COBRE
SPRING 2019

MUSC
Medical University of South Carolina
Changing What's Possible

SC COBRE IN OXIDANTS, REDOX BALANCE, AND STRESS SIGNALING

MARCH 22, 2019
TIDES OF FOLLY BEACH

COBRE EXECUTIVE ADVISORY BOARD

Kenneth D. Tew, PhD, DSc
Medical University of South Carolina

Franklin G. Berger, PhD
University of South Carolina

Yvonne Janssen-Heininger, PhD
The University of Vermont

Dean P. Jones, PhD
Emory University of Medicine

Garth Powis, D. Phil
Sanford Burnham Prebys Medical Discovery Institute

Peter J. van Bladeren, PhD
Nestlé: Retired Vice President of Regulatory & Scientific Affairs

ABOUT COBRE

We are about half way through the Phase II funding period and the COBRE program is now preparing to entertain that adaptive changes that will be required for transition into the third stage of its development. For this most recent cycle, we continue to bring together junior investigators from disparate backgrounds, but sharing research interests and expertise in oxidants, redox balance and stress signaling at the Medical University of South Carolina. To facilitate development of the Center during the period of 2018-2019, we have supported four individuals and maintained four scientific core facilities. Building the core infrastructure is a prerequisite for engineering longer-term plans of developing a viable Phase III application that focuses only on facilities support for the Center. Our plan continues to be to develop in South Carolina a Center of Excellence in the scientific discipline of redox biology.

Existing infrastructure has continued to provide a successful mentoring environment for target faculty members with research interests that span a number of human pathologies. RO1 successes from recent graduates continue to encourage the present trainee investigators with interests in cancer (Wang, G. & Wang, J.), cardiovascular disease (Angel) and liver disease (Kim). In each instance, the interface of these diseases with oxidant stress, redox homeostasis and stress signaling provides the fundamentals for the programmatic development of the Center. Their projects are substantially supported by four scientific cores in Proteomics, Bioenergetics, Cell and Molecular Imaging and Analytical Redox. Our central hypothesis has not changed and continues to be that redox regulated pathways impact significantly on the pathobiology of diseases such as cancer, aging, diabetes, inflammation and neurodegeneration. Our efforts have been enhanced by recruitment of a new junior investigator and faculty member Dr. Jen Wang) in the Department of Pharmacology in the fourth quarter of 2018.

The administrative core facilitates many functions including business management, faculty development, mentoring, pilot project assessments, program planning and sustainability. We have appointed oversight committees to include Steering, Internal Advisors and External Advisors. This year’s keynote speaker is the newest member of the EAB, Dr. Frank Berger from University of South Carolina who has just completed the third phase of a COBRE in Colon Cancer Biology. He brings extensive experience on how to achieve successful transition to a Phase III program. Our advisory groups contain individuals who have broad scientific expertise in chosen disciplines and also considerable mentoring experience. Future development of the program at MUSC is also presently served by existing financial commitments from the Deans of Medicine and Pharmacy and the Provost’s Office. As we move forward with additional recruitments and supplementation of our core facilities, our goals will continue to include attainment of peer review support for trainee faculty and continued enhancement of the capabilities of the core facilities. We continue to be on track to achieve these endpoints.

OVERVIEW OF THE DAY

***CONTINENTAL BREAKFAST** - 7:30 - 7:55 AM

OPENING REMARKS FROM THE CHAIR:
KENNETH D TEW, PHD, DSC - *COBRE P.I.*

7:55 - 8:00 AM

PEGGI ANGEL, PHD - *COBRE FACULTY*
8:00 - 8:30 AM
8:30 - 8:45 AM - *DISCUSSION*

JEN WANG, PHD - *COBRE FACULTY*
8:45 - 9:15 AM
9:15 - 9:30 AM - *DISCUSSION*

GAVIN WANG, MD, PHD - *COBRE FACULTY*
9:30 - 10:00 AM
10:00 - 10:15 AM - *DISCUSSION*

***BREAK** - 10:15 - 10:45 AM

SEOK-HYUNG KIM, PHD - *COBRE FACULTY*
10:45 - 11:15 AM
11:15 - 11:30 AM - *DISCUSSION*

PILOT AWARD GRANTEES - 11:30 AM - 11:45 AM

SHIKHAR MEHROTRA, PHD
"TARGETING ANTI-OXIDANT CAPACITY OF T CELLS FOR IMMUNOTHERAPY"

JESSICA THAXTON, PHD
TUMOR INDUCED DYSFUNCTION OF T CELL METABOLISM THROUGH ENDOPLASMIC RETICULUM OXIDOREDUCTASE 1 ALPHA

KYU-HO LEE, PHD
"A NOVEL ROLE FOR IRON SULFUR CLUSTER METABOLISM IN CARDIAC DEVELOPMENT."

OVERVIEW OF THE DAY

. . . continued

***LUNCH** - 11:45 AM - 12:00 PM

KEYNOTE SPEAKER: FRANK BERGER, PHD
RESEARCH AND OUTREACH DIRECTOR
COLORECTAL CANCER PREVENTION NETWORK
UNIVERSITY OF SOUTH CAROLINA
"COBRE PHASE 3: THINKING AHEAD"
12:00 - 1:00 PM

LAUREN BALL, PHD - *DIRECTOR OF CORE B*
1:00 - 1:30 PM
1:30 - 1:45 PM - *DISCUSSION*

CRAIG BEESON, PHD - *DIRECTOR OF CORE C*
1:45 - 2:15 PM
2:15 - 2:30 PM - *DISCUSSION*

DANYELLE TOWNSEND, PHD - *DIRECTOR OF CORE E*
2:30 - 3:00 PM
3:00 - 3:15 PM - *DISCUSSION*

JOHN LEMASTERS, MD, PHD - *DIRECTOR OF CORE D*
3:15 - 3:45 PM
3:45 - 4:00 PM - *DISCUSSION*

EXECUTIVE BOARD MEETING
4:00 - 4:30 PM

Peggi Angel, PhD

COBRE Project Director

Assistant Professor

Cell and Molecular Pharmacology
and Experimental Therapeutics

Systems-based analysis of redox activity in aortic valve stenosis

Oxidative stress plays a key but unknown role initiating fibrocalcific aortic valve disease (FAVD). Aortic valve stenosis (AVS) is a major cardiovascular disease diagnosed in more than 5 million Americans each year with main risk factors of male, age, and high fat diet. AVS progresses as obstructive narrowing of the aortic opening due to increased stiffening of the aortic valve leaflets. Stiffening is caused by deregulated extracellular matrix (ECM) with excess glycosylation, fibrosis and calcification of the leaflets; this leads to cardiac failure. Although clinically significant, AVS has no treatment other than surgical valve replacement. Molecular processes involved activation of the disease are poorly understood. In particular, while glycoprotein signaling is a hallmark of FAVD, the functional and structural changes of the N-glycans that control glycoprotein signaling are completely unknown. Our current experiments are revealing, for the first time, a unique interaction between oxidative stress signaling, N-glycosylation, and endothelial mesenchymal transformation. We propose that a “glyco-redox” response is a main contributor to changes in glycoprotein signaling that results in the initial deregulation of the valvular structure. Our central hypothesis is that the redox response of early FAVD induces aortic endothelial reprogramming to a mesenchymal phenotype by regulation of structural and functional changes in N-glycosylation; this leads to the rapid deregulation of ECM glycoproteins that triggers the degenerative cycle of valvular disease. This is the first time that a glyco-redox response has been explored as a determinant that initiates aortic valve disease. We anticipate that understanding the role of oxidative stress on functional N-glycosylation could lead to new therapies that inhibit disease progression and, potentially, diagnostic tests that monitor progression of the disease for intervention before symptoms of cardiac failure. Successful completion of this investigation relies on the unique resources and interdisciplinary team from the Medical University of South Carolina’s COBRE in Oxidants, Redox Balance and Stress Signaling and could not be accomplished without use of these resources. The proposed studies will have a broad impact on the field of valve biology by contributing to a large knowledge gap on the role of early oxidative stress in glycoprotein regulation of the valvular structure.

Haizhen "Jen" Wang, PhD

COBRE Project Director

Assistant Professor

Cell and Molecular Pharmacology
and Experimental Therapeutics

Targeting CDK6 for T-cell acute lymphoblastic leukemia (T-ALL) therapy

Cyclin D3/CDK6, the major cyclin D/CDK in T-cell acute lymphoblastic leukemia (T-ALL), are highly expressed and activated in T-ALL. Targeting cyclin D3/CDK6 is a promising therapeutic strategy as cyclin D3/CDK6 inhibition not only prevents leukemia cell proliferation, but also induces cancer cells into apoptosis.

We have shown that cyclin D3/CDK6 phosphorylate and inactivate phosphofructokinase, platelet (PFKP) and pyruvate kinase muscle isozyme M2 (PKM2) in T-ALL cells to regulate cellular ROS levels by shunting glycolysis to the pentose phosphate pathway (PPP) and serine synthesis pathway. Recent studies from my group indicate that cyclin D3/CDK6 regulate the cytosol-to-nuclear translocation of PFKP to potentially affect T-ALL invasion. Leukemia cell invasion is a critical step in T-ALL dissemination. And dissemination of leukemia cells is one of the major indicators of poor prognosis in clinics. Following this direction, we are interested to figure out: how cyclin D3/CDK6 regulates PFKP nuclear translocation, and what extra-cellular signals induce PFKP cytosol-nuclear translocation to mediate its nuclear function.

On the other hand, T cells in tumor microenvironmentnbplays critical functions in affecting tumor
progression. Our recent study found that CDK6 plays an important role in regulating cellular ROS in T cells, and controlling the population of T cells. To this end, we will demonstrate the detail mechanisms of CDK6 in regulating T cell population to affect tumor progression. In addition, my group are interested in optimizing the therapeutic effect by specific targeting CDK6, other than both CDK4 and CDK6, in certain types of cancer, such as T-ALL.

Gavin Wang, MD, PhD

COBRE Project Director

Assistant Professor

Pathology and Laboratory Medicine

The Response of Cancer Stem Cells to Oxidative Stress

There is a growing body of evidence showing that cancer stem cells (CSCs) are resistant to current anticancer therapies, as demonstrated by the fact that they are enriched in residual tumors after chemotherapy and/or radiation. These observations underscore a critical need for the development of novel therapeutic drugs that can more effectively eradicate CSCs in cancer treatment. Induction of oxidative stress has been found to be an important mechanism of action for many anticancer agents such as cisplatin and radiation; however it is largely unknown how CSCs respond to oxidative stress. Our studies show that the levels of reactive oxygen species (ROS) are markedly lower in breast CSCs (BCSCs) than that in non-cancer stem cells (NCSCs). Exposure of breast cancer cells to sub-lethal doses of hydrogen peroxide (H2O2) resulted in a dose-dependent increase of the epithelium-specific antigen (ESA)+/CD44+/CD24- subpopulations, a known phenotype for BCSCs. Despite BCSCs could survive low doses of H2O2 treatment, they lost the ability to form tumor spheres and failed to generate colonies as determined by mammosphere-formation and clonogenic assays, respectively. Mechanistic studies revealed that H2O2 treatment causes a marked increase of senescence-associated β-galactosidase activity, but only minimal apoptotic cell death in BCSCs. Furthermore, H2O2 triggers p53 activation and up-regulates p21 expression, indicating a role for the p53/p21 signaling pathway in oxidative stress-induced senescence in BCSCs. Collectively, these results demonstrate that the maintenance of a lower level of ROS is essential for CSCs and H2O2-mediated BCSC loss of function is attributable to oxidative stress-induced senescence. These new findings suggest that ROS-generating drugs may have the therapeutic potential to eliminate drug-resistant CSCs via induction of cellular senescence. To further delineate the mechanisms by which CSCs maintain lower levels of ROS and evade oxidative stress-induced apoptosis, using label-free proteomic approaches we have recently discovered a number of redox-modulating proteins that are preferentially over-expressed in BCSCs as compared to NCSCs. Our ongoing studies are focused on characterizing the functional roles of these differentially expressed proteins in modulating the response of BCSCs to oxidative stress. Successful completion of this project may have a high impact in the field of developmental cancer therapeutics as a better understanding of the molecular processes governing CSC’s response to oxidative stress will substantially facilitate the discovery and development of innovative therapeutic approaches to target drug-resistant CSCs for cancer treatment.

Seok-Hyung Kim, PhD

COBRE Project Director

Assistant Professor

Medicine

Mitochondrial Disease Associated Mutations as novel Genetic Risk Factors to Develop Advanced Fatty Liver Disease

Alcoholic and non-alcoholic fatty liver diseases (FLD) are common chronic liver disorders. A substantial proportion of FLD patients develop an inflammatory response with hepatitis, leading to fibrosis, cirrhosis, liver failure and/or hepatocellular cancer. Increase of oxidative stress caused by accumulation of reactive oxygen/nitrogen species (ROS/RNS) and mitochondrial dysfunction has been implicated in the pathogenesis of both non-alcoholic and alcoholic FLD. The effect of obesity and environmental factors such as alcohol and high-fat diet in FLD are relatively well established. However, genetic determinants of FLD and advanced FLD, including steatohepatitis, fibrosis and hepatocellular carcinoma have not been systematically investigated. Previously, to identify novel genetic variants involved in liver disease, we performed forward genetic screening of zebrafish mutants. Among 19 homozygous mutants identified from the screen, four novel mutants showed hepatomegaly, steatosis, and hepatocellular injury as well as increase of mitochondrial oxidative stress. We hypothesize that haploinsufficiency of those genes, causing liver phenotype in homozygous mutant condition may induce hepatic steatosis and injury phenotype in adult fish. Further, the liver phenotype may be exacerbated by a high fat diet and chronic ethanol consumption.

Our lab is currently focusing on impact of electron transfer flavoprotein alpha (etfa) haploinsufficiency in development of alcoholic fatty liver disease. The electron transfer flavoprotein (ETF) complex plays key role in mitochondrial fatty acid beta-oxidation activity as well as redox balance by controlling redox status of mitochondrial FAD/FADH2. We recently found evidence of liver injury and steatosis in etfa+/- adult zebrafish, and further analyses suggested that hepatic injury was from increased mitochondrial oxidative stress. In addition, decrease of FAD seen in the liver of etfa+/- mutant can be further exaggerated by chronic ethanol consumption. Thus, we predict that riboflavin treatment may reverse the deleterious effect of etfa haploinsufficiency by restoring FAD in the liver as well as exacerbated liver injury during ethanol treatment.

Lauren Ball, PhD

Director of CORE B: Proteomics

Associate Professor

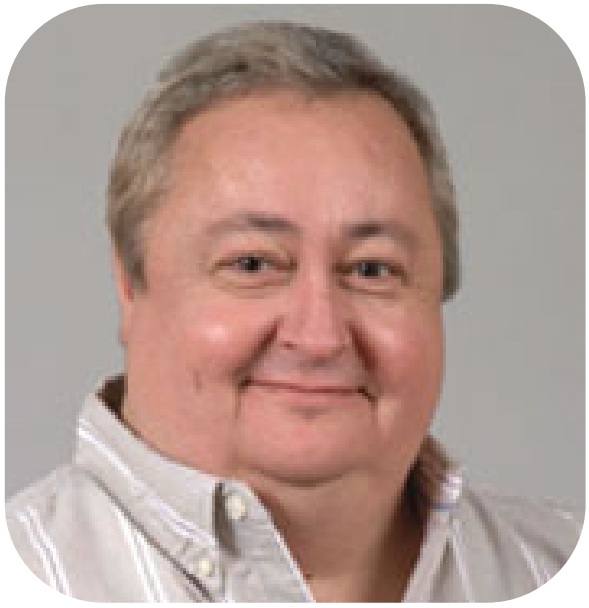
Cell & Molecular Pharmacology & Experimental Therapeutics

The goal of the SC COBRE in Oxidants, Redox Balance and Signaling Proteomics Core is to provide state-of-the-art LC-MS/MS instrumentation, expertise, and training for comprehensive proteomic analyses to advance the research endeavors and career development of junior investigators with interests in redox signaling. Dedicated core personnel assist with experimental design, method development, data acquisition, and computational analysis for protein characterization and quantitative proteomic applications using metabolic labeling (SILAC), isobaric tandem mass tagging (TMT), and label free quantitation (MaxQuant LFQ). The core has facilitated characterization of redox- and drug-sensitive post-translational modifications of cysteine (sulfenic, sulfinic, sulfonic acid, S-glutathionylation, disulfide bonds); arginine (glycation by methylglyoxal, dihydroxyimidazolidine); tyrosine (nitrated and crosslinked), and lysine (acetylation, ubiquitin). COBRE investigators have sequenced putative biomarker peptides discovered by MALDI-tissue imaging mass spectrometry and identified differentially expressed or post-translationally regulated proteins following genetic manipulation of anti-oxidant enzymes, drug treatment, or disease. Quantitative proteomic methodology has been established to determine the effects of altered redox-balance on differential protein abundance in FAC-sorted cell populations, primary cells, and exosomes. COBRE investigators have also utilized quantitative proteomics to elucidate the mechanism of action of HDAC inhibitors and identify the targets of drugs identified in phenotypic screens.

With the recent acquisition of a ThermoScientific Fusion Lumos Orbitrap ETD/UVPD MS, improvements in quantitative proteomics employing tandem mass tagging (TMT SPS-MS3), metabolic labeling (NeuCode SILAC), and label free proteomics (Boxcar) will be available. New complementary peptide fragmentation modes are available (EThcD, UVPD) to facilitate confident sequencing of challenging post-translational modifications. Integration of novel data acquisition modes will facilitate much needed global, targeted proteomics for verification of endogenously post-translationally modified peptides in vivo (MaxQuant.Live). The core has also acquired a Xevo TQ-S triple quadrupole MS which will expand our capabilities to include absolute quantitation and multiple reaction monitoring (MRM) assays for targeted proteomics.

In addition to these new discovery and targeted proteomic capabilities, the core is currently optimizing redox proteomics approaches for selective-reduction and enrichment of differentially modified cysteine residues. We will continue to assist COBRE investigators and members of the COBRE Redox Center with customized method development as needed to advance their research endeavors.

Reminder: Please acknowledge NIH support of the Proteomics Core (P20 GM103542-Proteomics Core) and shared instrumentation grants (S10 OD010731-Orbitrap Elite ETD MS awarded in 2012 and S10 OD025126 Orbitrap Fusion Lumos ETD/UVPD MS awarded in 2018).



Craig Beeson, PhD

Director of CORE C: Bioenergetics Profiling

Professor

Drug Discovery and Biomedical Sciences

Bioenergetics Profiling and Cellular Redox

Cellular redox species are produced directly or indirectly via bioenergetic metabolic reactions. Although the leak of electrons is often cited as the primary source of superoxide and hydroxy radicals, the leak is only a small contributor to the many different species involved in cellular redox reactions. Indeed, the fundamental basis of bioenergetics involves the oxidation (loss of electrons) of reduced nutrients and subsequent production of metabolites with a range of redox potentials (i.e., nicotinamides, reduced/oxidized metalloproteins, and thiol-containing species with varied redox potentials. Profiling the flux of these various metabolites through their attendant metabolic reactions is fundamental to any studies aimed at understanding cellular redox reactions.

The Bioenergetics Core provides several of the leading technologies that enable researchers to quantify the fluxes of these metabolic reactions in cells, tissues, organoids and small animal models such as zebrafish embryos and nematodes. The central technologies include high resolution respirometry using the XF technology from Seahorse Biosciences/Agilent. Dr. Beeson was involved in the original design of the XF technology profiles extracellular fluxes of oxygen, lactate and CO2 as the samples are interrogated with pharmacological and/or genetic manipulations. The instrumentation utilizes 96-well microplates to provide sufficient sample numbers to provide robust, statistically validated flux profiles of glycolysis, mitochondrial respiration, fatty acid oxidation, glutamine utilization and other related metabolic processes. Rapid, high-throughput imaging optimized to the XF plate architecture provides normalization of cell/tissue numbers, health, and other.

Isotopomer analyses of Krebs cycle intermediates and their byproducts utilizes LC-GC/MS. Typical 13C-labels at particular positions of, for example glucose, are introduced to cells or tissue small samples are periodically quenched, lysed and derivatized to volatile esters. The key advantage is that only the low molecular weight acids readily enter the gas phase for analyses that enable determination of enrichment of the 13C at positions that reveal the fluxes through specific pathways and/or the conversions to key species such as 2-hydroxy-glutamic acid – and oncometabolite produced by a mutated form of isocitrate dehydrogenase seen in many tumors that have escaped from therapeutic pressures. The oncometabolites affect epigenetic mechanisms that regulate tumor cell growth and proliferation.

A key feature of the core is that Dr. Beeson and his team also provide extensive training, data analyses support and aid in experimental design – it is fundamentally a collaborative unit.

Danyelle Townsend, PhD

Director of CORE E: Analytical Redox Biology

Associate Professor

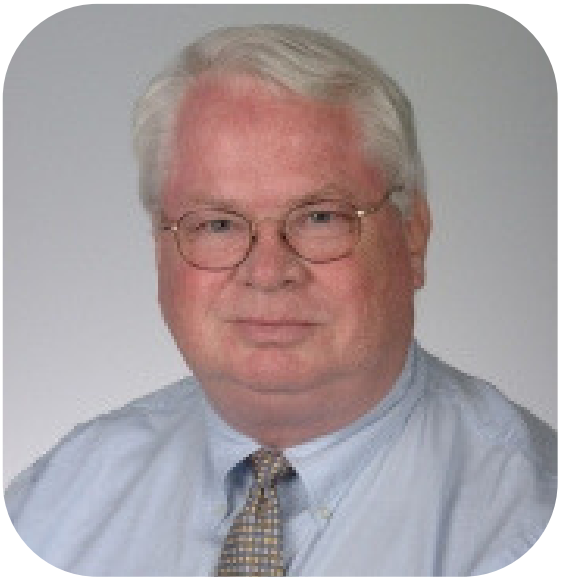
Drug Discovery & Biomedical Science

Analytical Redox Biochemistry Core

Understanding the complexities of redox mediated signaling events requires a multidisciplinary approach. The SC COBRE in Oxidants, Redox Balance and Stress Signalling has assembled a cohort of promising junior faculty with expertise in relevant biomedical model systems. Analytical biochemistry specific to the detection and quantification of redox sensitive molecules and coordinate protein changes that drive homeostasis is a unique niche fulfilled by the Analytical Redox Biology Core (ARBC).

The primary objective of the Core is to provide comprehensive analytical redox biochemistry methods and mentoring support for the COBRE junior faculty with the goal to advance their research endeavors, publications and fundability. The specific aims of the ARBC are: 1) Provide ROS /RNS identification and quantification using state-of-the-art techniques; 2) Perform quantitative analysis of ROS/RNS (redox molecules and metabolites), including those associated with calcium mobilization and changes in energy metabolism; 3) Provide expertise and technology for in depth biochemical analysis of thiol-centered enzyme activities and define protein:protein interactions.

Since oxidative (nitrosative) stress often is associated with a conditional increase in antioxidant protection, the Core has established methods to detect and measure various antioxidant enzyme activities as a function of oxidant stress/antioxidant protection equilibrium. Comprehensive analysis of redox status also includes measurement of intracellular GSH, GSSG, protein surface and “buried” thiols utilizing both endpoint and/ or real-time kinetic measurements with millisecond resolution. In complex studies of redox signaling, certain protein:protein interactions appear to be redox dependent and attributed to post-translational modifications, including S-nitrosylation and S-glutathionylation. The ARBC has developed fluorescent labeling and FRET analysis to evaluate redox dependent protein:protein interactions with subsequent in silico molecular modeling using ZDOCK, GOLD Suite (v 5.2) software. Collectively, these technologies will provide a multidisciplinary approach to advance the understanding of redox mediated signaling events specific to the model systems presented by the junior faculty in their research.



John Lemasters, MD, PhD

Director of CORE D: Cell and Molecular Imaging

Professor & GlaxoSmithKlein Distinguished Endowed Chair of Drug Discovery and Biomedical Sciences

Biochemistry and Molecular Biology

CMI Core D provides COBRE investigators access and assistance for high end laser scanning confocal/multiphoton/super-resolution microscopy and related imaging techniques. Core D houses five confocal/multiphoton systems: 1) a state-of-the-art Zeiss LSM 880 NLO Quasar confocal/multiphoton microscope with a Fast Airyscan super-resolution detector; 2) an Olympus FV1200 silicone oil optics multiphoton microscope configured especially for intravital imaging; 3) an Olympus FV10i LIV confocal microscope with water immersion optics for live cell imaging; 4) a Zeiss LSM 510 META laser scanning confocal microscope for general purpose imaging of live and fixed specimens; and 5) a BD CARV II disk-scanning confocal microscope for video rate “real-time” confocal imaging. Major recent upgrades include Fast Airyscan for the Zeiss LSM 880 for super-resolution imaging, a near UV laser upgrade for the Zeiss LSM 510 to permit imaging of DAPI and other blue-emitting fluorophores, and the acquisition of Bitplane Imaris software for 3- and 4-D visualization of image data sets. The Core together with the Drug Discovery Core at MUSC is currently preparing a Shared Instrumentation Grant (SIG) S10 application to secure a high content automated imager for submission in May, 2019, which would greatly help the drug discovery efforts of several current and graduated COBRE investigators.

In 2018-2019, CMI held workshops on newly acquired and emerging technologies, including “Zeiss Celldiscoverer 7: A New Platform for Automated Live Cell Imaging for Drug Discovery” (March 14, 2018), “Simplifying High Content Analysis for Cell Biology and Drug Development” (April 16), Imaris Image Visualization & Analysis Workshop” (June 4-5), “Zeiss Fast Airyscan Super-Resolution Microscopy” (November 26), “Streamline Imaging and Analysis with a Single Platform” (December 20), and “Current Landscape of Biological Testing using High Content Analysis” (January 31, 2019).

To ensure that COBRE members are expertly trained in cell and molecular imaging, especially as it relates to oxidative stress and redox signaling biology, the Core also organizes a biennial Charleston Workshop on Light Microscopy for the Biosciences (LMB), which will next be held June 9-14, 2019. Participation of COBRE investigators and their personnel is given priority. The 7th LMB Workshop will provide a solid introduction to the concepts and practical applications of light microscopy relevant to modern cell and molecular biology. Students will have opportunities for extensive hands-on experience with state-of-the-art equipment for optical imaging, digital image processing, fluorescence, confocal/multiphoton microscopy and super-resolution microscopy guided by experienced academic and commercial faculty. Commercial faculty representing leading microscope manufacturers will make available for students use of the latest and most advanced instrumentation for light microscopy, image detection and computerized image analysis. The keynote speaker and invited faculty for the workshop will be Dr. Eeva-Liisa Eskelinen of the University of Turku, who will give presentations on “Correlative Light-Electron Microscopy (CLEM)”.

By providing sophisticated imaging technologies, expertise and training, CMI promotes the success of the individual COBRE projects and also provides training and assistance to junior investigators studying oxidative stress and stress signaling related to the overall theme of the COBRE.

NOTES

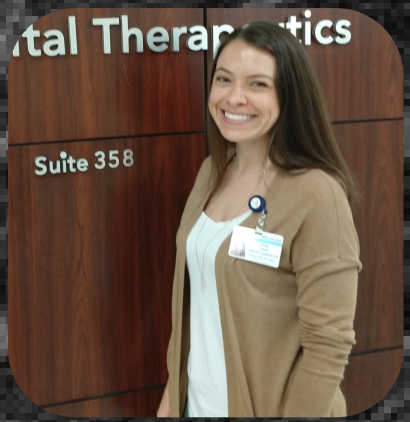
NOTES

COBRE SPRING 2019

THANK YOU FOR JOINING US

COBRE Administrative Core

Deza Finn
Administrative Manager



Carly Lauer
Grants Coordinator

Caitlin Myers Hernandez
Administrative Coordinator