Defense, Tuberous Sclerosis Complex Research Program Review Panel
Thomas Kensler, Ph.D.

Professor

Study Section:
- NCCAM/NIH Special Emphasis Panel ZAT1 SM (25) – Mechanistic Research on Natural Products
- Chair, ZRG1 OTC-C (02) Special Emphasis Panel (NIH)
- Chair, ZRG1 OTC-Y 04 M Special Emphasis Panel (NIH)
- ZCA1 SRB (J1) Special Emphasis Panel (NIH)

Ad Hoc Member:
- Chemo-Dietary Prevention Study Section (NIH)

Editorial Board:
- Reviews in Mutation Research
- Carcinogenesis
- Journal of Biological Chemistry
- Chemical Research in Toxicology
- Molecular and Cellular Biology

Senior Editor:
- Cancer Prevention Research

Committee Memberships:
- Society of Toxicology, Disease Prevention Task Force; Chair [2010-2012]

Ad Hoc Reviewer:
- Analytical Biochemistry
- Applied Occupational & Environmental Hygiene
- Archives of Biochemistry and Biophysics
- Biochemical Journal
- Biochemical Pharmacology
- Biochemistry
- Cancer Biochemistry Biophysics
- Cancer Detection and Prevention
- Cancer Epidemiology, Biomarkers & Prevention
- Cancer Prevention Research
- Cancer Research
- Carcinogenesis
- ChemBioChem
- Chemico-Biological Interactions
- Chemical Biology & Drug Design
- Chemical Research in Toxicology
- Drug Metabolism and Disposition
- Environmental Health Perspectives
- Environmental & Molecular Mutagenesis
- Environmental Research
- Experimental Cell Research
- FASEB Journal
- Free Radical Biology & Medicine
- Free Radical Research
- Gene
- Gut
- Hepatology
- International Journal of Cancer

In Vitro Toxicology
- Journal of Agricultural & Food Chemistry
- Journal of Biological Chemistry
- Journal of Cellular Biochemistry
- Journal of Clinical Investigation
- Journal of Inorganic Biochemistry
- Journal of Mass Spectrometry
- Journal of Medicinal Chemistry
- Journal of the National Cancer Institute
- Journal of Pharmacy and Pharmacology
- Laboratory Animal Sciences
- Molecular Carcinogenesis
- Molecular Cellular Biology
- Molecular Pharmacology
- Mutation Research
- Nutrition and Cancer
- Oncogene
- Proc. National Academy of Science USA
- Proc. Soc. Experimental Biology and Medicine
- Risk Analysis
- Science
- Toxicology
- Toxicology Letters
- Trends in Molecular Medicine
Nicholas Khoo, Ph.D.
Research Assistant Professor
Ad Hoc Reviewer:
  Antioxidants and Redox Signaling
  Biochemical Journal
  Disease Models & Mechanisms
  Journal of Biological Chemistry
  Molecular Nutrition & Food Research
  Diabetes
  British Journal of Pharmacology
  Free Radical Biology and Medicine
  PLOS ONE
  Nitric Oxide
  Brain Research Bulletin
  Vascular Pharmacology

Joan M. Lakoski, Ph.D.
Professor
Study Section:
  Neuroendocrinology, Neuroimmunology and Behavior
Editorial Board:
  American Journal of Translational Research
Committee Memberships:
  Travel Awards Selection Committee, National Postdoctoral Association
  Nomination Awards Committee, National Postdoctoral Association
  Program Committee, Annual Meeting, National Postdoctoral Association
  President’s Council, Society for Executive Leadership in Academic Medicine (SELAM) International
  Board Development Committee, Annual Meeting, National Postdoctoral Association
Ad Hoc Reviewer:
  Neuroscience Letters
  Journal of Pharmacology and Experimental Therapeutics
  Endocrinology
  Journal of Neuroscience Research
  International Journal of Developmental Neuroscience
  Molecular Endocrinology
  Synapse
  Psychopharmacology
  Brain Research Bulletin
  Neuroscience
  Life Sciences Journal
  Neuropsychopharmacology
  Gastroenterology
  Environmental Research
  Pharmacology, Biochemistry and Behavior
  Journal of Neuroscience
  Journal of Gerontology: Biological Sciences
  Academic Medicine
  Society for Clinical and Translational Science
  Cell Biology Education – Life Sciences Education
Jack Lancaster, Ph.D.
*Visiting Professor*

Ad Hoc Reviewer:
- Alcoholism: Clinical and Experimental Research
- American Journal of Physiology
- American Journal of Respiratory Cell and Molecular Physiology
- American Journal of Physiology
- Analytical Biochemistry
- Annals of Biomedical Engineering
- Antioxidants and Redox Signaling
- Archives of Biochemistry and Biophysics
- Biochemical Pharmacology Biochemistry
- Biochimica et Biophysica Acta
- Chemical and Engineering News
- Chemical Research in Toxicology
- Diabetes
- European Journal of Biochemistry
- FEBS Letters
- Free Radical Biology & Medicine
- Hepatology
- Hypertension
- Journal of Biological Chemistry
- Journal of Clinical Investigation
- Journal of Electroanalytical Chemistry
- Journal of Experimental Medicine
- Journal of the American Chemical Society
- Life Sciences
- Nature Neuroscience
- Nitric Oxide Biology and Chemistry
- Proceedings of the National Academy of Sciences
- Science

Grant Reviewer:
- National Institutes of Health
- National Science Foundation
- Department of Energy
- US Department of Agriculture
- American Heart Association

Adrian Lee, Ph.D.
*Professor*

Peer Review:
- Alternate Chair and Member, Career Catalyst Award Review, Susan G. Komen for the Cure
- Ad Hoc Member, CDMRP Integration Panel
- Member, CDMRP Breakthrough Proposal Review
- Member, MUSC Pilot Project Review
- Ad Hoc Member, SEP:ZCA SRB-C (J1) & (J2), NCI
- Ad Hoc Member, Komen Tissue Bank
- Permanent Member, NIH/NCI, Molecular Oncogenesis (MONC)

Advisory Board:
- Scientific Advisory Committee, Susan G. Komen for the Cure
- Boehringer Ingelheim, anti-IGF inhibitors
Editorial Boards:
- NPJ Breast Cancer
- Corresponding Editor, Hormone Molecular Biology and Clinical Investigation
- Member, Editorial Board, Hormones and Cancer
- Frontiers in Endocrinology
- Editor, Endocrinology

Ad Hoc Reviewer:
- Breast Cancer Research and Treatment
- Cancer Research
- Cancer Chemotherapy and Pharmacology
- European Journal of Cancer
- Cancer Investigation
- Molecular Endocrinology
- Cell Growth and Differentiation
- International Journal of Cancer
- Cancer Immunology and Immunotherapy
- Oncogene
- Journal of Neurobiology
- Cancer
- Breast Cancer Research
- Journal of Cellular Biochemistry
- Cancer Epidemiology Biomarkers & Prevention
- Biotechniques
- Molecular Cancer Therapeutics
- Endocrinology
- Trends in Biochemical Sciences
- Molecular and Cellular Biology
- Biochemistry
- Growth Hormone & IGF Research
- Journal of the National Cancer Institute
- Endocrine-Related Cancer
- Biomedical Microdevices
- Nature Clinical Practice Oncology
- Nature Reviews Cancer

Ad Hoc Grant Reviewer:
- VA Merit Award
- Breast Cancer Campaign, United Kingdom
- Associazione Italiana per la Ricerca sul Cancro (AICR)
- Health Research Board’s Clinical Research Training Fellowships, Ireland
- University of Texas, M.D. Anderson Smithville, CRED Pilot Project Review
- Cancer Research Ireland
- Hannah Research Institute, Scotland
- Icelandic Research Fund
- Komen Tissue Bank
- Prevent Cancer Foundation

Edwin Levitan, Ph.D.
Professor
Editorial Board:
- Molecular Endocrinology
Study Section:
   NIH NTRC Study Section

Carola Neumann, M.D.
Visiting Associate Professor
Review Editor:
   Frontiers in Molecular and Cellular Oncology
Editorial Board:
   Molecular & Cellular Oncology
Ad Hoc Reviewer:
   Biochemistry
   Endocrine-Related Research
   Journal of Pharmacology and Experimental Therapeutics
   Leukemia and Lymphoma
   Mitochondrial DNA
   Molecular Carcinogenesis
   Plos One
   Cancer Research
   Current Opinion in Pharmacology
   Journal of Cellular Biology
   Journal of Cellular Biochemistry
   Breast Cancer Research and Treatment
   Endocrinology
   Oncogene
   EMBO Reports

Steffi Oesterreich, Ph.D.
Professor
Editorial Boards:
   Endocrine-Related Cancer
   Frontiers in Epigenomics
   Hormones and Cancer
   GebFra
   Breast Cancer Research (Associate Editor)
Study Section:
   Permanent Member, NIH TCB Study Section
   Chair, NIH Tumor Cell Biology Study Section
Programmatic Reviewer:
   USAMRMC Breast Cancer Research Program Integration Panel
Ad Hoc Reviewer:
   Breast Cancer Research and Treatment
   Cancer Research
   Molecular Endocrinology
   Oncogene
   Cancer
   Breast Cancer Research
   Molecular Cancer Therapeutics
   Molecular and Cellular Biology
   Cancer Letters
   Carcinogenesis
   Clinical Chemistry
Experimental Cell Research
FEBS Letters
Genomics
Journal of Biological Chemistry
Journal of Clinical Investigation
Journal of Molecular Endocrinology
Journal of Molecular Medicine
Journal of the National Cancer Institute
Molecular Carcinogenesis
Molecular Medicine
New England Journal of Medicine
Nucleic Acid Research
NURSA
The Journal of Clinical Endocrinology & Metabolism
Nature Medicine

Grant Reviews:
The Breast Cancer Campaign, London, UK
The Dutch Cancer Society
Health Research Board Clinical Research Training Fellowship, Ireland
Science Foundation, Ireland
Association for International Cancer Research (AICR)
Plan Cancer, Inserm, France
Cancer Research UK, Cambridge Institute
Scientific Commission, F.R.S.-F.N.R.S., Belgium

Roderick O’Sullivan, Ph.D.
Assistant Professor
Ad Hoc Journal Reviewing:
Genome Research

Patrick Pagano, Ph.D.
Professor
Associate Editor:
Clinical Science
Editorial Board:
American Journal of Physiology
Cardiovascular Research
Free Radical Biology & Medicine
Clinical Science
Circulation Research

Study Section:
Permanent Member, NIH/NHLBI Study Section HM
RFA Special Emphasis Panel, Cellular and Molecular Mechanisms of Arterial Stiffening and its Relationship to Development of Hypertension
K99/R00 Study Section Emphasis Panel ZGM1 TWD-A

Ad Hoc Reviewer:
American Heart Association

Ad Hoc Journal Reviewer:
American Journal of Respiratory and Critical Care Medicine
Circulation Research
Antioxidants & Redox Signaling
Arteriosclerosis, Thrombosis & Vascular Biology
Circulation
Hypertension
Journal of the American Heart Association
Journal of Biological Chemistry
Journal of Clinical Investigation
Journal of Respiratory Cell & Molecular Biology
Journal of Physiology
Journal of Molecular and Cellular Cardiology
Journal of Innate Immunity
Kidney International
Nature Reviews
PloS One
Science Signaling
Society for Free Radical Biology and Medicine (abstract reviewer)
Toxicological Sciences

Michael Palladino, Ph.D.
Professor
Editorial Board:
    Neurobiology of Diseases
    ISRN
Guest Associate Editor:
    PloS Genetics
Ad Hoc Reviewer:
    Genetics
    FASEB
    RNA
    IUBMB Life
    Journal of Neuroscience
    Human Molecular Genetics
    Cell Metabolism
    Molecular Cell
    Proceedings of the National Academy of Sciences USA
    Annals of the New York Academy of Sciences
    Cell Death and Differentiation
    BBA-Molecular Basis of Disease
    PLoS ONE
    PLoS Biology
    Neurobiology of Disease
    Pharmacologic Reviews
    GENE
    Genes, Brain and Behavior
    Brain Research Bulletin
    Toxicological & Environmental Chemistry
    Frontiers in Genetics
    Journal of Cell Science
    Current Biology
    Orphanet Journal of Rare Diseases
    Experimental Neurology
    Nature Communications

52
Grant Reviews:
  United Mitochondrial Disease Foundation
  Chronic Fatigue Initiative
  Friedreich’s Ataxia Research Alliance
  Oregon Alzheimer’s Research Partnership

Study Section:
  NIH Neurogenetics (NMG) Study Section
  NIH NOMD Ad Hoc Panel Member

Ad Hoc Grant Reviewer:
  Wellcome Trust
  PEIR Grants

Wei Qian, Ph.D.
Research Instructor

Journal Reviews:
  PLOS One
  ACS Medicinal Chemistry Letters
  International Journal of Molecular Sciences
  BMC Cancer
  Annals of the New York Academy of Sciences
  Drug Design, Development and Therapy
  Current Cancer Therapy Reviews
  International Journal of Nanomedicine

Guillermo Romero, Ph.D.
Associate Professor

Editorial Board:
  Frontiers in Endocrinology

Ad Hoc Reviewer:
  Journal of Biological Chemistry
  Proceedings of the National Academy of Sciences
  Molecular Endocrinology
  Endocrinology
  Molecular Pharmacology
  Journal of Lipid Research
  Biochemistry
  American Journal of Physiology
  Journal of Pharmacology and Therapeutics
  Journal of Cell Biology
  Molecular Biology of the Cell

Ad Hoc Grant Reviewer:
  National Science Foundation
  Veterans Administration
  NIDDK
  American Cancer Society
  Israeli Science Foundation
  Instituto de Investigaciones Cientificas (Spain)

James Roppolo, Ph.D.
Research Assistant Professor

Ad Hoc Reviewer:
Francisco Schopfer, Ph.D.  
*Research Associate Professor*  
Grant Reviewer:  
- American Heart Association, Lipid Peer Review Study Group  
- ANII, Uruguay National Agency for Research and Innovation  
Study Section:  
- Ad hoc NIH review panel NIH/NIGMS (P01)  
Editorial Board:  
- Plos One  
Ad Hoc Reviewer:  
- Free Radical Biology and Medicine Journal  
- British Journal of Pharmacology  
- Journal Lipid Research  
- ACS Medicinal Chemistry  
- ACS Chemical Biology  
- American Journal of Clinical Nutrition  
- Journal of Physiology  
- Journal Leukocyte Biology  
- Biochimica et Biophysica Acta  
- Journal of Neuroscience Research  
- Plos One  
- Archives Biochemistry and Biophysics  
- Mediators of Inflammation  
- International Immunopharmacology  
- Life Sciences  
- Free Radical Research  

Sruti Shiva, Ph.D.  
*Associate Professor*  
Ad Hoc Reviewer:  
- Free Radical Biology and Medicine  
- Mitochondria  
- Circulation Reserach  
- Journal of Biological Chemistry  
- Antioxidant and Redox Signaling  
- Proceedings of the National Academy of Sciences  
- British Journal of Pharmacology  
- Nitric Oxide Biology and Medicine  
- American Journal of Physiology: Lung Cellular and Molecular Biology  
- Biochemical Journal  
- Cell Metabolism  
- Nature Protocols  
- Journal of Molecular and Cellular Cardiology  
- Journal of Lipid Research  
- FEBS Journal  
- Frontiers: Physiology  
- Redox Biology
Grant Reviewer:
  American Heart Association Review Committee Membranes and Subcellular Organelles

Study Section:
  Ad Hoc Reviewer, Myocardial Ischemia Metabolism NIH/MHLBI Study Section

Editorial Positions:
  Editorial Board Member, Redox Biology
  Editorial Board Member, British Journal of Pharmacology
  Associate Editor, Frontiers: Physiology

Matthew Sikora, Ph.D.

Research Instructor
Ad Hoc Reviewer:
  Breast Cancer Research
  Breast Cancer Research and Treatment
  Oncogene
  International Journal of Cancer
  Endocrine-Related Cancer

Shivendra Singh, Ph.D.

Professor
Study Section Member:
  Landon Foundation-AACR Innovator Award for Cancer Prevention Research
  Chemo/Dietary Prevention Study Section
  Department of Defense, Breast Cancer Research Program
  Ad Hoc Member, Special Emphasis Panel, ZRG1, OTC-Y
  Ad Hoc Member, ZRG OTC-Y, Small Business, SBIR/STIR Review
  Reviewer, Florida Department of Health
  Patterns of Interaction in Peer Review Meetings, UW-Madison, IRB-SBS

Editorial Board:
  Molecular Pharmacology
  Molecular Cancer Therapeutics
  International Journals of Cancer Studies & Research
  Open Access Pharmacology Journal
  Cancer Prevention Research
  Pharmaceutical Research
  Journal of Cancer Prevention
  Current Pharmacology Reports

Associate Editor:
  Molecular Carcinogenesis

Ad Hoc Reviewer:
  Cancer Research
  Carcinogenesis
  Prostate
  Molecular Cancer Therapeutics
  Urology
  Clinical Cancer Research

External Advisory:
  Head and Neck SPORE, Emory University, Atlanta, GA
Robert Sobol, Ph.D.
Associate Professor
Editorial Board:
   DNA Repair
   Journal of Carcinogenesis and Mutagenesis
   Mutation Research – Fundamental and Molecular Mechanisms of Mutagenesis
   PLoS ONE
Editorial Advisory Board:
   The Open Toxicology Journal
Associate Editorial Board:
   American Journal of Cancer Research
Invited Editor:
   Proceedings of the National Academy of Sciences of the USA
Ad Hoc Reviewer:
   American Journal of Biotechnology
   Analytical Biochemistry
   BBA
   Biochemistry
   Biology of Reproduction
   Brain Pathology
   Cancer
   Cancer Cell
   Cancer Chemotherapy & Pharmacology
   Cancer Research
   Carcinogenesis
   Cell Biology & Toxicology
   Cell Cycle
   Chemical Research in Toxicology
   Chemistry & Biology
   Clinical Cancer Research
   Digestive Diseases and Sciences
   DNA & Cell Biology
   DNA Repair
   EMBO J
   EMBO Reports
   Environmental and Molecular Mutagenesis
   Environmental and Toxicology & Pharmacology
   Expert Opinion on Investigational Drugs
   FASEB J
   FEBS Letters
   Gene Therapy TIBS
Ad Hoc Grant Reviewer:
   NIH Study Section, ZRG1
   NIH Study Section, Somatosensory and Chemosensory Systems

Laura Stabile, Ph.D.
Research Associate Professor
Editorial Board:
   Cancer and Clinical Research
Ad Hoc Reviewer:
  Cancer Chemotherapy and Pharmacology
  American Journal of Physiology- Lung Cellular and Molecular Physiology
  Steroids
  Molecular Carcinogenesis
  Cancer Biomarkers
  Lung Cancer
  Cancer Research
  Clinical Cancer Research
  Head and Neck
  Evidence-Based Complimentary and Alternative Medicine
  Molecular Endocrinology
  Journal of Clinical Oncology
  Oral Oncology
  Journal of Translational Medicine
  Tumor Biology
  Anti-Cancer Agents in Medicinal Chemistry

Grant Reviewer:
  Kamen Breast Cancer Foundation Grants
  Ireland Health Research Board
  Study Section Panel Member, Susan G. Komen for the Cure Postdoctoral Fellow Grants, Tumor Progression Panel
  Study Section Panel Member, Susan G. Komen for the Cure Postdoctoral Fellow Grants, Diagnostic and Therapeutic Target Panel, FAMRI Grants

Adam Straub, Ph.D.
Assistant Professor

Editorial Board:
  Microcirculation

Ad Hoc Reviewer:
  American Journal of Physiology Lung and Cellular and Molecular Physiology
  Cell Calcium
  Microcirculation
  PLOS One
  Free Radical Biology and Medicine
  American Journal of Pathology
  Journal of Pharmacology and Experimental Therapeutics

Grant Reviewer:
  Member, Blood Pressure Regulation 2, American Heart Association
  Ad Hoc Member, Medical Research Council, UK

Ben Van Houten, Ph.D.
Professor

Ad Hoc Reviewer:
  Cell Metabolism
  Chem. Res. Tox. Chemical Biology
  Chemical Biology
  Current Biology
  Free Radical Biology and Medicine
  Molecular and Cell Biology
  Molecular Cell
Molecular Pharmacology
Nucleic Acids Research
Oncogene
PLoS One
PLos Genetics
Proceedings National Academy of Sciences
Nature Reviews
Science Signaling
Science
Tox Sci

Editorial Board:
  DNA Repair (Associate Editor)
  Molecular Carcinogenesis (Associate Editor)

Committees:
  NINDS, Scientific Advisor for Specialized Neuroscience Research Program

Advisory Board:
  Graduate School of Biomedical Sciences, Wurzburg University, Wurzburg, Germany
  Winship Cancer Institute of Emory University

Grant Reviewer:
  United Mitochondrial Disease Foundation
  NIH, Cancer Etiology (Chair)
  Netherlands Organization for Health Research and Development
  Department of Defense: CDMRP PRMRP PRE-FXS
  National Centre for the Replacement, Refinement and Reduction of Animals in Research Fellowship Proposal, United Kingdom

Jean-Pierre Vilardaga, Ph.D.
Professor

Ad Hoc Reviewer:
  Nature Chemical Biology
  Journal of Biological Chemistry
  Nature
  Proceedings of the National Academy of Sciences
  American Journal of Physiology
  Molecular Endocrinology
  EMBO Report
  Biochim Biophys Acta
  Science Signaling
  Endocrinology
  FASEB Journal

Reviewer:
  NIH: Cell Biology Integrated Review Group
  European Research Council: Molecular and Structural Biology and Biochemistry Review Group

Consulting:
  Medicine, Massachusetts General Hospital

Board Membership
  Chair, IUPHAR for PTH Receptors
Dario Vitturi, Ph.D.
Research Instructor
Ad Hoc Reviewer:
  - Nitric Oxide: Biology and Chemistry
  - American Journal of Respiratory Cell and Molecular Biology
  - Prostaglandins and Other Lipid Mediators
  - Chemical Research in Toxicology
  - Oxidative Medicine and Cellular Longevity
  - Nutrients

Daniela Volonte, Ph.D.
Research Assistant Professor
Editorial Board:
  - H Bioscience

Q. Jane Wang, Ph.D.
Associate Professor
Editorial Board:
  - PLoS ONE
  - International Journal of Clinical and Experimental Medicine
Academic Editor:
  - PLoS ONE
Journal Reviewer:
  - Molecular Cancer Research
  - Oncotarget
Grant Reviewer:
  - Department of Defense, Ovarian Cancer Research Program (OCRP), Clinical Translational Research (CTR) Panel
Study Section:
  - NIH/CSR, ZRG1 BMCT-C Study Section

Stacy Gelhaus Wendell, Ph.D.
Research Assistant Professor
Ad Hoc Reviewer:
  - Nutrients
  - Chemical Research in Toxicology
  - Analytical Chemistry
  - Prostaglandins, Leukotrienes and Essential Fatty Acids
  - Chemico-Biological Interactions
  - Journal of Clinical Investigation

Steven Woodcock, Ph.D.
Research Instructor
Ad Hoc Reviewer:
  - Synthetic Communications
  - Studies in Natural Products Chemistry

Kevin (Kunhong) Xiao, M.D., Ph.D.
Visiting Associate Professor
Journal Reviewer:
  - Journal of Proteome Research
Tumor Biology

Grant Reviewer:
Medical Research Council, United Kingdom

**Cheng Zhang, Ph.D.**
*Assistant Professor*

Ad Hoc Reviewer:
- Cell and Biology International
- Molecular Biosystem
- Molecular and Cellular Biochemistry

**Lin Zhang, Ph.D.**
*Professor*

Editorial Board:
- Scientific Reports
- Oncology Research
- Current Cancer Drug Targets

Associate Editor:
- Molecular Carcinogenesis
- Genes and Diseases

Ad Hoc Reviewer:
- BBA Review Cancer
- Carcinogenesis
- Cell Cycle
- Current Cancer Drug Targets
- Free Radical Biology and Medicine
- Oncotarget
- Scientific Reports

Study Section Review Panel Membership:
- Standing Member, NIH ONC Drug Discovery and Molecular Pharmacology
- National Cancer Institute, Division of Cancer Prevention PREVENT Cancer Program’s Special Emphasis Panel
- ZCA1 RPRB-B (A2) S NCI Special Emphasis Panel: Early Detection Research Network

Review Panels:
- The Health Research Board, Ireland
- Israel Science Foundation, Israel
# FY15 Extramural Sponsored Project Funding

<table>
<thead>
<tr>
<th>Program Description</th>
<th>Funding (in $)</th>
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<tbody>
<tr>
<td>Army Grants</td>
<td>743,143</td>
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<tr>
<td>Foundations, Societies and Associations Funding</td>
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<td>Industry Grants/Contract</td>
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<td>NIH Center Grants</td>
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<td>NIH Contracts</td>
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<td>NIH Developmental Grants</td>
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<td>NIH Program Project Awards</td>
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<td>NIH Research Awards</td>
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<td>NIH Training Grants</td>
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<td>Fellowships</td>
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<td>Veterans Administration and Other Government Awards</td>
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<td><strong>Total Extramural Funding</strong></td>
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# FY15 National Institutes of Health Funding

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<thead>
<tr>
<th>Institute Name</th>
<th>Funding (in $)</th>
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<tbody>
<tr>
<td>National Cancer Institute (NCI)</td>
<td>4,692,888</td>
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<tr>
<td>National Heart, Lung and Blood Institute (NHLBI)</td>
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<tr>
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<td>National Institute of General Medical Sciences (NIGMS)</td>
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<tr>
<td>National Institute of Neurological Disorders and Stroke (NINDS)</td>
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<tr>
<td>National Institute of Environmental Health Science (NIEHS)</td>
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<td>National Center for Complementary and Alternative Medicine (NCCAM)</td>
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<td>Other NIH</td>
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<tr>
<td><strong>Total National Institute of Health Funding</strong></td>
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</tbody>
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DEPARTMENT OF PHARMACOLOGY AND CHEMICAL BIOLOGY
FY15 FUNDING BY NIH INSTITUTE

National Cancer Institute (NCI) 34.0%
National Heart, Lung and Blood Institute (NHLBI) 23.1%
National Institute on Diabetes and Digestive and Kidney Diseases (NIDDK) 22.1%
National Institute of General Medical Sciences (NIGMS) 8.3%
National Institute of Neurological Disorders and Stroke (NINDS) 4.7%
National Institute of Environmental Health Science (NIEHS) 3.7%
National Center for Complementary and Alternative Medicine (NCCAM) 2.6%
Other NIH 1.3%
DEPARTMENT OF PHARMACOLOGY AND CHEMICAL BIOLOGY
FY15 SPONSORED PROJECT FUNDING

NIH Research Awards
63.6%

NIH Program Project Awards
4.5%

NIH Developmental Grants
5.9%

NIH Center Grants
6.2%

NIH Contracts
3.1%

Industry Grants/Contract
1.0%

Foundations, Societies and Associations Funding
8.4%

Army Grants
4.6%

NIH Training Awards
1.1%

Veterans Administration and Other Government Awards
1.4%

Fellowships
0.3%
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<th>LAST NAME</th>
<th>GRANT NUMBER</th>
<th>AGENCY</th>
<th>TITLE</th>
<th>BEGIN DATE</th>
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<th>ANNUAL F&amp;A COST</th>
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<td>ALTSCHULER</td>
<td>R03TW009001</td>
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<td>ANDERSON</td>
<td>F31 CA186367-01</td>
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<td>Role of Estrogen Receptor-Alpha in Ovarian Cancer</td>
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<td>IGF1</td>
<td>Susan Komen Cancer Fund</td>
<td>IGF1 Regulated MIRNAs in Breast Cancer</td>
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<td>CSANYI</td>
<td>K99HL114648</td>
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<td>A Novel Role of Macrophage TSP1-CD47 Signaling in Atherosclerosis</td>
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<td>An Implantable Neuroprosthetic Device to Normalize Bladder Function</td>
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<td>Use of Optical Mapping to Evaluate Mechanisms and New Therapies for</td>
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<td>DE GROAT</td>
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<td>Central sites of action for bladder neuromodulation</td>
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<td>DE GROAT</td>
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<td>DEFRANCO</td>
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<td>DEFRANCO</td>
<td>P01AI106684-01A1</td>
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<td>Immune Airway-Epithelial Interactions in Steroid-Refractory</td>
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<td>DEFRANCO</td>
<td>Nazarbayev University (NU)</td>
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<td>Partnership to Develop Nazarbayev University School of Medicine</td>
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## SPONSORED PROJECT FUNDING FOR FY15 (07/01/14 - 06/30/15)

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### Participants in research

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<td>Samantha Cavolo</td>
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<td>Shannon Granahan</td>
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Major Collaborations

Bruce Freeman, Ph.D.
Professor and Chair

Fadi Lakkis and Timothy Billiar (University of Pittsburgh): Organ preservation for transplantation
Mitchell Fink and Derek Angus (University of Pittsburgh): Anti-inflammatory strategies for treating sepsis/ARDS
Robert Squires and David Hackam (University of Pittsburgh): Anti-inflammatory strategies for treating GI surgical patients
Mark Gladwin (University of Pittsburgh): Treatment of pulmonary hypertension

Daniel Altschuler, Ph.D.
Associate Professor

Matthias Buck (Cleveland, OH): dynamic NMR studies to address conformational changes on Rap1b upon phosphorylation
James Inglese (NIH): collaborative agreement U01 funded for a qHTS assay development
Yuri Nikiforov (University of Pittsburgh, Department of Pathology): development of an animal model for a new ALK fusion found in thyroid cancer
Carlos Camacho (University of Pittsburgh, Department of Computational Biology): MD simulations on the Epac-radixin interaction
Mikael Nilsson (University of Gothenburg, Sweden): thyroid embryology and stem cells
Martin Edreira (Buenos Aires): role of Epac1 in T. cruzi invasion

Dr. Alessandro Bisello
Associate Professor

Dale Mierke (Dartmouth): interaction between EBP50 and Skp2
Dean Madden (Dartmouth) and Zachary Bell (Rice): design, synthesis and characterization of metallo-peptide inhibitors of EBP50

Donald DeFranco, Ph.D.
Professor

Dr. Gordon Rintoul (Simon Fraser University Vancouver, Canada)
Dr. Selma Witchel (Division of Pediatric Endocrinology, Children’s Hospital of Pittsburgh)
Dr. Elias Aizenman (Department of Neurobiology, University of Pittsburgh)
Dr. Robert Bowser (Department of Pathology, University of Pittsburgh)
Dr. Charlene Chu (Department of Pathology, University of Pittsburgh)
Dr. Wen Xie (Department of Pharmaceutical Sciences, University of Pittsburgh)
Dr. Zhou Wang (Department of Urology, University of Pittsburgh)
Dr. Dean Bacich (Department of Urology, University of Pittsburgh)

W. Chet de Groat, Ph.D.
Distinguished Professor

Dr. Naoki Yoshimura (Department of Urology, University of Pittsburgh): Collaborative urologic research in spinal cord injury
Dr. Lori Birder (Department of Medicine, University of Pittsburgh): Collaborative urologic research in spinal cord injury
Dr. Tony Kanai (Department of Medicine, University of Pittsburgh): New treatment for spinal cord injury
Dr. Changfeng Tai (Department of Urology, University of Pittsburgh): Mechanisms underlying neurogenic bladder disorders
Dr. Seong-Gi Kim and P. Wang (Radiology-Imaging Center, University of Pittsburgh): Neuroplasticity in the spinal cord
Drs. C. Bates and K. Walker (Pediatric Nephrology, University of Pittsburgh)
Dr. C. L. Cheng (Department of Urology, Taichung Veterans Hospital): Pharmacology of the lower urinary tract
Dr. M. Miyazato (Department of Urology, Japan)
Dr. Michael Chancellor (Department of Urology, Beaumont Hospital, Michigan): Use of liposomes in drug delivery
Drs. Pradeep Tyagi and M. Kashyap (Department of Urology, University of Pittsburgh): Use of liposomes in drug delivery
Dr. G. Salama (Department of Cardiology/Medicine, University of Pittsburgh)
Dr. Yan Xu (Department of Anesthesiology, University of Pittsburgh)
Dr. Sun Ok Yoon (Department of Neurosciences, Ohio State University)
Dr. Yoshio Arai (Department of Radiation Oncology)
Dr. Michael Ruggieri (Department of Physiology, Temple University)

Julie Eiseman, Ph.D.
Professor

Andreas Vogt, PhD (Drug Discovery): collaborations on preclinical studies of compounds to treat renal disease identified from zebra fish screen
Paul Johnston, PhD (Pharmaceutical Sciences): collaborations on the STAT 3 small molecule inhibitors and on reevaluation of combinations of older therapeutics for the NCI
Peter Wipf, PhD (Department of Chemistry): collaborations on preclinical studies of phosphatase inhibitors and tubulin or motor protein interactive small molecules, Wortmanin analogues, and protein kinase D inhibitors.
Dennis Curran, PhD (Department of Chemistry): collaborations on preclinical studies of tubulin or motor protein interactive small molecules.
Ed Prochownik, MD (Pediatrics, Children's Hospital): preclinical studies of myc inhibitors.
Thomas Smithgall, PhD (Department of Microbiology and Molecular Genetics): preclinical studies of NEF targeted small molecules.
Charles Horn, PhD (UPCI Behavioral Health): Pharmacokinetics and toxicity of cisplatin in shrews and determination of shrew body surface areas
John T. Comerci, MD (Department of OB/GYN Oncology, Magee Women's Hospital): Pc 4 PDT and Uterine cervical cancer
Ron Montelaro, PhD (Department of Microbiology and Molecular Genetics): cationic antibacterial agents
Jennifer Grandis, MD: Toxicity Studies of STAT 3 cyclic decoy in mice and STAT-3 cyclic decoy with radiolabel and microbubbles. Small molecule inhibitors of STAT 3
Jerry Collins, PhD (CDER, FDA, Rockville, MD): Studies of TS in high and low thymidylate synthase expressing tumors and development of PET probes, Zebularine.
Joe Covey, PhD (DTP, NCI, NIH, Rockville MD): animal pharmacology.
Doug Ross, MD (University of Maryland, Greenebaum Cancer Center, Baltimore, MD): mechanisms of drug resistance due to BCRP.
Ken Bauer, PhD (School of Pharmacy, University of Maryland, Baltimore, MD): pharmacokinetics (PK) of fumitremorganc in mice and topotecan PK following fumitremorganc.
Drs. Stan Gerson and Afshin Dowlati (Case Western Reserve University, Ireland Cancer Center, Cleveland, OH): Phase I studies.
David Zagzag, PhD (NY University Cancer Institute, Department of Pathology, New York, NY): Preclinical studies of geldanamycin analogues in gliomas.

Ivana Vucenik, PhD (University of Maryland, Baltimore): IP6 pharmacokinetics and inositol.

Irving Bigio, PhD (Boston University, Boston, MA): Use of optical pharmacokinetics system (ESS) to measure drugs with absorbance spectra in the long wavelength visible spectra including motexafin gadolineum, motexafin lutetium, PC4, and mitoxanthrone.

Steve Musser, PhD (FDA, Washington, DC): Characterization of metabolites by LC/MS/MS.

David D’Argenio, PhD (BMSR, School of Engineering, UCLA, Los Angeles CA): Pharmacokinetic and pharmacodynamic modeling of 17-AAG and DMAG, zebularine, and FdC.

Patrick McCarthey, PhD and Nicolay V. Tsarevsky, PhD (Chemistry, Carnegie Mellon University): delivery systems for anti-cancer agents such as the prototype agent, doxorubicin.

Nancy Olienick, PhD (Department of Radiation Biology, Case Western Reserve University): Pc 4 and Pc 181.

Alicja Bielawski, PhD (Department of Biochemistry, Medical University of South Carolina): studies of interaction of sphingolipid analogues with photodynamic therapeutics.

Anirban Sen Gupta, PhD (Department of Biomedical Engineering, Case Western Reserve University): preclinical studies of EGFR-targeted Pc4 containing micelles for improving PDT.

John S. Lazo, Ph.D. (Department of Pharmacology, University of Virginia): small molecule inhibitors of phosphatases

G. David Roodman MD PhD (Indiana University School of Medicine, Indianapolis, IN): sequestosome (p62) inhibitor

Peter Friedman, Ph.D.

Professor

Dr. John Wysolmerski (Yale University)
Dr. John Scott (HHMI, University of Washington)
Dr. Jean-Luc Parent (Sherbrooke, Quebec)
Dr. Kevin Xiao (Duke)
Dr. Marcel Bruchez (Carnegie-Mellon University)
Dr. Manoj Puthenveedu (Carnegie-Mellon University)
Dr. Simon Watkins (Department of Cell Biology, University of Pittsburgh)
Dr. Harry Blair (Department of Pathology, University of Pittsburgh)
Dr. Nathan Yates (Department of Cell Biology, University of Pittsburgh)
Dr. Tony Ferreira (Department of Computational and Systems Biology, University of Pittsburgh)

William Furey, Ph.D.

Professor

Dr. F. Jordan (Department of Chemistry, Rutgers University): E. coli pyruvate dehydrogenase multi-enzyme complex component structures
Dr. Guillermo Calero (Department of Structural Biology, University of Pittsburgh): Production of pyruvate dehydrogenase
Dr. J. Conway (Department of Structural Biology, University of Pittsburgh): Cryo-EM studies on pyruvate dehydrogenase

Ferrucio Galbiati, Ph.D.

Professor

Dr. Donald DeFranco (Department of Pharmacology & Chemical Biology, University of Pittsburgh): Nuclear receptor and caveolin-1
Sruti Shiva (Department of Pharmacology & Chemical Biology, University of Pittsburgh): caveolin-1 and mitochondrial function
Yu Jiang (Department of Pharmacology & Chemical Biology, University of Pittsburgh): caveolin-1, cellular senescence and primary cilium
Michael Lisanti (Paterson Institute for Cancer Research, The University of Manchester, Manchester, England): cigarette smoke and the tumor microenvironment

Jing Hu, Ph.D.
Visiting Associate Professor

Dr. Shihfan Kuan (Department of Pathology, University of Pittsburgh): Expression profiles of several Wnt pathway components and regulators in human colon cancer tissues and PDAD (pancreatic ductal adenocarcinoma) tissues
Dr. David Schlaepfer (Department of Reproductive Medicine, Moores Cancer Center, UCSD): FAK field; collaborated on the Elife publication
Dr. Farzad Esni (Department of Surgery, University of Pittsburgh): collaborating on two manuscripts and one grant application
Dr. Tim Burns (Department of Medicine, University of Pittsburgh): BRAF inhibitor resistance project
Dr. Laura Stabile (Department of Pharmacology & Chemical Biology, University of Pittsburgh): MAP4K4 in lung cancer
Eli Lilly and Plexxikon, Inc.: will provide next generation of BRAF inhibitors

Edwin Jackson, Ph.D.
Professor

Pat Kochanek, M.D. (Department of Critical Care Medicine, University of Pittsburgh): Adenosine in traumatic brain injury
Ed Dixon, Ph.D. (Department of Critical Care Medicine, University of Pittsburgh): Adenosine in brain dysfunction
Elieser Gorelik, Ph.D. (Department of Pathology, University of Pittsburgh): Role of adenosine in cancer
Gerard Apodaca, Ph.D. (Department of Medicine, University of Pittsburgh): Role of adenosine in bladder function
Derek W. Gilroy, Ph.D. (Division of Medicine, University College London): Role of adenosine in inflammation
Stevan P. Tofovic, M.D., Ph.D.; Center for Clinical Pharmacology, University of Pittsburgh): Estradiol metabolites in renal disease
Raghvendra K. Dubey, Ph.D. (Department of Obstetrics and Gynecology, University Hospital Zurich): Vascular biology of estradiol metabolites
Lisa Satlin, M.D. (Division of Pediatric Nephrology, Mount Sinai School of Medicine): Purine metabolomics
Virginia Miller, Ph.D. (Department of Surgery, Mayo Clinic): Estradiol Metabolomics

Yu Jiang, Ph.D.
Associate Professor

Dr. Ferro-Novick (University of California, San Diego): Autophagy-related study
Dr. Freeman (Department of Pharmacology & Chemical Biology, University of Pittsburgh): develop new areas of research
Dr. Weisz (Department of Medicine, University of Pittsburgh): develop new areas of research
Dr. Liu (Department of Pathology, University of Pittsburgh): develop new areas of research
Dr. Marston Linehan (NIH): kidney cancer research
Dr. Peter Harris (Mayo): studying polycystic kidney diseases
Nicholas Khoo, Ph.D.  
*Research Assistant Professor*

Drs. Shiva, Chartoumpekis (Kensler), Hackam and Bailey: collaborative studies  
Eric Kelley, Ph.D. (Department of Anesthesiology): high fat mouse study and the inhibition of xanthine oxidase

Jack Lancaster, Ph.D.  
*Visiting Professor*

Francisco Schopfer (Department of Pharmacology & Chemical Biology): endogenous mechanisms of nitro lipid formation  
Adam Straub (Department of Pharmacology & Chemical Biology): mechanisms of control of sags by cyB5R  
Courtney Watkins: EPR of Marc  
Adrie Steyn: pathogenesis of mycobacterium tuberculosis

Edwin Levitan, Ph.D.  
*Professor*

John Horn (Department of Neurobiology, University of Pittsburgh): Dopamine neuron project  
Elias Aizenman (Department of Neurobiology, University of Pittsburgh): Potassium channels and apoptosis  
David Deitcher (Cornell) and Randy Hewes (Oklahoma): Neuropeptide release in Drosophila  
Glenn Fishman (NYU): Cardiac potassium channel expression

Tatyana Mamonova, Ph.D.  
*Research Instructor*

Zimei Bu

Carola Neumann, M.D.  
*Visiting Associate Professor*

Drs. Michael Becich, Uma Chandra, Soumya Luthra, Adrian Lee, Steffi Oesterreich: development of a ROS-gene signature in basal like breast cancer  
Dr. Tom Smithgall: Rad51 project  
Drs. Carolyn Anderson and Steven Thorne (University of Pittsburgh): will support us in developing a mouse model allowing imaging of mammary stroma associated fibroblasts in the context of metastasis  
Drs. David Root and Glenn Cowley (Broad Institute, Boston): will support us in performing a shRNA kinase screen to identify kinases promoting CAFs  
Drs. Christine Winterbourn and Mark Hampton (University of Otago), Elizabeth Veal (Newcastle University): all PIs are leading in the Prdx field  
Drs. Li Lan and Liu Yang (UPCI): RAD51 project and telomers and ROS  
Dr. Da Yang (School of Pharmacy): evaluating mirlet7 in breast cancer and genomic stability in PRDX1 high and low expressing tumor samples  
Drs. Steffi Oesterreich and Kevin Levine: TCGA and metabric analysis of PRDX1/FOXO3/Let7 expression and survival with FOXO3 target genes  
Dr. Bruce Freeman and Julia Woodcock: Rad51 project  
Dr. Adam Feinberg (CMU): collagen compaction assay; quantifying collagen structures in mammary glands; building a microfluidic device to examine collective invasion *in vitro*  
Dr. Philip DeLuc (CMU): building a microfluidic device to mimic hypoxic gradients in 3D
Dr. Daniel Normolle (Department of Biostatistics): involvement in all projects

**Roderick O’Sullivan, Ph.D.**  
*Assistant Professor*

Kara Bernstein (University of Pittsburgh Cancer Institute): The role of RNF4 in the alternative lengthening of telomeres pathway  
Chris Bakkenist (University of Pittsburgh Cancer Institute): Localization of ASF1 to replication forks  
Genevieve Almouzni (Institut Curie, Paris, France): Deregulation of the histone supply chain leads to activation of the ALT pathway  
Robert Sobol (University of Southern Alabama): The role of PARP and PARG in telomere maintenance and replication fork stability  
Matthias Fischer (University of Cologne, Germany): Investigation of ALT in pediatric neuroblastoma

**Patrick Pagano, Ph.D.**  
*Professor*

P. Michael Bauer (Department of Surgery, University of Pittsburgh)  
Aaron Barchowsky (Department of Environmental and Occupational Health, University of Pittsburgh)  
Robin Gandley (Magee Women’s Hospital)  
Jeffrey Isenberg (Department of Medicine, University of Pittsburgh)  
Xiang Gao (Department of Pharmaceutical Sciences, University of Pittsburgh)  
Song Li (Department of Pharmaceutical Sciences, University of Pittsburgh)  
Carlos Camacho (Department of Computational Biology, University of Pittsburgh)  
Guangjie Cheng (Emory University)  
Phil Palade (University of Arkansas)  
Xiao-Ping Yang (Henry Ford Hospital)  
William Beierwaltes (Henry Ford Hospital)

**Michael Palladino, Ph.D.**  
*Professor*

Ron Wetzel (University of Pittsburgh): The study of TPI aggregation and involvement in amyloidopathies  
Andrew Van DeMark (University of Pittsburgh): The study of TPI protein pathogenic structure  
Peter Andolfatto (Princeton University): collaborate to study pharmacology of Na/K ATPase alpha  
Brett Kaufman (University of Pennsylvania): small mitochondrial imported RNAs modulate mito DNA copy number  
Larry Rieter (University of Tennessee Health Science Center, Memphis, TN): Ubiquitin-mediated turnover of the Na/K ATPase

**Wei Qian, Ph.D.**  
*Research Instructor*

Peter Wipf  
Patricia Opresko

**Guillermo Romero, Ph.D.**  
*Associate Professor*

Susan Amara (University of Pittsburgh): elucidation of the mechanisms by which amphetamines regulate the uptake of the dopamine transporter
Jing Hu (Department of Pharmacology & Chemical Biology, University of Pittsburgh): project that focuses on the role of HDAC2 in the regulation of the Wnt signaling pathway

James Roppolo, Ph.D.
Research Assistant Professor

Changfeng Tai (Department of Urology, University of Pittsburgh): Neurourology and pharmacology studies
Lori Birder (Department of Medicine, University of Pittsburgh): Neurourology and IC studies

Francisco Schopfer, Ph.D.
Research Associate Professor

Eugene Chen (University of Michigan): Study of the mechanisms of PPAR gamma activation by nitrated fatty acids
Anna Lisa Levonen (University of Kuopio, Finland): Study of the activation of phase 2 genes by nitroalkenes, mainly focusing on KEAP/Nrf2 pathway

Sruti Shiva, Ph.D.
Assistant Professor

Jeffrey Isenberg (Department of Medicine, University of Pittsburgh)
Mark Gladwin (Department of Medicine, University of Pittsburgh)
Anje Cauwels (University of Ghent, Belgium)
Tienush Rassaf (University Hospital Aachen, Germany)
William Frazier (Washington University, St. Louis, MO)

Shivendra Singh, Ph.D.
Professor

Sruti Shiva (Department of Pharmacology & Chemical Biology, University of Pittsburgh)
Rohit Bhargava (Department of Pathology, University of Pittsburgh)
Rachel Jankowitz (Department of Medicine, University of Pittsburgh)
Saumen Sarkar (Department of Microbiology and Molecular Genetics, University of Pittsburgh)
Yong Wan (Department of Cell Biology and Physiology, University of Pittsburgh).

Robert Sobol, Ph.D.
Associate Professor

Ben Van Houten (Department of Pharmacology & Chemical Biology, University of Pittsburgh): PARP activation and the impact on mitochondrial function (SeaHorse)
Patty Opresko and Ben Van Houten (University of Pittsburgh) and Marcel Bruchez (Carnegie Mellon University): Development of targeted in vivo protein-encoded DNA damaging agents (specific to telomeres, mitochondria and site-specific nuclear sites)
Carlos Camacho (University of Pittsburgh) and Geoff Wahl (Salk): Small molecule inhibitors of PolB/XRCC1 heterodimer formation
Ichiro Nakano (OSU): glioma stem cells
Charlie Brenner (Iowa): NAD metabolomics
Guy Poirier (CHUL Research Center): PARP proteomics
Nathan Yates (University of Pittsburgh): PolB and UBE3B interacting proteins
Conchita Vens (NKI): PolB in cancer
Laura Stabile, Ph.D.
Research Associate Professor

Phouthone Keovahong and Y. P. Peter Di (Department of Environmental and Occupational Health, University of Pittsburgh): Lung Inflammation Project and KRAS Project, R01 submitted
Richard Pietras, Edward Garon and Diane Marquez-Garbin (UCLA): clinical trials
Jennifer Grandis, Julie Bauman and Ann Marie Egloff (Department of Otolaryngology, University of Pittsburgh): Head and Neck SPORE Project 4 and R01 submitted with Dr. Bauman
Sanja Dacic (Department of Pathology, University of Pittsburgh): Lung SPORE Project 1
Pamela Hershberger (Roswell Park Cancer Institute and UPCI): Vitamin D and EGFR mutation project
Brenda Diergaard (Department of Epidemiology, University of Pittsburgh): Obesity and Lung Cancer Risk – R21 Project and SPORE Project 3
Yan Lin and Brenda Kurland (UPCI Biostatistics): Lung SPORE Project 1
Jill Siegfried (University of Minnesota): Lung SPORE Project 1 and HGF/Estrogen Project
Timothy Burns (Division of Hematology-Oncoology, University of Pittsburgh): EGFR, MET and estrogen related projects
Ahmad Tarhini (Department of Medicine, University of Pittsburgh): clinical trials
Lisa Villaruz (Department of Medicine, University of Pittsburgh): clinical trials
Guitain Xiao (Department of Microbiology and Molecular Genetics): NF-ƙ and lung cancer project
Sam Rothstein (Qroni, Inc.): SBIR Award - reformulation of failed tublin inhibitor

Adam Straub, Ph.D.
Assistant Professor

Donald DeFranco (Department of Pharmacology & Chemical Biology, University of Pittsburgh)
Liza Villanueva (Department of Cardiology, University of Pittsburgh)
Edwin Jackson (Department of Pharmacology & Chemical Biology, University of Pittsburgh)

Ben Van Houten, Ph.D.
Professor

Marcel Bruchez (Carnegie Mellon University): developing fluorogen activating peptides and chemicals for creating and imaging ROS with high temporal and spatial resolution in living cells
Neil Kad (University of Kent, Canterbury, UK): single-molecule dynamics of DNA repair enzymes.
Caroline Kisker (University of Wurzburg, Wurzburg, Germany): structure and function of nucleotide excision repair proteins. Cynthia McMurray, Lawrence Berkeley Laboratories, Single molecule analysis of Msh2/Msh6
Jung-Hyun Min (University of Illinois, Chicago): single molecule analysis of XPC-HR23b and Rad4-Rad23 interaction with DNA. Alan Tomkinson, University of Arizona, DNA ligases and cancer
Yukching Tse Dinh (Florida International University): Single molecular analysis of bacterial Topoisomerase I
Samuel H. Wilson (NIEHS, NIH): single molecule analysis of PARPI and APEI
John Wyrick (Washington State University, Pullman): analysis of Rad4 mutants in yeast.
Ed Burton: zebrafish models of PD using protein targeting of ROS
Charleen Chu: effect of Parkin on mitochondrial oxidative phosphorylation
Ed Prochownik: effect of Myc on mitochondria biogenesis and structure. Alex Dieters, photoactivatable switches in helicases
Simon Watkins: visualization of single-molecules of QDOT labeled DNA repair proteins, development of fluorogen activating peptide imaging techniques for the generation and measurement of ROS in high spatial and temporal resolution in living cells.

Peter Wipf: development of unique series of thioquinazolinone compounds that work synergistically with cisplatin to kill cisplatin resistant tumors in a bax/bak independent manner; mitochondrially targeted anti-cancer compounds

Kara Bernstein: analysis of Rad4 mutants in yeast

**Jean-Pierre Vilardaga, Ph.D.**

*Professor*

Harvard University, Endocrine Unit
Harvard University, Center for Systems Biology
University of Wuerzburg (Germany), Institute of Pharmacology
University of Barcelona (Spain), Department of Pathology and Experimental Therapeutics
University of Santiago de Compostela (Spain), Department of Pharmacology

**Daniela Volonte-Calbiati, Ph.D.**

*Research Assistant Professor*

Donald DeFranco (Department of Pharmacology & Chemical Biology, University of Pittsburgh): role of Caveolin-1 in nuclear receptor signaling

**Bennett Van Houten, Ph.D.**

*Professor*

Carolyn Anderson, Barry Edwards: fatty acid metabolism in cancer cells
Kara Bernstein: binding of the shu complex to DNA
Dana Bovbjerg and Frank Jenkins: role of stress in ROS generation, and mitochondrial and nuclear DNA damage
Louis Falo: action mechanisms of radiation toxicity amelioration by mitochondrially targeted antioxidants
Charlee Chu: mitochondrial bioenergetics and Parkinson’s disease
Nancy Davidson: analysis of metabolic flux in human breast cancer cell lines
Robert Edwards: analysis of fatty acid metabolism in ovarian cancer cells
Timothy Greenamyre: mitochondrial DNA damage and Parkinson’s disease
Li Lan: use of killer red to examine DNA repair in real time
Michael Lotze: mitochondrial bioenergetics and T cell maturation
Patty Opresko: mitochondria and telomere cross-talk
Edward Prochownik: myc and mitochondrial function and dynamics
Simon Watkins: signal molecule analysis of DNA repair enzymes
Peter Wipf: XJB protection of oxidant injury to mitochondria
Marcel Bruchez (Carnegie Mellon University): development of fluorogenic activating peptides for targeted ROS generation
Sheila David (University of California, Davis): analysis of nucleotide excision repair in the removal of oxidative lesions
Neil Kad (University of Essex): single molecule studies of bacterial repair proteins
Peter McHugh ((Oxford University): analysis of yeast repair proteins using single molecular techniques
Jung-Hyun Min (Department of Chemistry, University of Illinois): analysis of human DNA damage recognition proteins by single molecular techniques
Alan Tomkinson (University of New Mexico): development of anti-DNA ligase drugs
Roger Woodgate (NIH, NICHD): role of nucleotide excision repair on the removal of RNA incorporated in DNA

**Q. Jane Wang, Ph.D.**
*Associate Professor*

Billy Day (Department of Pharmaceutical Sciences, University of Pittsburgh)  
Adam Glick (Penn State University)  
J. Frederic Mushinski (National Cancer Institute)  
Peter M. Blumberg (National Cancer Institute)

**Stacy Gelhaus Wendell, Ph.D.**
*Research Assistant Professor*

Fernando Holguin and Sally Wenzel (Asthma Institute, University of Pittsburgh)  
Donald DeFranco and Ben van Houten (Department of Pharmacology & Chemical Biology, University of Pittsburgh)  
Robert Sobol (Mitchell Cancer Center)  
Natalia Kedishvili (University of Alabama at Birmingham)  
Nathaniel Snyder (Drexel University)

**Kevin (Kunhong) Xiao, M.D., Ph.D.**
*Visiting Associate Professor*

Dr. Yutong Zhao (Division of Pulmonary, Allergy, and Critical Care)  
Dr. Nathan Yates (Department of Cell Biology and BioMS Center)  
Dr. Pei Tang (Department of Anesthesiology)  
Dr. George C. Tseng (Department of Biostatistics)  
Dr. Tim Lezon (Department of Computational & Systems Biology)  
Dr. Ivet Bahar (Department of Computational & Systems Biology)  
Dr. Robert Lefkowitz (Duke University)  
Dr. Howard Rockman (Duke University)  
Dr. Neil Freedman (Duke University)  
Dr. Sudha Shenoy (Duke University)  
Dr. Steven Gygi (Harvard University)  
Dr. Jonathan Stamler (Case Western Reserve University)  
Dr. Walter Koch (Temple University)  
Dr. Jinfeng Sun (Shandong University, China)  
Dr. Scott Prosser (University of Toronto, Canada)  
Dr. Dawn Z Chen (Cedars Sinai Medical Center)  
Dr. Richard Kurten (University of Arkansas for Medical Sciences)  
Dr. Richard Bouley (Harvard University)

**Cheng Zhang, Ph.D.**
*Assistant Professor*

Brian Kobilka (Stanford University)  
Kayoung Chung (Sungkyunkwan University, Korea)  
Yulong Li (Peking University, China)  
Hao Fan (A*Star, Singapore)
Peijun Zhang (University of Pittsburgh)

**Lin Zhang, Ph.D.**
Professor

Xiao-Ming Yin (Department of Pathology, University of Pittsburgh): Studying apoptosis induced by proteasome inhibitor
Cary Wu (Department of Pathology, University of Pittsburgh): Studying apoptosis caused by chance in extracellular matrix
Tao Cheng (Department of Radiation Oncology, University of Pittsburgh): Studies of PUMA-knockout mice
Robert Schoen (Department of Medicine, University of Pittsburgh): Studying chemoprevention of colon cancer by NSAIDs
Wei Zhou (Emory University): Developing molecular markers of lung cancer
Chuanshu Huang (New York University): Studying apoptosis induced by anti-cancer drugs
Jim Herman (Johns Hopkins University): Studying DNA methylation in lung cancer
Gerry Zambetti (St. Jude’s Children’s Hospital): Studies of PUMA-knockout mice
Chinese Academy of Medical Sciences: Studying esophageal cancer drug response

**Entrepreneurial Activities**
None.

**Awards and Honors**

**W. Chet de Groat, Ph.D.**
*Distinguished Professor*
Ferdinand C. Valentine Award, New York Academy of Medicine, 2015

**Thomas Kensler, Ph.D.**
*Professor*
Honorary Professor, Nantong Tumor Institute, Nantong, China
NCI Outstanding Investigator Award (R35), 2015-2022
Touching Jiangsu Award for Outstanding Expatriates from the Jiangsu Government, Nanjing, Jiangsu, China, 2015
Thomas Reuters Highly Cited Researcher (top 1% in Pharmacology and Toxicology), 2015

**Jack Lancaster, Ph.D.**
*Professor*
Fellow of the Society for Redox Biology and Medicine

**Edwin Levitan**
*Professor*
NARSAD Distinguished Investigator Grant, 2015
Invited Talks

Bruce Freeman, Ph.D.
Professor and Chair


“Formation and Signaling Actions of Electrophilic Fatty Acids.” The Environmental Response IV International Symposium, Tohoku University, Sendai, Japan, March 1, 2014.

“Pleiotropic Anti-Inflammatory Signaling Actions of Redox-Derived Fatty Acid Electrophiles.” Oxidative Stress Conference, Toledo, Spain, October 4, 2014.

“Forging New Drugs from the Fires of Inflammation.” College of Wooster, Wooster, OH, March 26, 2015.

Daniel Altschuler, Ph.D.
Associate Professor

School of Sciences, University of Buenos Aires, December 3, 2013.

School of Sciences, University of Buenos Aires, August 14, 2014.

School of Sciences, University of Buenos Aires, May 15, 2015.

Dr. Alessandro Bisello
Associate Professor


“Coordination of Tissue Remodeling by the PDZ Protein EPB50/NHERF1.” Dartmouth College, July 2014.

Donald DeFranco, Ph.D.
Professor

4th International Recent Advances in Health & Medical Sciences/International Conference on Oncology and Anticancer. Cyprus, June 2013.


Rapid Responses to Steroid Hormones International Conference, Erie, PA, September 2013.

Symposium Speaker, Society for Basic Urology Research Annual Meeting, Dallas, TX.

Invited Seminar: Cedars-Sinai Cancer Institute and Medical Center, 2015.
Invited Seminar: Department of Biochemistry, University of Southern California, 2015.

Invited Seminar: Department of Cell and Molecular Biology, Baylor College of Medicine, 2015.

Invited Seminar: Center for Nuclear Receptors and Cell Signaling, University of Houston, 2015.

Plenary Speaker: Ohio Physiological Society, 2015.

W. Chet de Groat, Ph.D.
Distinguished Professor


Opportunities for the SPARC Program. IN: Stimulating Peripheral Activity to Relieve Conditions


Peter Friedman, Ph.D.
Professor

Hans Prydz Lecture, Norway Centre for Molecular Medicine, University of Oslo, May, 2013.

Department of Pharmacology, University of Washington, September, 2013.

Sullivan Lecture, Kansas University Medical Center, November, 2013
University of California San Francisco, Endocrine, February 2014


Ferruccio Galbiati, Ph.D.
Professor


William Furey, Ph.D.
Professor


Ryan Hartmaier, Ph.D.
Research Instructor


Jing Hu, Ph.D.
Visiting Associate Professor

Targeting Deregulated PTM Pathways for the Treatment of CRC and PDAC. Southwest Hospital, Third Military Medical University, Chongqing, China, October 29, 2014.

Targeting Deregulated PTM Pathways for the Treatment of CRC and PDAC. Cancer Center, Daping Hospital and Research Institute of Surgery Third Military Medical University, Chongqing, China, October 30, 2014.
Yi Huang, M.D., Ph.D.
Assistant Professor


Edwin Jackson, Ph.D.
Professor

Jackson, E.K.: Role of the 2',3'-Cyclic AMP-Adenosine Pathway in Kidney and Brain Injury. Presented to the Department of Anesthesiology, University of Colorado Denver, August 26, 2013.


Jackson, E.K.: Role Of The 2',3'-cAMP-Adenosine Pathway in Traumatic Brain Injury. Presented to the 33rd Annual Symposium of the National Neurotrauma Society, Santa Fe, New Mexico, July 1, 2015.


Yu Jiang, Ph.D.
Assistant Professor

“Connecting mTOR to mitochondria” (2013) Brain Institution, Fudan University School of Medicine. Shanghai.

“Regulation of mTOR” (2013) National Center for Animal Models, Shanghai.

“A crosstalk between life and death: The role of FKBP38 in cell growth and apoptosis” (2013), Dean’s Inaugural Lecture, Suzhou University College of Medicine, Suzhou, China.

“The role of FKBP38 in tumor suppression” (2013). Department of Biochemistry and Molecular Biology, Louisiana State University Health Sciences Center, Shreveport, LA.

“A novel crosstalk between apoptotic proteins and mTOR” (2013). (Session Chair). 4th World Gene Convention. Haikou, China

Gcn2 is an amino acid sensor for TORC1 in yeast. Metabolism, Diet and Disease: Cancer and Metabolism, Washington DC, May 2014.

Signaling Mechanism of tumor suppressor folliculin. International Frontier Biological Science Symposium, Chengdu, China, June 2014.

Regulation of mTOR. Department of Pathology, Indiana University School of Medicine, Indianapolis, IN, September 2014.

Signaling mechanisms of primary cilium. Brain Institution, Fudan University School of Medicine, Shanghai, China, November 2014.


Signaling through primary cilia. Department of Biology, New Orleans University, New Orleans, LA, March 2015.

Signaling through primary cilia. Chongqing Medical University, Chongqing, China, July 2015.

Nutrient signaling in yeast. School of Life Science and Biotechnology, Dalian University of Technology, Dalian, China, July 2015.

mTOR signaling in cancer. School of Life Science, South China University of Sciences and Technology, Guangzhou, China, November 2015.

Regulation of mTORC1 through primary cilia. Rutgers Cancer Institute, New Brunswick, NJ, November 2015.


Thomas Kensler, Ph.D.

Professor

Cancer Epidemiology, Prevention and Control Program, University of Pittsburgh Cancer Institute, 2013


International Workshop on Ageing and Cancer Cell Biology: Convergent and Divergent Molecular Mechanisms, Athens, Greece, 2013

XIII International Congress of Toxicology, Seoul, Korea, 2013

College of Pharmacy, Catholic University of Korea, Seoul, Korea, 2013

Gordon Research Conference: Cellular and Molecular Mechanisms of Toxicity, Andover, NH, 2013

Department of Food Science, Penn State University, University Park, PA, 2013

C Malcolm Trout Annual Lecture, Michigan State University, East Lansing, MI, 2013

Department of Pharmacology & Toxicology, Michigan State University, East Lansing, MI, 2013

Xiangshan Science Conference on Frontiers in Cancer Chemoprevention, Beijing, China, 2013
Aflatoxin Symposium, Vanderbilt University, Nashville, TN, 2013


Rutgers Cancer Institute of New Jersey, New Brunswick, NJ, 2013

Miller Lectureship, Weil Cornell Medical School, New York, NY, 2013

Environmental Response IV Conference, Sendai, Japan, 2014

Society for Free Radical Research International Biennial Meeting, Kyoto, Japan, 2014


Department of Pediatric Oncology, MD Anderson Cancer Center, Houston, TX, 2014


School of Public Health, University of Washington, Seattle, WA, 2014

FHCRC – China Workshop on Environmental Exposure and Lung Cancer, Seattle, WA, 2014

Biochemical Society Meeting on the Keap1/Nrf2 Pathway in Health and Disease, Cambridge, UK, 2015

International Symposium on Current Advances in Radiobiology, Stem Cells and Cancer Research, Jawaharlal Nehru University, New Delhi, India, 2015

Department of Medical Biochemistry, Tohoku University School of Medicine, Sendai, Japan, 2015

Spectroscopy Society of Pittsburgh, Duquesne University, Pittsburgh, PA, 2015

Nantong Tumor Institute, Nantong, China, 2015


College of Pharmacy, Catholic University of Korea, Seoul, Korea, 2015

Plenary Lecture, International Conference on Natural Products for Cancer Prevention and Therapy, Istanbul, Turkey, 2015

Conference on “Diet and Optimum Health”, Oregon State University, Corvallis, OR, 2015

Keynote Lecture: Advances in Cell Signaling, Cancer Prevention and Therapy Symposium, UT Health Science Center at San Antonio, South Padre Island, TX, 2015
Nicholas Khoo, Ph.D.
Research Assistant Professor

Nitrite and Nitro-Fatty Acids Potential Therapeutics against Metabolic Syndrome. Presented at Fifth International Meeting on the Role of Nitrite and Nitrate in Physiology, Pathophysiology, and Therapeutics; Pittsburgh, PA, May 2013.

Jack Lancaster, Ph.D.
Professor

Dinitrosyliron Complexes: What May We Know about the Cellular Function of a Tumor Biomarker First Described 50 Years Ago? Gordon Research Conference on Oxygen Radicals: From Detection to Disease, Ventura, California, 2014.

A Radical Notion: Specialization is for Insects. 2014 Lifetime Achievement Award, Society for Free Radical Biology and Medicine, Seattle, WA, 2014.

Hypoxia-Induced Cellular Nonheme Iron Mobilization and Nitric Oxide: Biological Functions of an Unusual EPR Signal Observed in Tumors 50 Years ago. University of Illinois, Chicago, Department of Medicinal Chemistry and Pharmacognosy, 2014.

Joan M. Lakoski, Ph.D.
Professor


Invited Speaker, “Preparing for Success: Career Opportunities in the Health Sciences”, La Roche College, Pittsburgh, PA, April 15, 2013. (Target Audience: Undergraduate Students)

Invited Speaker, “Grant Award Types”, University of Pittsburgh Faculty Development Fellowship Program, Pittsburgh, PA, April 18, 2013.

Invited Speaker, “Essential Networking as a Professional”, America’s Next Top Infectious Disease Model: HIV & Influenza Conference hosted by the Department of Epidemiology, Harvard School of Public Health Annual Conference for Undergraduate Students, Chicago, IL, April 21, 2013.

Invited Speaker with Dr. Robert J. Milner, “Know Your Kangaroo: Pathway to Independence Award (K99/R00), NIH Career Development Award Workshop, New York University Langone Medical Center, New York, NY, May 1, 2013.
Invited Speaker with Dr. Robert J. Milner, “Know Your NRSA” New York University Langone Medical Center, New York, NY, May 1, 2013.

Invited Speaker, “Applying for the NIH Pathway to Independence (K99/R00) Award, Center for Neuroscience, West Virginia University, Morgantown, WV, May 15, 2013.

Invited Presenter, “Maximizing Success in a Dynamic Mentoring Relationship”, Faculty Mentoring Workshop, Center for Neuroscience, West Virginia University, Morgantown, WV, May 15, 2013.

Invited Faculty; “Maximizing Success as a Research Mentee”, 2013 MIDAS Summer Undergraduate Research Program, University of Pittsburgh MIDAS Center of Excellence, Pittsburgh, PA, May 24, 2013.


Invited Speaker, “Achieving Your Career Goals through Dynamic Mentoring”, Department of Ophthalmology Retreat, University of Pittsburgh School of Medicine, August 14, 2013.


Invited Presenter and Workshop Host, “Discovering the Leader within You: Exploring the Full Spectrum of Science Careers”, held on campus at Morehouse Medical College, Atlanta, GA, September 18, 2013. (Target audience of graduate students and postdoctoral fellows)


    General Workshop: “Steps to a Competitive Application”
    “Navigating NIH”
    “Mock Study Section”
    “15 Steps to the Payline”
    “Know Your NRSA”
    “Know Your K”
    “Know Your Kangaroo”

Invited speaker with Dr. Robert J. Milner, “NIH Career Development & Training Award Workshops”, Rosalind Franklin University of Medicine and Science, North Chicago, IL, September 30, 2013.
General Workshop: “Steps to a Competitive Application”
“Know Your NRSA”
“Know Your K: A Guide to applying for a Career Development Award”


Invited Speaker with Dr. Robert J. Milner, “NIH Career Development & Training Award Workshops”, University of Massachusetts Medical School, Worcester, MA, October 31, 2013.

General Workshop: “Steps to a Competitive Application”
“Know Your Awards:
Know your NRSA
Know your K
Know Your Kangaroo (Pathways to Independence (K99/R00 Award))”

Adrian Lee, Ph.D.
Professor

"IGFs and breast cancer" Insulin, obesity and Cancer, Taormina, Italy, 2013.

"Tumor heterogeneity" 22nd Breast Cancer Think Tank, Dominican Republic, 2013.

"Applying Personalized Genomic Medicine to Breast Cancer" Center for Integrated Oncology, Cologne, Germany, 2013.


“Genomic and Transcriptomic Changes in Metastatic Breast Cancer.” Grand Rounds, Case Western Research University, Cleveland, OH, 2014.

"Cancer Genomics: The more we sequence the more we find". Science 2014, University of Pittsburgh, Pittsburgh, PA, 2014.

"SHNG7 is an IGF regulated lncRNA critical for cell growth". Pharmacology and Chemical Biology seminar series, University of Pittsburgh, PA, 2014.


"Sequencing breast cancer metastases reveal therapeutic targets". Susan G. Komen Annual Meeting, Dallas, TX, 2015.


"Genome-wide Systems Biology in Breast Cancer'.” CNAST, Carnegie Mellon University, Pittsburgh


"Understanding cancer metastasis through massively parallel sequencing." 5th Symposium of Shanghai Jiao Tong University and UPSOM, Shanghai, China, 2015.

"Branched evolution in breast cancer metastasis.” Cancer Center symposium, Ohio State University, Columbus, OH, 2015.

"Cancer therapy in the era of genomic medicine.” RIMED symposium, Rome, Italy, 2015.


Carola Neumann, M.D.
Visiting Associate Professor


"A novel FOX03-PRDX1 signaling pathway acts as an oxidative stress sensor and induces FOX03-dependent transcription of let-7 miRNAs.” Deutsches Krebs Forschungs Zentrum, Heidelberg, Germany, April 16, 2015.
“Prdx1 in breast cancer signaling.” Great Lakes Breast Cancer Symposium, Case Comprehensive Cancer Center, Cleveland, OH, June 2015.

“A PRDX1-specific redox regulation of FOX03 subcellular localization and transcription of mirLet7s.” PRDX Meeting, Yonsei University College of Medicine, Korea, November 2015.

**Steffi Oesterreich, Ph.D.**

*Professor*


“Invasive Lobular Cancer (ILC): A Different B(r)east.” University of Denver Cancer Center, March 2014.


“Endocrine Resistance in Lobular Breast Cancer.” Harbin Medical School, Harbin, China, April 2014.

“Endocrine Response in Women’s Malignancies.” Charite, Humboldt University, Berlin, August 2014.

“Estrogen Response in Lobular Cancer.” Wright State University, Department of Biochemistry and Molecular Biology, Dayton, OH, October 2014.

“Role of Epigenetics in Endocrine Resistant Breast Cancer.” Fels Institute, Temple University, Philadelphia, PA, November 2014.


**Roderick O’Sullivan, Ph.D.**

*Assistant Professor*

NIH DNA Repair Video Seminar Series. NIH, Frederick, MD, 2014.
Patrick Pagano, Ph.D.

Professor

Georgia Regents University, Section of Experimental Medicine, "Sly as a NOX, a Retrospective on the Challenges & Triumphs of Targeted Therapy", Augusta, GA, September 5, 2013.

2013 Redox Biology Center Symposium, University of Nebraska – Lincoln, Redox Signaling: A Potential Therapeutic Target for Human Disease, Nox Inhibitor Discovery & Vascular Disease, a Retrospective, Lincoln, NE, October 10, 2013.

British Heart Foundation Glasgow Cardiovascular Research Centre, University of Glasgow, The Quest for Nox Therapeutics, Perspectives on the Challenges, Pitfalls & Triumphs, Glasgow, Scotland, October 16, 2013.

Jagiellonian Centre for Experimental Therapeutics (JCET), Jagiellonian University, NOX, a Retrospective on the Challenges & Triumphs of Targeted Therapy, Krakow, Poland, October 18, 2013.

University of Oklahoma Health Sciences Center, Department of Physiology, New Frontiers in Nox Regulation, From Mechanism to Therapy, Oklahoma City, OK, February 13, 2014.

“Frontiers in Nox Regulation, from Novel Modulators to Nox Inhibitors.” Virginia Commonwealth University, Department of Pharmacology & Toxicology, Richmond, VA, March 27, 2014.

“New Insights into Nox Regulation, from Mechanism to Therapy.” University of Missouri-Columbia, Health Sciences Center, Department of Pharmacology & Physiology, Columbia, MO, May 6, 2014.

“New Players on the Nox Stage, From Chaperones to Effectors.” Klinikum der Goethe-Universität, Institut für Kardiovaskuläre Physiologie, Vascular Research Centre, Frankfurt am Main, Germany, May 14, 2014.


“Modulators and Signaling Pathways Controlling Vascular Nox, From Chaperones to New Inhibitors.” Northeast Ohio Medical University, Department of Integrative Medical Sciences, Dr. Hans G. Folkesson Memorial Seminar Series, Rootstown, OH, November 4, 2014.

"New Players on the Nox Stage, From Chaperones to Effectors.” American Heart Association Scientific Sessions, Cardiovascular Seminar Series: Redox Signaling in Cardiovascular Disease: NADPH Oxidase and Beyond, Chicago, IL, November 17, 2014.

“Phenotypic Switching of Vascular Smooth Muscle, Role of MEF2B & NOX1.” Tulane University School of Medicine, Department of Pharmacology, New Orleans, LA, February 2015.

“Oxidation-Dependent and –Independent Phenotypic Switching of Vascular Smooth Muscle, Role of MEF2B & C and NOX1”. Temple University School of Medicine, Cardiovascular Research Center, Department of Physiology, Philadelphia, PA, April 2015.

“Oxidation-Dependent Phenotypic Switching of Vascular Smooth Muscle, Role of MEF2B and NOX1.” University of Louisville, Department of Biochemistry & Molecular Genetics, Louisville, KY, May 2015.

“Phenotypic Switching of Vascular Smooth Muscle, Role of MEF2B and MEF2C.” Louisiana State University Health Sciences Center – Break & Malcolm Feist Cardiovascular Disease Seminar, Center for Cardiovascular Diseases and
Michael Palladino, Ph.D.
Professor


“Genetic Mitochondrial Manipulation Strategies.” UMDF Mitochondrial Medicine, 2014.

Wei Qian, Ph.D.
Research Instructor

Molecular and Cellular Cancer Biology Retreat, Pittsburgh, PA, February 2015.

Samuel Wilson Symposium, Pittsburgh, PA, May 2015.

Guillermo Romero, Ph.D.
Associate Professor

From Macrocephaly to Breast Cancer: The Surprising Biology of NHERF1, an Adapter Protein. California State University-Northridge, January 2015.

Francisco Schopfer, Ph.D.
Research Associate Professor

Bioactive Lipids Conference, Puerto Rico, 2013

Mass Spectrometry Course, Department of Medicine, Universidad de la Republica, Uruguay, 2014

Laboratory of Oxygen Metabolism, School of Medicine, University of Buenos Aires, 2014.

Department of Experimental Biology, School of Experimental Sciences, University of Jaen, Spain, 2015.

Sruti Shiva, Ph.D.
Associate Professor


“Platelet mitochondria: From mechanism to biomarker of Disease.” Department of Hematology, Emory
University, Atlanta, Georgia, April 2013.

“Nitrite modulates mitochondrial function through fusion in normoxia.” International Meeting of the Role of Nitrite in Health and Disease, Pittsburgh, PA, May 2013.


TrMAD regional meeting

International Nitric Oxide Meeting

University of Nevada Reno

Mitochondria and Cellular Metabolism Symposium; Montevideo, Uruguay

Mitochondrial Bioenergetics and Metabolism Course; Montevideo, Uruguay


The Dose Response Society – Biological Preconditioning and Hormesis Conference. “Nitrite mediates preconditioning through mitochondrial modulation.” Amherst, MA, April 2015.


Matthew Sikora, Ph.D.
Research Instructor


"Endocrine response and resistance in invasive lobular carcinoma of the breast,” Dept. of Pathology Seminar Series, University of Colorado Comprehensive Cancer Center, 2015.

"Endocrine response and resistance in invasive lobular carcinoma of the breast,” Breast and Ovarian Cancer Program, Johns Hopkins Medical Institute I Kimmel Cancer Center, 2015.

"Endocrine response and resistance in invasive lobular carcinoma of the breast,” Cancer Epidemiology Program Research Seminar Series, University of Hawai‘i Cancer Center, 2015.
Shivendra Singh, Ph.D.
Professor

Elkin Lecture, Emory University Winship Cancer Institute, Atlanta, GA. Title: Biomarkers of Cancer Prevention by Dietary Isothiocyanates, January 11, 2013.

University of Texas, Austin, TX. Title: Bioactive Food Components and Cancer Chemoprevention: Cancer Prevention with Dietary Isothiocyanates, March 29, 2013.

Phytochemicals and Cancer Prevention. Department of Pharmacology and Toxicology, University of Arkansas for Medical Sciences, Little Rock, AR, April 2014.


Cancer Chemoprevention with Isothiocyanates. International Symposium on Current Advances in Radiobiology, Stem Cells, and Cancer Research, Jawaharlal Nehru University, India, February 2015.


Redox Signaling in Cancer Chemoprevention by Dietary Phytochemicals. Frontiers in Dietary Cancer Chemoprevention: Fulfilled Promises and Future Directions, University of Gdansk, Gdansk, Poland, June 2015.


Robert Sobol, Ph.D.
Associate Professor

PARP1 - A mediator of genotoxin response pathway crosstalk. University of North Texas, Department of Molecular Biology and Immunology Seminar, Denton, TX, February 4, 2013.

Proteosome-mediated regulation of base excision repair. LUMC, Department of Toxicogenetics, Leiden, Netherlands, February 4, 2013.


Coordinated response of PARP1 and PARG to facilitate DNA repair pathway choice. FEBS Workshop, Nucleotide Excision Repair and Interstrand Crosslink Repair- From Molecules to Man, Smolenice, Slovakia,
June 9, 2013.


DNA damage-induced PARP1 hyperactivation negatively regulates glycolysis independently of NAD$^+$ depletion. PARP2013 – 19th International Conference on ADP-ribosylation, Quebec City, Canada, September 6-9, 2013.


Proteosome-mediated regulation of base excision repair. V-FARM DNA, V Fundamental Aspects of DNA Repair and Mutagenesis, Sao Paulo, Brazil, October 31-November 2, 2013.

Development of a high throughput Comet platform and its applications. 11th ICEM, Symposium: Comet takes off, Foz do Iguassu, Brazil, November 3-6, 2013.

Exploring the PARP-interactome in the cellular response to genotoxins. 11th ICEM, Symposium: Survival and death pathways triggered by chemotherapeutics, Foz do Iguassu, Brazil, November 3-6, 2013.

Proteosome-mediated regulation of base excision repair. University of Toledo, College of Medicine, Department of Biochemistry & Cancer Biology, November 21, 2013.

Laura Stabile, Ph.D.
Research Associate Professor

Lung Cancer SPORE Investigators’ Annual Meeting, Rockville, MD, July 2013.


West Virginia University, Center for Cancer Cell Biology, Morgantown, WV, February 2015.

Adam Straub, Ph.D.
Assistant Professor

Putting the brakes on nitric oxide signaling: the emerging biology of hemoglobin and methemoglobin reductase in vascular cells. West Virginia University, Morgantown, WV, March 2014.

Mechanisms of heme iron redox regulation in vascular cells. Metals in Biology Symposia, Duquesne University, September 2015.


Ben Van Houten, Ph.D.
Professor

Department of Animal Biology & Comparative Oncology Seminar Series, University of Pennsylvania, PA, April 17, 2013.

Department of Chemistry, University of Illinois, Chicago, IL, October 1, 2013.

Department of Genetics, Erasmus University Medical Center, Rotterdam, Holland, October 7, 2013.

Scientific Crosstalk, 8th International Graduate School of Life Sciences Symposium. “Watching DNA repair, one molecule at a time: The use of single molecule techniques to investigate nanomachines.” Wurzburg, Germany, October 9-10, 2013.

V Meeting in Fundamental Aspects of DNA Repair and Mutagenesis, (Invited Speaker) "Communication between mitochondria and the nucleus - a novel approach to cancer chemotherapy" University of Sao Paulo, SP, Brazil, October 31- November 2, 2013.

11th International Conference on Environmental Mutagens (Invited Speaker) "Watching DNA repair one molecule at a time: UV-DDS stoichiometry, dynamics and implications in xeroderma pigmentosum" and 'Watching DNA repair, one molecule at a time: reconstituting nucleotide excision using quantum-dot labeled proteins." Foz Do Iguassu, Brazil, November 3-8, 2013

Department of Microbiology and Molecular Genetics, University of Vermont, Burlington, VT, November 20, 2013.


DNA Damage, Mutation & Cancer - Gordon Research Conference (Invited Speaker) 'Watching Nucleotide Excision Repair, One Molecule at a Time" Ventura Beach Marriott, Ventura, CA, March 16-21, 2014.

Department of Biochemistry & Molecular Biology, University of Kent, Canterbury, UK, October 17, 2014.

Department of Biochemistry & Molecular Biology, University of Miami, FL, May 30, 2014.

NER damage recognition: Kicking the tires during conformational proofreading. Gordon Research Conferences Mammalian DNA Repair, Ventura, CA, February 8-13, 2015.

College of Medical Science, Experimental and Systems Pharmacology, Washington State University, Spokane, WA, March 9-12, 2015.

Biochemistry and Biophysics, Molecular Biosciences, Washington State University, Pullman WA, March 9-12, 2015.

Physiology and Biophysics, Department of Biochemistry, University of Puerto Rico, San Juan, PR, March 15-19, 2015.

Integrative Structural and Computational Biology, Life Sciences Division, Lawrence Berkeley National Laboratory, Berkeley, CA April 6-9, 2015.

Department of Chemistry and Biochemistry, Florida International University, Miami, FL, May 30- June 2, 2015.


Rudolf Virchow Center for Experimental Biomedicine Julius-Maximilians-University, Wurzburg, Germany, October 10-14, 2015.

Department of Biochemistry & Molecular Biology, University of Kent, Canterbury, UK, October 14-17, 2015.


Jean-Pierre Vilardaga, Ph.D.
Professor

“Non-Canonical Signaling of GPCR.” The Great Lakes GPCR Annual Retreat, Quebec, Canada, 2013.

Keystone Symposia, plenary session GPCRs: Structural Dynamics and Functional Implications, 2014

ENDO 2014 annual meeting, Endocrine Society, 2014.
Rudolf Virchow Center for Experimental Biomedicine, University of Würzburg, Germany, 2014.

Charité Universitätsmedizin Berlin, Symposium on Translational Medicine, Germany, 2014.

University of Montreal, Institute for Research in Immunology and Cancer, Distinguished Scientist lecture, Canada, 2014.

Annual Hawaiian GPCR Workshop, 2015.

Washington University School of Medicine, 2015.

**Dario Vitturi, Ph.D.**

*Research Instructor*


Gordon Research Conference on Nitric Oxide: Nitric Oxide Signaling and Therapeutics, 2015.

**Nobunao Wakabayashi, Ph.D.**

*Assistant Professor*

Gordon Research Conference, Cellular & Molecular Mechanisms of Toxicity, Proctor Academy, Andover, NH August 11-16, 2013.

“The Keap1Nrf2 Signaling Pathway: Role in Disease and Pharmacological Approaches.” The Society for Free Radical Biology and Medicine’s Annual Meeting, Seattle, WA, November 19-23, 2014.

The Environmental Response IV, Sendai, Japan, February 28-March 2, 2014.

**Q. Jane Wang, Ph.D.**

*Associate Professor*


GTC 8th Protein Kinases in Drug Discovery, Boston, MA, 2013.

Institute of Molecular Biology, Three Gorges University Medical College, Yichang, Hubei province, China, 2013.

Translational Cancer Research Forum, Center from Translational Medicine, Key Laboratory of Longevity and Ageing-related Diseases, Ministry of Education, China, and Guangxi Medical University, Nanning, Guanxi, China, 2013.

Session 5-1: Targeting Protein Kinases, BIT's 11th Annual Congress of International Drug Discovery Science & Technology, Therapy and EXPO, Haikou, Hainan, China, 2013.

Purdue University Interdisciplinary Life Science Graduate Program (PULSe) and Department of Biochemistry, Purdue University, 2015.

Department of Physiology & Biophysics, Stony Brook University Medical Center, 2015.

School of Pharmacy, Nanjing University of Chinese Medicine, Nanjing, Jiangsu, China, 2015.

**Stacy Gelhaus Wendell, Ph.D.**

*Research Assistant Professor*

FASEB Conference on Phospholipid Metabolism and Signaling in Cancer & Inflammation, Niagara Falls, NY, 2014.

Nitrate-Nitrite NO Society Meeting, Cleveland, OH, 2014.

17th International Carbonyl Workshop, Skytop, PA, 2014.

**Lin Zhang, Ph.D.**

*Professor*

Roswell Park Cancer Institute, Buffalo, NY, January 18, 2013.

Guangxi Medical University, Nanning, P.R. China, June 3, 2013.

The Third Military Medical University, Research Institute of Daping Hospital, Chongqing, P.R. China, June 5, 2013.

University of Oklahoma School of Medicine, August 5, 2013.

Department of Pharmaceutical Sciences and Experimental Therapeutics, University of Iowa, October 15, 2013.

The 22nd Asia Pacific Cancer Conference, Tianjin, China, October 31-November 2, 2013.

SUNY Stony Brook, December 9, 2013.

University of Minnesota Center for Drug Design January 15, 2014.

Nankai University, Tianjin, China, June 27, 2014.

International Forum on the Frontier Life Sciences, Sichuan University, Chengdu, China, July 5, 2014.

International Symposium on Translational Medicine (ISTM), Guangxi Medical University, Nanning, China, Nov. 19-21, 2014.

St Jude Children's Hospital, Memphis, TN, March 18, 2015,

"Mechanism of Resistance to Targeted Therapies in Colon Cancer Cells" in 2015 Genes &Diseases Symposium, Chongqing, China, April 9-11, 2015.
"Mechanism of cancer cell apoptosis and potential applications" in Second Forum on Advanced and Basic Experimental Techniques on Cell Biology, Chengdu, China, July 21, 2015.

M.D. Anderson Cancer Center, Houston, TX, December 9, 2015.
Teaching Activities
<table>
<thead>
<tr>
<th>MS-1 and MS-2</th>
<th>def of unit</th>
<th>ECURVs</th>
<th>n</th>
<th>ECU</th>
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<td><strong>TEACHING</strong></td>
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<tr>
<td>Lecture</td>
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<td>167.1</td>
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<td>Laboratory</td>
<td>con hrs</td>
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<tr>
<td>Small group - experiential (for physical examination courses only)</td>
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<td>Precepting (for Clinical Experience courses only)</td>
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<td>Other</td>
<td>con hrs</td>
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<td>9.7</td>
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<td>SP session (e.g., Medical Interviewing courses)</td>
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<tr>
<td>Simulator session</td>
<td>con hrs</td>
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<tr>
<td><strong>ADMINISTRATIVE</strong></td>
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<td>Block Director</td>
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<td>Course Director</td>
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<td>AOC/Longitudinal Curriculum Program Director</td>
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<td>Elective or Mini-Elective Course Director</td>
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<td>Course Segment Coordinator</td>
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<tr>
<td>Course Laboratory Segment/Session Coordinator</td>
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**MS-1 and MS-2 Total ECUs**: 613.8

<table>
<thead>
<tr>
<th>MS-3 and MS-4</th>
<th>def of unit</th>
<th>ECURVs</th>
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<th>ECU</th>
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<td><strong>PRECEPTING</strong></td>
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<tr>
<td>Required clerkship</td>
<td># stu/wks</td>
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<tr>
<td>Acting internship clerkship</td>
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<td>0.0</td>
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<tr>
<td>Elective clerkship(s) where enrollment = 1 or more students</td>
<td># stu/wks</td>
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<td>0.0</td>
<td>0.0</td>
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<tr>
<td><strong>TEACHING</strong></td>
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<tr>
<td>Lecture</td>
<td>con hrs</td>
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</tr>
<tr>
<td>Small group (e.g., PBL, conference, workshop)</td>
<td>con hrs</td>
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<tr>
<td>Laboratory</td>
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<tr>
<td>Other</td>
<td>con hrs</td>
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<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>SP session</td>
<td>con hrs</td>
<td>2.0</td>
<td>0.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Simulator session</td>
<td>con hrs</td>
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<td>0.0</td>
<td>0.0</td>
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<td><strong>ADMINISTRATIVE</strong></td>
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<tr>
<td>Clerkship Director for required clerkship</td>
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<td>Clerkship Co-Director for required clerkship</td>
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<tr>
<td>Clerkship Director for selective clerkship</td>
<td>clerkship</td>
<td>20.0</td>
<td>0.0</td>
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<tr>
<td>Clerkship Director for ILS selective (base)</td>
<td>course</td>
<td>30.0</td>
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<td>Clerkship Director for ILS selective (enrollment)</td>
<td>student</td>
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<td>Clerkship Director for elective clerkship</td>
<td>clerkship</td>
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<td>Clerkship Segment/Session Coordinator</td>
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**MS-3 and MS-4 Total ECUs**: 0.0
### Medical Student Program

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<th>Role/Committee</th>
<th>def of unit</th>
<th>ECU Rs</th>
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<th>ECU</th>
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<tbody>
<tr>
<td>Member, Curriculum Committee</td>
<td>comm/yr</td>
<td>20.0</td>
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<tr>
<td>Member, Admissions Committee</td>
<td>comm/yr</td>
<td>75.0</td>
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<td>Member, Promotions Committee</td>
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<tr>
<td>Member, Retention Committee</td>
<td>comm/yr</td>
<td>5.0</td>
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<tr>
<td>Member, Scholarly Project Executive Committee</td>
<td>comm/yr</td>
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<td>Applicant Interviewer</td>
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<tr>
<td>Coordinator, Undergraduate Medical Education Teaching</td>
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<tr>
<td>Chair, Task Force/Work Group/Subcommittee/or other SOM Committee</td>
<td>comm/yr</td>
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<tr>
<td>Member, Task Force/Work Group/Subcommittee/or other SOM Committee</td>
<td>comm/yr</td>
<td>5.0</td>
<td>1.0</td>
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<tr>
<td>Member, Evaluation Sub-Committee</td>
<td>comm/yr</td>
<td>20.0</td>
<td>0.0</td>
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<tr>
<td>Mentoring medical students (e.g., FAST, AOC, or academic advising)</td>
<td>student</td>
<td>2.0</td>
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<tr>
<td>Mentored Scholarly Project (MSP) Mentor</td>
<td>student/yr</td>
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<tr>
<td>Mentored Scholarly Project (MSP) Mentor (partial year)</td>
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<tr>
<td>Summer Research Mentor</td>
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<tr>
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<tr>
<td>Research Mentor, Other (non-MSP)</td>
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**Medical Student Program Total ECU** **147.5**

### Graduate Student Program

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<tr>
<td>Lecture</td>
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<td>Laboratory supervision (e.g., MSTP, Ph.D. &amp; M.Sc.)</td>
<td># stu/wks</td>
<td>1.0</td>
<td>133.0</td>
<td>133.0</td>
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<td>Ph.D. or M.Sc. Mentor</td>
<td>student/yr</td>
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<td>Mentoring other SOM graduate students (e.g., MSTP, Ph.D. or M.Sc.)</td>
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### ADMINISTRATIVE

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<td>Course Co-director</td>
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<td>Journal Club/Seminar Series Program Director</td>
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### OTHER

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## University of Pittsburgh School of Medicine
### Educational Credit Units (AY 14-15)
### Department of Pharmacology and Chemical Biology
### Summary of Faculty ECU's

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**Total Faculty Reporting: 36**

**Total ECU's for Pharmacology and Chemical**

**4545.5**
Teaching Awards

Donald DeFranco, Ph.D.
Professor
Merit Award, Medial Student Research Mentor
Kenneth Schuit Award, Dean’s Award for Master Educator

Post-doctoral Fellows

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<td>Postdoctoral Associate</td>
<td>Jiang</td>
</tr>
<tr>
<td>Jonathan Verrie</td>
<td>Postdoctoral Scholar</td>
<td>Jackson</td>
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<tr>
<td>Avani Vyas</td>
<td>Postdoctoral Associate</td>
<td>Galbiati</td>
</tr>
<tr>
<td>Lei Wang</td>
<td>Postdoctoral Associate</td>
<td>Zhang</td>
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<tr>
<td>Min Wang</td>
<td>Postdoctoral Associate</td>
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<tr>
<td>Rebecca Watters</td>
<td>Postdoctoral Associate</td>
<td>Oesterreic</td>
</tr>
<tr>
<td>Gonghong Yan</td>
<td>Research Associate</td>
<td>Lee</td>
</tr>
<tr>
<td>Yongbei Yu</td>
<td>Visiting Research Associat</td>
<td>de Groat</td>
</tr>
<tr>
<td>Qiangmin Zhang</td>
<td>Postdoctoral Associate</td>
<td>Friedman</td>
</tr>
<tr>
<td>Xuefeng Zhang</td>
<td>Postdoctoral Associate</td>
<td>Altschuler</td>
</tr>
<tr>
<td>Chao-Ming Zhou</td>
<td>Research Associate</td>
<td>Levitan</td>
</tr>
</tbody>
</table>
Faculty Data
# Current Faculty

## Primary Faculty

<table>
<thead>
<tr>
<th>Name</th>
<th>Academic Title</th>
</tr>
</thead>
<tbody>
<tr>
<td>Imad Al Ghouleh</td>
<td>Research Instructor</td>
</tr>
<tr>
<td>Daniel Altschuler</td>
<td>Associate Professor</td>
</tr>
<tr>
<td>Palaniappa Arjunan</td>
<td>Research Instructor</td>
</tr>
<tr>
<td>Jonathan Beckel</td>
<td>Research Instructor</td>
</tr>
<tr>
<td>Alessandro Bisello</td>
<td>Associate Professor</td>
</tr>
<tr>
<td>Dinara Bulgari</td>
<td>Research Assistant Professor</td>
</tr>
<tr>
<td>Eugenia Cifuentes-Pagano</td>
<td>Research Instructor</td>
</tr>
<tr>
<td>Gabor Csanyi</td>
<td>Research Instructor</td>
</tr>
<tr>
<td>W. Chet de Groat</td>
<td>Distinguished Professor</td>
</tr>
<tr>
<td>Donald DeFranco</td>
<td>Professor</td>
</tr>
<tr>
<td>Julie Eiseman</td>
<td>Professor</td>
</tr>
<tr>
<td>Keri Fogle</td>
<td>Research Instructor</td>
</tr>
<tr>
<td>Bruce Freeman</td>
<td>Professor and Chairman</td>
</tr>
<tr>
<td>Peter Friedman</td>
<td>Professor</td>
</tr>
<tr>
<td>William Furey</td>
<td>Professor</td>
</tr>
<tr>
<td>Ferruccio Galbiati</td>
<td>Professor</td>
</tr>
<tr>
<td>Eun-Ryeong Hahm</td>
<td>Research Instructor</td>
</tr>
<tr>
<td>Ryan Hartmaier</td>
<td>Research Instructor</td>
</tr>
<tr>
<td>Jing Hu</td>
<td>Visiting Associate Professor</td>
</tr>
<tr>
<td>Yi Huang</td>
<td>Assistant Professor</td>
</tr>
<tr>
<td>Edwin Jackson</td>
<td>Professor</td>
</tr>
<tr>
<td>Tija Jacob</td>
<td>Assistant Professor</td>
</tr>
<tr>
<td>Yu Jiang</td>
<td>Associate Professor</td>
</tr>
<tr>
<td>Thomas Kensler</td>
<td>Professor</td>
</tr>
<tr>
<td>Nicholas Khoo</td>
<td>Research Assistant Professor</td>
</tr>
<tr>
<td>Joan Lakoski</td>
<td>Professor</td>
</tr>
<tr>
<td>Jack Lancaster</td>
<td>Visiting Professor</td>
</tr>
<tr>
<td>Adrian Lee</td>
<td>Professor</td>
</tr>
<tr>
<td>Edwin Levitan</td>
<td>Professor</td>
</tr>
<tr>
<td>Tatyana Mamonova</td>
<td>Research Instructor</td>
</tr>
<tr>
<td>Carola Neumann</td>
<td>Visiting Associate Professor</td>
</tr>
<tr>
<td>Steffi Oesterreich</td>
<td>Professor</td>
</tr>
<tr>
<td>Roderick O’Sullivan</td>
<td>Visiting Assistant Professor</td>
</tr>
<tr>
<td>Patrick Pagano</td>
<td>Professor</td>
</tr>
<tr>
<td>Michael Palladino</td>
<td>Professor</td>
</tr>
<tr>
<td>Wei Qian</td>
<td>Research Instructor</td>
</tr>
<tr>
<td>Guillermo Romero</td>
<td>Associate Professor</td>
</tr>
<tr>
<td>James Roppolo</td>
<td>Research Assistant Professor</td>
</tr>
<tr>
<td>Francisco Schopfer</td>
<td>Research Associate Professor</td>
</tr>
<tr>
<td>Sruti Shiva</td>
<td>Associate Professor</td>
</tr>
<tr>
<td>Matthew Sikora</td>
<td>Research Instructor</td>
</tr>
<tr>
<td>Shivendra Singh</td>
<td>Professor</td>
</tr>
<tr>
<td>Robert Sobol, Jr.</td>
<td>Associate Professor</td>
</tr>
<tr>
<td>Laura Stabile</td>
<td>Research Associate Professor</td>
</tr>
<tr>
<td>Adam Straub</td>
<td>Assistant Professor</td>
</tr>
<tr>
<td>Kristal Tucker</td>
<td>Research Instructor</td>
</tr>
<tr>
<td>Name</td>
<td>Academic Title</td>
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<td>-------------------------------</td>
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</tr>
<tr>
<td>Ben Van Houten</td>
<td>Professor</td>
</tr>
<tr>
<td>Jean-Pierre Vilardaga</td>
<td>Professor</td>
</tr>
<tr>
<td>Dario Vitturi Iglesias</td>
<td>Research Instructor</td>
</tr>
<tr>
<td>Daniela Volonte-Galbiati</td>
<td>Research Assistant Professor</td>
</tr>
<tr>
<td>Nobunao Wakabayashi</td>
<td>Visiting Research Assistant Professor</td>
</tr>
<tr>
<td>Q. Jane Wang</td>
<td>Associate Professor</td>
</tr>
<tr>
<td>Stacy Gelhaus Wendell</td>
<td>Research Assistant Professor</td>
</tr>
<tr>
<td>Steven Wendell</td>
<td>Visiting Assistant Professor</td>
</tr>
<tr>
<td>Steven Woodcock</td>
<td>Research Instructor</td>
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<tr>
<td>Kunhong (Kevin) Xiao</td>
<td>Associate Professor</td>
</tr>
<tr>
<td>Li Yang</td>
<td>Research Instructor</td>
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<tr>
<td>Cheng Zhang</td>
<td>Visiting Assistant Professor</td>
</tr>
<tr>
<td>Lin Zhang</td>
<td>Professor</td>
</tr>
</tbody>
</table>

**Secondary Faculty**

<table>
<thead>
<tr>
<th>Name</th>
<th>Position</th>
<th>Primary Department</th>
</tr>
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<tbody>
<tr>
<td>Carolyn Anderson</td>
<td>Professor</td>
<td>Radiology</td>
</tr>
<tr>
<td>Christopher Bakkenist</td>
<td>Associate Professor</td>
<td>Radiation Oncology</td>
</tr>
<tr>
<td>Aaron Barchowsky</td>
<td>Professor</td>
<td>Env. Occup. Health</td>
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<tr>
<td>P. Michael Bauer</td>
<td>Assistant Professor</td>
<td>Surgery</td>
</tr>
<tr>
<td>Lori Birder</td>
<td>Professor</td>
<td>Medicine</td>
</tr>
<tr>
<td>Robert Branch</td>
<td>Professor</td>
<td>Medicine</td>
</tr>
<tr>
<td>Timothy Burns</td>
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<tr>
<td>Clifton Callaway</td>
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<tr>
<td>Jun Chen</td>
<td>Professor</td>
<td>Neurology</td>
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<tr>
<td>Nancy Davidson</td>
<td>Distinguished Professor</td>
<td>Medicine</td>
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<tr>
<td>John Fernstrom</td>
<td>Professor</td>
<td>Psychiatry</td>
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<tr>
<td>Gerald Gebhart</td>
<td>Professor</td>
<td>Anesthesiology</td>
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<tr>
<td>Jennifer Grandis</td>
<td>Distinguished Professor</td>
<td>Otolaryngology</td>
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<tr>
<td>Gregg Homanics</td>
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<td>Anesthesiology</td>
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<tr>
<td>Jeffrey Isenberg</td>
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<td>Daniel Johnson</td>
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<td>Valerian Kagan</td>
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<td>Eric Kelley</td>
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<td>Yong Lee</td>
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<td>Chester Mathis</td>
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<tr>
<td>Jerome Parness</td>
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<tr>
<td>James Perel</td>
<td>Professor Emeritus</td>
<td>Psychiatry</td>
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<tr>
<td>Bruce Pitt</td>
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<td>Env. Occup. Health</td>
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<tr>
<td>Richard Steinman</td>
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<td>Medicine</td>
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<td>Dandan Sun</td>
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<td>Neurology</td>
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<tr>
<td>Changfeng Tai</td>
<td>Associate Professor</td>
<td>Urology</td>
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<tr>
<td>Pei Tang</td>
<td>Professor</td>
<td>Anes. &amp; Struct. Biol.</td>
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<tr>
<td>Margaret Tarpey</td>
<td>Professor</td>
<td>Anesthesiology</td>
</tr>
<tr>
<td>Gonzalo Torres</td>
<td>Associate Professor</td>
<td>Neurobiology</td>
</tr>
<tr>
<td>Zhou Wang</td>
<td>Professor</td>
<td>Urology</td>
</tr>
</tbody>
</table>
Name | Position | Primary Department
---|---|---
Wen Xie | Professor | Pharm. Sciences
Yan Xu | Professor | Anesthesiology
Naoki Yoshimura | Professor | Urology

**New Faculty**

Imad Al Ghouleh, Ph.D., Research Instructor  
Keri Fogle, Ph.D., Research Instructor  
Matthew Sikora, Ph.D., Research Instructor  
Kristal Tucker, Ph.D., Research Instructor  
Kunhong (Kevin) Xiao, M.D., Ph.D., Associate Professor  
Li Yang, Ph.D., Research Instructor

**Membership in Professional Societies**

**Bruce Freeman, Ph.D.**

*Professor and Chair*

- American Association for the Advancement of Science  
- American Chemical Society  
- American Heart Association  
- American Physiological Society  
- American Society for Cell and Molecular Biology  
- American Thoracic Society  
- Biochemical Society  
- Society for Free Radical Biology and Medicine

**Imad Al-Ghouleh, Ph.D.**

Research Assistant Professor  
American Heart Association  
Society for Free Radical Biology and Medicine

**Daniel Altschuler, Ph.D.**

*Associate Professor*

The Endocrine Society

**Palaniappa Arjunan, Ph.D.**

*Research Instructor*

American Crystallographic Association  
Pittsburgh Diffraction Society

**Jonathan Beckel, Ph.D.**

*Research Instructor*

Society for Neuroscience  
American Society of Pharmacology and Experimental Therapeutics  
American Physiological Society  
International Continence Society  
American Society for Cell Biology  
American Association for the Advancement of Science
Dr. Alessandro Bisello  
*Associate Professor*  
American Society for Bone & Mineral Research  
Endocrine Society  
American Heart Association

Dinara Bulgari, Ph.D.  
*Research Instructor*  
Society for Neuroscience

M. Eugenia Cifuentes-Pagano, Ph.D.  
*Instructor*  
American Cancer Society

Donald DeFranco, Ph.D.  
*Professor*  
American Association for the Advancement of Science (AAAS)  
Endocrine Society  
Society for Neuroscience

W. Chet de Groat, Ph.D.  
*Distinguished Professor*  
Rho Chi Pharmaceutical Honor Society  
Philadelphia Physiological Society  
American Association for the Advancement of Science  
Sigma Xi  
American Society for Pharmacology and Experimental Therapeutics  
Society for Neuroscience  
Pittsburgh Neuroscience Society  
New York Academy of Sciences  
Urodynamics Society  
International Brain Research Organization  
American Gastroenterological Association  
International Medical Society of Paraplegia  
Society for Basic Urologic Research  
Mid-Atlantic Pharmacology Society  
American Motility Society  
International Continence Society  
The American Autonomic Society  
The Dana Alliance for Brain Initiatives  
International Society for Autonomic Neuroscience (Member of Executive Committee)  
International Spinal Cord Society

Julie Eiseman, Ph.D.  
*Professor*  
American Association for Cancer Research  
FASEB  
American Association for the Advancement of Science  
Society of Toxicology  
SPIE
Keri Fogle, Ph.D.
*Research Instructor*
United Mitochondrial Disease Foundation
American Heart Association

Peter Friedman, Ph.D.
*Professor*
American Physiological Society
American Society for Biochemistry and Molecular Biology
American Society for Bone and Mineral Research
American Society of Pharmacology & Experimental Therapeutics
Endocrine Society
Salt & Water Club
Society of General Physiologists
British Society for Endocrinology
International Bone & Mineral Society

William Furey, Ph.D.
*Professor*
American Crystallographic Association
Pittsburgh Diffraction Society
New York Academy of Sciences
American Association for the Advancement of Science
American Society for Biochemistry and Molecular Biology

Ferruccio Galbiati, Ph.D.
*Professor*
American Society of Pharmacology & Experimental Therapeutics
American Society of Cell Biology
American Physiological Society
American Association for Cancer Research
American Society for Biochemistry and Molecular Biology

Eun-Ryeong Hahm, Ph.D.
*Research Instructor*
American Association for Cancer Research

Jing Hu, Ph.D.
*Visiting Associate Professor*
American Association for Cancer Research

Yi Huang, M.D., Ph.D.
*Assistant Professor*
American Association for Cancer Research
American Association for Advancement of Science

Edwin Jackson, Ph.D.
*Professor*
American Heart Association
American Society for Pharmacology and Experimental Therapeutics
Council for High Blood Pressure Research

**Tija Jacob, Ph.D.**
*Assistant Professor*
American Society for Pharmacology and Experimental Therapeutics
Society of General Physiologists
Society for Neuroscience

**Yu Jiang, M.D., Ph.D.**
*Associate Professor*
American Society for Microbiology
American Society for Pharmacology & Experimental Therapeutics
American Society of Genetics
American Society for Biochemistry and Molecular Biology

**Thomas Kensler, Ph.D.**
*Professor*
American Association for the Advancement of Science
American Association for Cancer Research
Society of Toxicology
American Society for Pharmacology and Experimental Therapeutics
Oxygen Society
American Chemical Society: Division of Chemical Toxicology

**Nicholas Khoo, Ph.D.**
*Research Assistant Professor*
Society for Free Radical Biology and Medicine
Biomedical Engineering Society
South East Lipid Research

**Joan Lakoski, Ph.D.**
*Professor*
American Association for the Advancement of Science
American Society for Pharmacology & Experimental Therapeutics
The Endocrine Society
International Society for Developmental Neuroscience
International Society of Neuroendocrinology
Serotonin Club
Sigma Xi
Society for Neuroscience
American Endocrine Society
Women Executives in Science and Healthcare
National Postdoctoral Association
Association for Clinical Research Training
Society for Clinical and Translational Science

**Jack Lancaster, Ph.D.**
*Visiting Professor*
American Association for the Advancement of Science
American Chemical Society
Adrian Lee, Ph.D.
Professor
American Association for Cancer Research
International Society of IGF Research
The Endocrine Society
American Association for the Advancement of Science
American Society for Molecular Biology and Molecular Biology

Edwin Levitan, Ph.D.
Professor
Society for Neuroscience
American Society of Cell Biologists

Carola Neumann, M.D.
Visiting Associate Professor
Society for Free Radical Biology and Medicine
American Association for Cancer Research

Steffi Oesterreich, Ph.D.
Professor
American Association for Cancer Research
The Endocrine Society
Women in Endocrinology
Women in Cancer Research
American Society for Biochemistry and Molecular Biology
American Society for Pharmacology and Experimental Therapeutics
American Society for Microbiology

Patrick Pagano, Ph.D.
Professor
American Heart Association
Basic Science Council
Circulation Council
Council for High Blood Pressure Research
American Physiological Society
American Association for the Advancement of Science
Society for Free Radical Biology and Medicine
International Society for Free Radical Research

Michael Palladino, Ph.D.
Professor
Genetics Society of America
Society for Neuroscience
American Society for Pharmacology and Experimental Therapeutics

Wei Qian, Ph.D.
Research Instructor
American Association for Cancer Research
Guillermo Romero, Ph.D.
Associate Professor
American Society for Pharmacology and Experimental Therapeutics
Endocrine Society
American Society of Cell Biology

James Roppolo, Ph.D.
Research Assistant Professor
American Association for the Advancement of Science
Society for Neuroscience
The New York Academy of Sciences

Francisco Schopfer, Ph.D.
Research Associate Professor
American Heart Association
Society for Free Radical Biology and Medicine
American Society for Biochemistry and Molecular Biology

Sruti Shiva, Ph.D.
Assistant Professor
Society for Free Radical Biology (Elected to Council 2010)
Nitric Oxide Society
American Heart Association
American Society of Pharmacology and Experimental Therapeutics
American Physiological Society

Matthew Sikora, Ph.D.
Research Instructor
American Association for Cancer Research
American Society for Clinical Oncology

Shivendra Singh, Ph.D.
Professor
American Association for Cancer Research

Robert Sobol, Ph.D.
Associate Professor
American Association for the Advancement of Science
American Association for Cancer Research
American Society for Microbiology
American Society for Cell Biology
Environmental Mutagen Society
American Chemical Society
International Society for Cell & Gene Therapy of Cancer
American Society for Pharmacology and Experimental Therapeutics

Laura Stabile, Ph.D.
Research Assistant Professor
National Lung Cancer Partnership
American Association for Cancer Research
Association for Women in Science
International Association for the Study of Lung Cancer

**Adam Straub, Ph.D.**  
*Assistant Professor*  
American Physiological Society  
Microcirculation Society  
American Heart Association  
Nitric Oxide Society

**Kristal Tucker, Ph.D.**  
*Research Instructor*  
Association for Chemoreception Sciences

**Bennett Van Houten, Ph.D.**  
*Professor*  
Environmental Mutagen Society  
American Association for Cancer Research  
American Chemical Society  
American Society for Pharmacology and Experimental Therapeutics

**Jean-Pierre Vilardaga, Ph.D.**  
*Associate Professor*  
Endocrine Society  
Biophysical Society  
American Society for Pharmacology and Experimental Therapeutics  
American Society for Bone and Mineral Research  
American Society for Biochemistry and Molecular Biology

**Dario Vitturi, Ph.D.**  
*Research Instructor*  
Society for Free Radical Biology and Medicine  
American Heart Association  
Nitric Oxide Society

**Daniela Volonte, Ph.D.**  
*Research Assistant Professor*  
American Society of Cell Biology  
American Association for Cancer Research

**Nobunao Wakabayashi, Ph.D.**  
*Research Assistant Professor*  
Japan Society for Bioscience, Biotechnology and Agrochemistry  
The Molecular Biology Society of Japan  
The Japan Biochemical Society  
American Association for Cancer Research  
American Society for Microbiology
Q. Jane Wang, Ph.D.
Associate Professor
American society for Pharmacology and Experimental Therapeutics
American Association for Cancer Research
American Association for the Advancement of Science

Stacy Gelhaus Wendell, Ph.D.
Research Assistant Professor
American Society for Mass Spectrometry
American Chemical Society
National Postdoctoral Association
Society for Free Radical Biology and Medicine
American Society for Biochemistry & Molecular Biology
American Thoracic Society

Steve Wendell, Ph.D.
Assistant Professor
National Postdoctoral Association

Steven Woodcock, Ph.D.
Research Instructor
American Chemical Society
Society for Free Radical Biology and Medicine

Kevin (Kunhong) Xiao, M.D., Ph.D.
Visiting Associate Professor
Biophysical Society
American Association for the Advancement of Science
American Heart Association/American Stroke Association (professional member)
American Society of Mass Spectrometry

Lin Zhang, Ph.D.
Professor
American Association for Cancer Research
American Association for the Advancement of Science
American Society for Pharmacology and Experimental Therapeutics
Three Year Bibliography
Bruce Freeman, Ph.D.
Professor and Chair


Charles RL, O Rudyk, O Prysyazhna, A Kamyina, J Yang, C Morisseau, BD Hammock, BA Freeman and PE Eaton. Protection from hypertension in mice by the Mediterranean diet is mediated by nitro fatty acid inhibition of soluble epoxide hydrolase. PNAS 111:8167-8172.


Imad Al-Ghouleh, Ph.D.
Research Assistant Professor


Daniel Altschuler, Ph.D.
Associate Professor


**Palaniappa Arjunan, Ph.D.**

*Research Instructor*


**Jonathan Beckel, Ph.D.**

*Research Instructor*


Coffey EE, Beckel JM, Laties AM and Mitchell CH. Lysosomal alkalinization and dysfunction in fibroblasts with the Alzheimer’s disease-linked presenilin-1 A246E mutation can be reversed with cAMP. Neuroscience 263:111-124, 2014.


Dr. Alessandro Bisello
Associate Professor


Dinara Bulgari, Ph.D.
Research Instructor


Li L, Tian X, Zhu M, Bulgari D, Böhme MA, Goettfert F, Wichmann C, Sigrist SJ, Levitan ES and Wu C. Drosophila Syd-1, liprin-α, and protein phosphatase 2A B’ subunit Wrk function in a linear pathway to prevent


**Eugenia Cifuentes-Pagano, Ph.D.**

Research Instructor


**Donald DeFranco, Ph.D.**

Professor


W. Chet de Groat, Ph.D.
*Distinguished Professor


Schwen Z., Roppolo J.R, de Groat W.C., and Tai C.


Julie Eiseman, Ph.D.

Professor


Keri Fogle, Ph.D.

Research Instructor


Peter Friedman, Ph.D.

Professor

Wang B, Yang Y, Liu L, Blair HC, and Friedman PA. NHERF1 regulation of PTH-dependent bimodal Pi


**William Furey, Ph.D.**

*Professor*


**Ferruccio Galbiati, Ph.D.**

*Professor*


Eun-Ryeong Hahm, Ph.D.

Instructor


Ryan Hartmaier, Ph.D.
Research Instructor


Jing Hu, Ph.D.
Visiting Associate Professor


Yi Huang, M.D., Ph.D.
Assistant Professor


Edwin Jackson, Ph.D.
Professor


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Tija Jacob, Ph.D.
Assistant Professor


Brady ML, Moon CE and Jacob TC. Using an alpha-Bungarotoxin Binding Site Tag to Study GABA A Receptor Membrane Localization and Trafficking. J Vis Exp. 2014(85).

Brady ML and Jacob TC. Synaptic localization of alpha5 GABA (A) receptors via gephyrin interaction regulates dendritic outgrowth and spine maturation. Dev Neurobiol 75(11):1241-1251, 2015.

Yu Jiang, Ph.D.
Associate Professor


Luciana Gallo, Yong Liao, Wily Ruiz, Dennis Clayton, Min Li, Yong Jian Liu, Yu Jiang, Mitsunori Fukuda, Gerard Apodaca, and Xiao-Ming Yin. TBC1D9B functions as a GTPase-activating protein for Rab11a in polarized MDCK cells. Mol. Biol. Cell. 25:3779-3797, 2014.


Thomas Kensler, Ph.D.
Professor


Nicholas Khoo, Ph.D.
Research Assistant Professor


Jack Lancaster, Ph.D.
Professor


Adrian Lee, Ph.D.
Professor


Lee AV, Oesterreich S, Davidson NE. MCF-7 cells--changing the course of breast cancer research and care for 45 years. J Natl Cancer Inst. 2015 Mar 31;107(7)

**Edwin Levitan, Ph.D.**

*Professor*


**Tatyana Mamonova, Ph.D.**

*Research Instructor*


Carola Neumann, M.D.  
Visiting Associate Professor


Roderick O'Sullivan, Ph.D.  
Assistant Professor


Steffi Oesterreich, Ph.D.  
Professor


Roderick O’Sullivan
Assistant Professor


Patrick Pagano, Ph.D.
Professor


Michael J. Palladino, Ph.D.  
Professor


Wei Qian, Ph.D.

Research Instructor


Guillermo Romero, Ph.D.

Associate Professor


Romero, G. The role of the cell background in biased signaling. In Biased Signaling in Physiology, Pharmacology and Therapeutics, Brian J Arey (Editor), Academic Press, Waltham, MA, pp. 41-79.

James Roppolo, Ph.D.
Research Assistant Professor


Chen M, C Chermansky, B Shen, JR Roppolo, WC de Groat and C Tai. Electrical stimulation of somatic afferent


Francisco Schopfer, Ph.D.

Research Associate Professor


Matthew Sikora, Ph.D.
Research Instructor


Shivendra Singh, Ph.D.
Professor


Robert Sobol, Ph.D.
Associate Professor


Laura Stabile, Ph.D.
Research Assistant Professor


Adam Straub, Ph.D.
Assistant Professor


**Kristal Tucker, Ph.D.**
Research Instructor


**Ben Van Houten, Ph.D.**
Professor


Jean-Pierre Vilardaga, Ph.D.
Professor


Dario Vitturi, Ph.D.
Research Instructor


Daniela Volonte, Ph.D.
Research Assistant Professor


Nobunao Wakabayashi, Ph.D.
Research Assistant Professor


Q. Jane Wang, Ph.D.
Associate Professor


Stacy Gelhaus Wendell, Ph.D.
Research Assistant Professor


Steven Woodcock, Ph.D.
Research Instructor


Kevin (Kunhong) Xiao, M.D., Ph.D.
Visiting Associate Professor


Cheng Zhang, Ph.D.
Assistant Professor


Lin Zhang, Ph.D.
Associate Professor


Financial Plan
Executive Summary

I. Mission and Goals
The principal goal of the Department of Pharmacology and Chemical Biology is the creation of an intellectual and physical environment in which teaching and research in Pharmacology and Chemical Biology are pursued as one common enterprise. The major responsibilities of the Department are to: (1) educate medical students and physicians in the rationale for drug selection; (2) train contemporary pharmacologists; (3) develop new knowledge in the biomedical sciences; and (4) provide information about existing and emerging drugs to members of the University of Pittsburgh Medical Center, the University of Pittsburgh and the general community.

The philosophical approach of the Department is guided by the view that the field of Pharmacology exhibits a unique combination of characteristics that distinguish it from other basic medical sciences. Pharmacology encompasses a broad range of interests extending from the abstract domain of the physical chemistry of ligand-receptor interactions to the therapeutic use of drugs in patients. Thus, Pharmacology has stronger clinical ties than most other basic science disciplines. In the current revised medical curriculum, the faculty inculcates both core pharmacological principles and places them in the context of specific organ systems and bodily functions. Collectively, we provide a key educational experience to both medical and graduate students. Our faculty members also maintain vigorous research programs that are interactive and interdisciplinary. We have bridged with the Department of Chemistry in a unique and constructive manner through our activities in Drug Discovery.

The goals of the Department are:

- To be one of the top five NIH funded Departments of Pharmacology in the USA.
- To define pharmacological research for the 21st century.
- To improve the presence of Structural Pharmacology and Drug Discovery at the University of Pittsburgh.
- To educate premier future basic researchers, physician-scientists and teachers.
- To enhance the quality of graduate students matriculating and graduating from our PhD program at the University of Pittsburgh.

II. Departmental History and Status:
Major growth in the Department of Pharmacology and Chemical Biology occurred within the last 6 years after a new Chair was recruited and new funds were directed to the Department. As evidenced by the tremendous growth in research support funds, faculty publications, numbers of postdoctoral fellows and the number of members on the Graduate Faculty, we have evolved to become one of the top departments in the country based on our extramural research support and our impact on postdoctoral training. Last year we were ranked eighth in the nation for NIH funding among the more than 100 medical school departments and this year we were ranked seventh. Our Department now views its primary peer programs to be the following institutions: Yale University, University of Michigan, University of North Carolina, University of Pennsylvania, University of Virginia, Emory University, University of Texas Southwestern, University of Washington, Washington University, Vanderbilt University and the Johns Hopkins University.

III. Strengths:
In January 2006, Dr. Bruce A. Freeman was appointed Chair of the Department of Pharmacology and Chemical Biology to lead our strong cohort of well-funded and nationally recognized pharmacologists, cell and chemical biologists and geneticists. The department has about 50 primary faculty and 35 secondary faculty that contribute
to the missions of the department. The members of the Department of Pharmacology and Chemical Biology are highly interactive with frequent co-authorship and co-funding. In spite of challenging times, our faculty members are well-funded, with departmental primary faculty currently receiving $7.4 million in total direct annual costs and $10.3 million in total annual costs. During the past five years, the Department has emphasized cell signaling as an area of excellence. These interests address primary themes such as cancer, cardiovascular, renal and neurobiology. We are now extending this research focus to include two new areas of excellence: drug discovery and structural pharmacology. To supplement our training in these areas, the Department of Pharmacology has an NIH Predoctoral Training Grant in Pharmacological Sciences. The faculty also is well recognized for both their Medical School and Graduate School teaching. The prominence of our faculty members is recognized by their important leadership positions with Centers and Institutes, such as the UPCI, the PINDS, the Drug Discovery Institute, the School of Medicine and the University.

**IV. Initiatives:**
The Department will initiate a search for two or three new faculty members. The Department also intends to continue to replace aging equipment, renovate laboratory space and in this context, relocate faculty within thematic areas. We will also continue developing a strong interdisciplinary Drug Discovery Program. The Department will continue to partner with the emerging programs in Computational and Structural Biology as we emphasize Structural Pharmacology in faculty recruiting processes.

**SWOT Analysis**

**Strengths**
Since Bruce Freeman, PhD assumed the Chair, the Department of Pharmacology and Chemical Biology has grown from 14 tenure-stream faculty to 53 faculty of which 22 are either tenured or in the tenure stream. This growth reflects department-initiated recruitment as well as “opportunistic” recruitments in collaboration with the UPCI that have benefited both the UPCI and the University. Thus, eight of our current tenure stream faculty members are physically located within the UPCI as well as seven non-tenure stream faculty. One tenure stream faculty member is physically located in the Center for Clinical Pharmacology. Virtually all faculty are well-funded, with the Department currently receiving more than $12.9 million in total direct annual costs, approximately $2.0 million of which is co-credited to the UPCI or the Center for Clinical Pharmacology because the faculty members have appointments and space there. Our research success reflects strong independent investigator-initiated research support, a key factor for the development of future thematic research projects. We have already begun to define areas for interactive intra-institution research teams. During the past five years, the Department has emphasized cellular signaling and communication as an area of excellence. These interests are spread over three existing disease/organ areas: cancer, cardiovascular/renal and neurobiology. We are now intending to complement this research focus on cellular signaling with two new areas of excellence: drug discovery and structural pharmacology.

The members of the Department of Pharmacology and Chemical Biology have extensive interactions with other Basic Science Programs. In particular, strong collaborative relationships exist with School of Medicine faculty studying cellular communication and signaling, including faculty from the Departments of Cell Biology and Physiology, Molecular Microbiology and Genetics, Pathology, Neurobiology, and Immunology. Topics of interest range from protein phosphorylation and dephosphorylation, cell cycle checkpoints, G proteins, receptor biology, cell death, pain, combinatorial chemistry, neurotransmitters, channels and redox signalling. Forceful relationships with clinical elements of the Medical Center also exist. These include strong collaborative projects with the Departments of Medicine, Surgery, Anesthesiology, Critical Care Medicine, Pediatrics, Neurology, Urology, Psychiatry and Pathology. The laboratories of members of the Department of Pharmacology and Chemical Biology house advanced fellows from several clinical units: Pulmonary Medicine, Medical Oncology, Surgery, Anesthesiology and Critical Care Medicine. Interactions also exist with the key Centers and Institutes
within the Medical Center and Main Campus including the CNUP, UPCI and the Vascular Medicine Institute. These activities reflect the strong commitment of the Department of Pharmacology and Chemical Biology to engage in translational research and to provide a forum for integrative sciences. The Department considers its role in bridging the basic and clinical sciences of UPMC as a core element of its missions related to fundamental investigation and drug discovery.

Active programs reaching out to the Main Campus have also been instituted. Consequently, there are major collaborations between members of the Department of Pharmacology and the Department of Chemistry. Members of the Department also interact with investigators in the Departments of Environmental and Occupational Health, Biological Sciences and Neuroscience, as well as investigators at Carnegie Mellon University, particularly from the National Science Foundation Center for Fluorescence. Because of this multidisciplinary research activity, the Department took a leadership role in the submission of the multimillion dollar Pittsburgh Molecular Target Laboratory application, which is making Pittsburgh an epicenter for academic drug discovery.

The Department of Pharmacology and Chemical Biology was honored that it was selected to receive an NIH Predoctoral Training Grant in Pharmacological Sciences. This was the only new graduate Training Grant for Pharmaceutical Sciences to be awarded by the NIH in 1994. Moreover, our program was one of only a few recently initiated grants to be renewed for a second cycle. The acquisition of this training grant, which supports four students, was a primary goal of the Department for several years and we are proud to have obtained it.

In addition to their splendid research record, the faculty has displayed outstanding teaching records, both in the Medical School and Graduate School courses. We believe this is due primarily to placing special emphasis on quality teaching and limiting the student interactions of those teachers rated less effective by the students. Our faculty members have also assumed important leadership positions with Centers and Institutes, such as the UPCI, the School of Medicine and the University. To summarize, we have created:

- Strong research activities and NIH grant support
- Interactive faculty
- Interdisciplinary program with the Department of Chemistry
- NCI funded Program Project on Drug Discovery
- Funded NIH Predoctoral Training Grant in Pharmacological Sciences
- NCI funded Specialized Program of Research Excellence in Lung Cancer
- Focus on Cell Communication, Drug Discovery and Structural Pharmacology
- Outstanding teachers of medical and graduate students, e.g. Professor de Groat, who is a five-time winner of the School of Medicine “Golden Apple Award”.

**Weaknesses**

During the past five years tenured and non-tenured faculty left the Department. We expect that one of our prized lecturers, Professor de Groat, will select retirement within the next three years. Thus, the Department must continue to recruit new faculty to ensure quality teaching to medical school students and retain the critical mass required to be among the top five programs nationally. The curriculum for medical students is routinely being reviewed so that we can develop more blueprints for teaching pharmacology to medical students. We are
encouraged by the medical students who realized the importance of strong pharmacological training not only for scoring highly on board examinations but also for treating patients. A basic introductory lecture series on classical pharmacology is currently deemed essential to the current organ system-based training of medical students.

The Department was criticized in the most recent review of its Training Grant that it had too few junior faculty members. To allow the Department to function effectively and to achieve critical mass, additional faculty will be needed.

Currently there are only two program project-type research grants (PO1, P50) within the Department of Pharmacology and Chemical Biology; the national emphasis on specific disease areas lends itself to programmatic efforts and the Department should exploit this. The increased awareness of the productive aspects of linking contemporary chemistry with modern biology also should be an area in which Pharmacology plays a key role. Indeed, we organized a response to an NIH Request for Proposals on Molecular Targets Laboratory, because of the close research links between these two programs. The Department has now focused on Cellular Signaling and Communication as a major theme. We also believe our interest in Drug Discovery and Structural Pharmacology is both timely and institutionally appropriate. In contrast to the Cellular Signaling and Communication, we have not yet reached critical mass in Drug Discovery and Structural Pharmacology. We plan to fortify these areas by recruiting new faculty members in a manner that would complement the academic mission of the University.

There are limited amounts of pharmaceutical research dollars awarded to the Department of Pharmacology and Chemical Biology. The Department has not placed enough emphasis on obtaining funding from pharmaceutical organizations but rather has paid more attention to Federal dollars. We are now placing more focus on the commercial sector to support research, but intellectual property issues and data sharing are still hurdles.

There continues to be a need for capital investment within the Department of Pharmacology and Chemical Biology to replace aging equipment and to advance our depth in new technological capabilities. The rapid advances in new technologies mandate that new investments be made for our faculty and trainees to maintain our national competitive standing. In summary, we need to:

- Maintain critical mass of faculty
- Grow and better integrate space and facilities for research
- Limited financial research support from pharmaceutical and biotechnology firms and budget cuts at the National Institutes of Health

**Opportunities**

During the past four years there has been a remarkable and unprecedented growth in drug discovery and development in the US. Both large pharmaceutical firms and biotechnology companies have invested heavily to exploit this new knowledge. Consequently, major new therapeutic advances directed against important disease groups are now emerging. The completion of the Human Genome Project has increased the number of potential therapeutic targets by more than one order of magnitude. The future challenge will be to identify the drugs that will interact with these emerging biochemical and molecular targets. Because of the unique attributes at the University of Pittsburgh that place Chemistry physically close to Biology, we posit:

- There is a unique opportunity for a few academic institutions to participate and profit from this changing paradigm in drug discovery. The Bayh-Dole Act now allows resourceful Universities the opportunity to replace the income lost from managed care with income derived from its intellectual
property. A strong Department of Pharmacology is a vital component of such an activity.

- There will be a significantly increased industrial and academic need for well-trained graduates from Ph.D. granting programs with a concentration in Pharmacological Sciences and Drug Discovery. A strong Department of Pharmacology and Chemical Biology is a vital component of such an activity.
- Important new knowledge and reagents continue to emerge that are relevant to many biological systems and all aspects of cell communication. A strong Department of Pharmacology and Chemical Biology is a vital component of such an activity.
- Advances in Structural Biology and Bioinformatics should make it possible in the near future to optimize small molecules that are more selective and potent towards their molecular targets. The area of Structural Pharmacology will be grown to be a vital component of the Department of Pharmacology.
- Ph.D., D.M.D. and M.D. students will need to become even more cognizant and thoughtful about the highly selective therapies of the future that may be used based on the genetic profile of each patient. A strong Department of Pharmacology is a vital component of such an activity.

The University of Pittsburgh is uniquely situated to participate in defining future research and graduate education in Pharmacology and in recruiting to its campus some of the best students. The University of Pittsburgh’s advantages are:

- A cohort of dedicated faculty members, who are eager to teach graduate students
- The presence of strong existing programs in neuroscience, virology, tumor immunology, cancer biology, developmental biology, structural biology and computational biology
- The presence of strong clinical programs
- A growing drug discovery enterprise

Barriers

One of the greatest threats to the Department of Pharmacology and Chemical Biology would be to lose its vigor and enthusiasm during the current downward trend in NIH funding. Currently the program is nationally identified as a model of growth. This has helped in the recruitment of new faculty. Nonetheless, other institutions have become eager to develop programs in drug discovery and to enhance their pharmacology departments. We believe it is likely that they will seek to recruit our valuable faculty.

Initiative and Implementation Strategies

- To achieve the overall goal of becoming one of the top three Departments of Pharmacology in the next three years, the Department will recruit new faculty members during the next few years. The Department of Pharmacology and Chemical Biology intends to focus on the three defined areas of research interest previously identified: Cellular Communication, Structural Pharmacology and Drug Discovery. In particular, the Department will continue to partner with the new Drug Discovery Institute, providing unique instrumentation, archived chemical libraries and specialized research services and teaching for members of the University and UPMC.
# University of Pittsburgh School of Medicine
## Statement of Revenues and Expenses – June 30, 2015

<table>
<thead>
<tr>
<th></th>
<th>Hard Money</th>
<th>Self Supporting</th>
<th>Discretionary and Restricted</th>
<th>Total</th>
</tr>
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<tbody>
<tr>
<td><strong>Revenue</strong></td>
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<td>School of Medicine - ECU</td>
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<td>2,543,679</td>
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<td>Direct Grants</td>
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<td>Medical Faculty Fringes</td>
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<td>Staff Fringes</td>
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<td><strong>Subtotal Compensation</strong></td>
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<td>Other Expense</td>
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<td>Transfers (intra-department)</td>
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<td>Transfers (inter-department)</td>
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<td><strong>Subtotal Other Operating</strong></td>
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<td><strong>Stepdown</strong></td>
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### June 30, 2015 Fund Balance
- Restricted Net Activity Year to Date: 393,809
- Prior Year Settlement Transfer: -1,591
- Current Month end Restricted Fund Balance: 4,402,045
- SOM Quasi Endowment Market value – March 2014: 2,146,630
- **Total Available Balances**: 6,548,675