MOLECULAR MECHANISMS OF DISEASE

Predoctoral Training Program

Annual Symposium

April 19, 2023

8:00 AM - 5:30 PM AKRS Champions Club

Program Contacts

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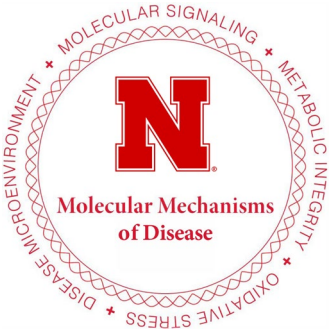

MOLECULAR SIGNALING
METABOLIC INTEGRITY
OXIDATIVE STRESS
DISEASE MICROENVIRONMENT
Molecular Mechanisms
of Disease

Training Program Summary

The goal of the program is to develop outstanding new scientists who work in collaborative multi-disciplinary teams to research disease mechanisms using quantitative approaches that ultimately yield tangible strategies for prevention and therapy. Participants are highly qualified, motivated students with a strong interest in the underlying causes of human disease. The program provides a framework to encourage all students to assemble a broad knowledge base, actively seek research collaborations, produce an outstanding record of original published research, and develop presentation, proposal-writing, and leadership skills that position them for future excellence as independent researchers focused on mechanisms of disease progression.

Administrative Advisors

Ron Yoder
Associate Vice Chancellor, Institute of Agricultural and Natural Resources

Bob Wilhelm
Vice Chancellor for Research and Economic Development

Daniel Duncan
Executive Director of Nebraska Innovation Campus

Debra Hope
Dean of Graduate Studies

Oleh Khalimonchuk
Susan Rosowski Professor of Biochemistry

Angie Pannier
Maxcy Professor of Agriculture and Natural Resources, Biomedical Engineering

Jennifer Wood
Professor, Animal Science

David Hage
James Hewett University Professor of Chemistry

External Advisors

Leslie Poole
Professor, Biochemistry, Wake Forest School of Medicine

Melanie Simpson
Professor and Head**,** Molecular and Structural Biochemistry, North Carolina State University

Molecular Mechanisms of Disease Mentoring Faculty

Matt Andrews, School of Natural Resources
Induction and maintenance of hibernation in mammals

Jennifer Auchtung, Food Science and Tech
Microbiome-targeted therapies

Donald Becker, Biochemistry
Proline metabolism in health and disease, enzyme mechanisms of substrate channeling

David Berkowitz, Chemistry
Chemical biology and synthetic organic chemistry

Nicole Buan, Biochemistry
Contribution of methanogenic bacteria to gut function

James Checco, Chemistry
Neuropeptides and peptide hormones as cell-to-cell signaling molecules

Lindsey Crawford, Biochemistry
Virus manipulation of the human immune system

Andrea Cupp, Animal Science
Role of VEGF in testis morphogenesis

Eric Dodds, Chemistry
Chemical glycobiology

Liangcheng Du, Chemistry
Discovering new anti-infective agents from *Lysobacter*

Catherine Eichhorn, Chemistry
RNA structural dynamics and intermolecular recognition

Rodrigo Franco, Vet and Biomedical Science
Signaling mechanisms and neurotoxicity

Jiantao Guo, Chemistry
Protein tyrosine-O-sulfation, molecular interaction probes

David Hage, Chemistry
Chromatographic automation of immunoassays

Ed Harris, Biochemistry
Liver endothelial scavenger receptor function; systemic clearance of heparin

Tomas Helikar, Biochemistry
Dynamics of molecular and cellular mechanisms, Immune systems

Nicole Iverson, Biol Systems Engineering
Delivery, monitoring and analysis of in vivo nanoparticles as biological sensors

Oleh Khalimonchuk, Biochemistry
Mitochondrial oxidative function and protein homeostasis in health and sickness

Sri Kidambi, Chem & Biomol Engineering
Tissue engineering, stem cells

Forrest Kievit, Biol Systems Engineering
Nanoparticle-based delivery vehicles for brain cancer and brain injury treatments

Jaekwon Lee, Biochemistry
Mechanistic insights in homeostatic copper acquisition and cellular metal detoxification

Colin Meiklejohn, Biological Sciences
Genetic basis of speciation and regulatory evolution between nuclear and mitochondrial genomes

Angela Pannier, Biol Systems Engineering
Non-viral gene delivery, synthetic extracellular matrices, protein-cell adhesion

Kurt Piepenbrink, Food Science and Tech
Glycoproteins in bacterial pathogenesis

Robert Powers, Chemistry
NMR metabolomics

Amanda Ramer-Tait, Food Science and Tech
Host-microbial interactions in inflammatory bowel disease

Wayne Reikhof, Biological Sciences
Lipid distribution and storage mechanisms

Seung-Hyun Ro, Biochemistry
Sestrins and TORC signaling in metabolism

Xinghui Sun, Biochemistry
Long non-coding RNA regulation of gene expression in obesity and diseas e

Molecular Mechanisms of Disease Mentoring Faculty-Continued

Jay Storz, Biological Sciences
Functional genetic variation in high-altitude mammals and birds, hypoxia adaptation

Alex Vecchio, Biochemistry
Catalytic asymmetric hydroboration and “green chemistry” synthesis

Rebecca Wachs, Biol Systems Engineering
Tissue engineering and biomaterials-based therapeutics

Mark Wilson, Biochemistry
Redox regulation of DJ-1 function

Jennifer Wood, Animal Science
Steroid hormone sensing and signaling

Dustin Yates, Animal Science
Metabolic fetal programming related to maternal stress

Limei Zhang, Biochemistry
Structural biology, Mycobacterium tuberculosis infectivity

Schedule of Events

Wednesday, April 19, 2023

University of Nebraska AKRS Champions Club

8:00-9:00 AM POSTER SET UP AND BREAKFAST

9:00-9:15 AM WELCOME AND OPENING REMARKS

Dr. Donald Becker, Program Director

9:15-10:15 AM MORNING PLENARY PRESENTATION

Combining mass spectrometry and nanodiscs to investigate membrane protein-lipid interactions

Michael Marty, Ph.D. Associate Professor
Department of Chemistry & Biochemistry
University of Arizona

10:15-11:10 AM CURRENT TRAINEES

Sydney Caparasao
PARSING THE ROLE OF MECHANICS AND INFLAMMATION IN CHRONIC LOW BACK PAIN

Daisy Guiza Beltran
A SHARED ANCHOR ON PRIMARY SIGMA FACTOR SIGA BY THE WHIB-LIKE PROTEINS

Noelle Waltenburg
DESIGN OF SYNTHETIC RECEPTOR ACTIVITY MODIFYING PROTEINS AS CHEMICAL PROBES TO STUDY G PROTEIN – COUPLED RECEPTORS

11:15-12:00PM SESSION I POSTER VIEWING (ODD NUMBER POSTERS)

12:00-1:00PM LUNCH

1:00-2:00 PM AFTERNOON PLENARY PRESENTATION

Using protein engineering to develop next-generation vaccines for highly infectious pathogens such as SARS-CoV-2, influenza, or HIV

Dr. Mihai Azoitei, Ph.D. Professor
Duke University School of Medicine

2:00-2:40 PM AFTERNOON TRAINEE TALKS: SET 1

Brandon McDonald
ROLE OF OXIDATIVE AND ELECTROPHILIC STRESS ON CELLULAR METABOLISM AND ANTIOXIDANT DEFENSE FOLLOWING TRAUMATIC BRAIN INJURY

Amanda Maliva
FUNCTIONAL GENOMICS OF PHOSPHATIDYLCHOLINE BIOSYNTHESIS IN *SACCHAROMYCES CEREVISIAE*

2:40-2:55 PM LIGHTNING TALKS

Selected from Poster Abstracts

2:55-3:40 PM SESSION II POSTER VIEWING (EVEN NUMBER POSTERS)

3:40-4:40 PM AFTERNOON TRAINEE TALKS: SET 2

Cole Dolamore
NEW STRUCTURAL APPROACHES TO UNDERSTANDING METHYLGLYOXAL DETOXIFICATION

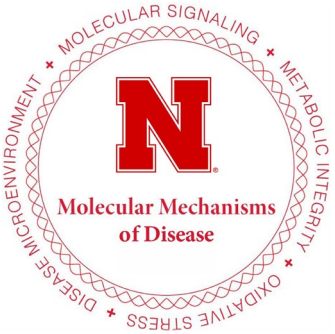
Tyrell Rossman
EXPLORATION OF CYSTEINE MODIFICATIONS IN HUMAN Δ^1-PYRROLINE-5-CARBOXYLATE REDUCTASE 2 (PYCR2)

Josh Loecker
ADVANCING THE AUTOMATED CREATION OF CONSTRAINT-BASED METABOLIC MODELS OF THE HUMAN IMMUNE SYSTEM

4:40-5:00 PM AWARD PRESENTATIONS

Dr. Donald Becker, Program Director

5:00-5:30 PM POSTER TAKE DOWN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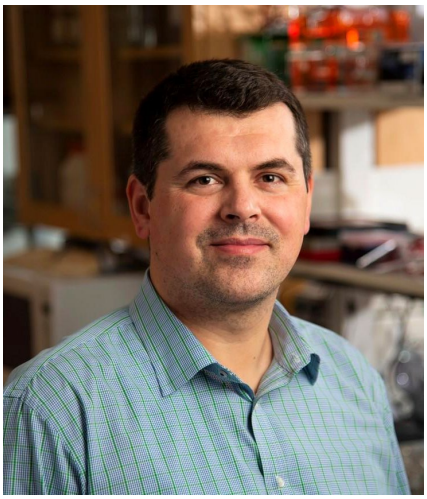
PLENARY SPEAKER PROFILES

Dr. Michael Marty is Assistant Professor of Chemistry & Biochemistry at The University of Arizona. Dr. Marty's research centers on developing new technologies to study interactions at biological membranes, with a special focus on combining lipoprotein nanodiscs and native mass spectrometry. One aspect of his research focuses on characterizing membrane protein-protein and membrane protein-lipid interactions. Membrane proteins are important drug targets, and the Marty lab seeks to understand how lipids influence membrane proteins important in cancer. The second aspect of Prof. Marty's research focuses on assembly of antimicrobial peptide complexes, which also show anticancer activity. By characterizing the biophysics of peptide interactions with and within lipid bilayers, they seek to shed light on the mechanisms of anticancer peptide toxicity and selectivity.



Dr. Mihai Azoitei is an assistant professor in Medicine and Pathology at the Duke University School of Medicine, and a member of the Duke Human Vaccine Institute. Research in Dr. Azoitei's Lab is focused on two synergistic areas that are critical for the development of vaccines against highly infectious pathogens: 1) rational immunogen design for the induction of broadly neutralizing antibodies and 2) determining the biophysical properties of antigens that lead to B cell activation and development for antibody secretion.

MMoD Trainee Abstracts

Morning Session

PARSING THE ROLE OF MECHANICAL AND INFLAMMATORY NEURONAL ION CHANNELS IN CHRONIC LOW BACK PAIN

Sydney Caparaso[1], Rebecca Wachs[1]

[1]Department of Biological Systems Engineering, University of Nebraska-Lincoln

Chronic low back pain (cLBP) is a leading source of disability worldwide. Patients with cLBP often have discs characterized by nerve innervation, inflammation, and altered mechanics. Nociceptors (pain-sensing neurons) present various ion channels to sense inflammatory and mechanical stimuli. Repeated stimulation can lead to neuronal sensitization, or maladaptive changes resulting in both an upregulation of ion channels and a lowered firing threshold. Neuronal sensitization can be induced in vitro with chemical sensitizers that are natively present in inflamed discs, such as nerve growth factor (NGF). However, no studies have examined which factor (inflammation or mechanics) is the predominant driver of sensitization. We **hypothesize** that an interaction between inflammation and mechanical loading drives nerve stimulation ultimately leading to neuronal sensitization and cLBP. Therefore, the **objective** of this work is to 1) screen agonists and antagonists for mechanically and inflammatory sensitive ion channels using in vitro rodent neuron culture, and 2) probe changes in inflammatory and mechanical ion channels in response to native sensitizers present in the disc. Using calcium imaging, we identified two compounds to uniquely agonize inflammatory and mechanically sensitive ion channels: capsaicin and GSK1016790A, respectively. To induce neuronal sensitization, DRGs were incubated with NGF to elicit an inflammatory response. Future work will screen agonists and antagonists for additional ion channels implicated in LBP. Experiments are ongoing to characterize mechanical and inflammatory sensitive neuronal ion channels in discs and pain cells from painful animals from our cLBP rodent model. Ultimately, we aim to translate these in vitro data to antagonize ion channels in our established rat model of CLBP to parse the differential roles of mechanics and inflammation to improve clinical translation.

A SHARED ANCHOR ON PRIMARY SIGMA FACTOR SIGA BY THE WHIB-LIKE PROTEINS

Daisy Guiza Beltran[1], Magdaléna Horová[1], Huey-Xian Wong[1], Li-Mei Zhang[1,2]

[1]Department of Biochemistry and Redox Biology, University of Nebraska-Lincoln

[2]Nebraska Center of Integrated Biomolecular Communication, University of Nebraska-Lincoln

WhiB-like (Wbl) proteins are a unique group of iron-sulfur cluster ([4Fe4S])-containing transcription factors exclusive to actinobacteria. They play crucial roles in the virulence, survival, and propagation of Mycobacterium tuberculosis (Mtb), the causative agent of tuberculosis. Seven Wbl proteins (WhiB1-WhiB7) are found in Mtb with diverse regulatory roles. All Mtb Wbl proteins except for WhiB5

have been shown to interact with the conserved region 4 of the primary sigma factor ($SigAr_4$) in RNA polymerase holoenzyme. Our recent structural and biochemical analyses indicate that two Wbl proteins, WhiB1 and WhiB7, bind to the same site on $SigAr_4$ unexpectedly through tight hydrophobic interactions involving several conserved aromatic residues of the Wbl proteins and His516 of $SigAr_4$. Substitution of these aromatic residues in either WhiB1 or WhiB7 at the molecular interface abolishes their binding to $SigAr_4$. Here, we hypothesize that all Mtb Wbl proteins share a similar molecular interface when in complex with $SigAr_4$, based on the primary sequence analysis and structural modeling. Using the site-directed mutagenesis and the in vitro protein-protein interaction assays, we confirm that all Mtb Wbl proteins, including WhiB5, bind to the same site on SigA centered on His516, and the conserved aromatic residues within the Fe-S cluster binding pocket are required for SigA binding. The results from this study will offer insights into how the Wbl proteins are utilized in Mtb to orchestra gene expression in response to environmental cues in the host.

DESIGN OF SYNTHETIC RECEPTOR ACTIVITY MODIFYING PROTEINS AS CHEMICAL PROBES TO STUDY G PROTEIN – COUPLED RECEPTORS

Noelle Waltenburg[1], James W. Checco[1], and Robert Powers[1]

[1]Department of Chemistry, University of Nebraska-Lincoln

Receptor-activity modifying proteins (RAMPs) are small transmembrane proteins associated with class B G protein-coupled receptors (GPCRs). These RAMPs aid in receptor trafficking, ligand recognition, and act as GPCR messengers. These proteins are composed of three domains: a three-helix extracellular domain (ECD), transmembrane domain (TM), and an inner membrane domain that aid in its functionality. We hypothesize studies using truncations of RAMP called synthetic RAMPs (sRAMPs) can be used to test their ability to act as allosteric modulators to GPCRs to study RAMP+GPCR interactions. Studies of RAMP+GPCR interactions have been focused almost exclusively on the interaction of RAMPs with the calcitonin-like receptor, where they play major role in determining ligand specificity. However, many other GPCRs are known to associate with RAMPs, including the secretin receptor, vasoactive intestinal peptide/pituitary adenylate cyclase-activating peptide receptor, parathyroid hormone receptors, and glucagon receptors. The roles RAMPs play in the signaling of receptors other than CLR are not well understood. Therefore, understanding the molecular mechanisms by which RAMPs modulate signaling for these GPCRs will increase our understanding of diseases caused by RAMP dysregulation including asthma and cardiovascular disease. Current work has been done to derive sRAMPs using both bacteria expression and Fmoc solid-phase peptide synthesis (SPPS). Full three-helix ECD has been successfully expressed in *E. coli* and purified. Additionally, synthesis of truncated ECD composed of helices two and three and the TM have successfully been completed using SPPS. Current work is underway to optimize both ECD refolding using disulfide bond formation and SPPS analogue yields. Upon completion of sRAMPs, these analogues will be tested to determine their capability to act allosteric modulators in CHO-K1 cells by measuring cAMP accumulation through ligand stimulation. Successful sRAMPs that can modulate activity can be used as chemical probes to identify additional RAMP+GPCR interactions and better understand diseases that are linked to this system.

MMoD Trainee Abstracts

Afternoon Session #1

ROLE OF OXIDATIVE AND ELECTROPHILIC STRESS ON CELLULAR METABOLISM AND ANTIOXIDANT DEFENSE FOLLOWING TRAUMATIC BRAIN INJURY

Brandon McDonald[1], Forrest Kievit[1]

[1]Department of Biological Systems Engineering, University of Nebraska-Lincoln

Traumatic brain injury (TBI) is a leading cause of injury-related morbidity and mortality worldwide. Primary impact forces trigger a cascade of biochemical dysfunction, led by an aberrant production of reactive oxygen species (ROS) and lipid peroxidation products (LPOx), corresponding to oxidative and electrophilic stress, respectively. Indeed, following TBI, both ROS and LPOx activate the Nrf2 antioxidant defense pathway and contribute to impaired autophagic flux, through inducing lysosomal membrane permeabilization (LMP) and activating the mechanistic target of rapamycin (mTOR). Additionally, antioxidant defense and autophagy may be linked through transcriptional regulation of the lncRNA, MALAT1. However, the respective roles of oxidative and electrophilic stress on cellular metabolism and antioxidant defense following TBI is unclear. We have previously shown that the administration of antioxidant nanoparticles (ANPs) may alleviate secondary damage following TBI, due to their ability to neutralize oxidative and/or electrophilic stress following injury. To this end, we have developed a series of ANPs, including oxygen reactive polymers (ORPs) and neuroprotective copolymers (NPCs), that contain either thioether or thiol residues to neutralize ROS and LPOx, respectively. Thus, our project goal is to identify how the alleviation of oxidative and/or electrophilic stress via ANPs impacts the molecular and transcriptional changes associated with antioxidant defense and autophagy following TBI. We hypothesize that NPCs will provide a greater therapeutic effect following injury and impart neuroprotection through the amelioration of LMP, downregulation of MALAT1, and/or inhibition of mTORC1. Preliminary Western blot analysis following controlled cortical impact in mice suggests NPCs may acutely alleviate autophagic substrate accumulation and restore Nrf2 activation. Future work will examine differences between ANP formulation to determine the respective role of ROS and LPOx on pathophysiological dysfunction.

FUNCTIONAL GENOMICS OF PHOSPHATIDYLCHOLINE BIOSYNTHESIS IN *SACCHAROMYCES CEREVISIAE*

Amanda Maliva [1], Wayne Riekhof[1]

[1]School of Biological Sciences, University of Nebraska-Lincoln

Phosphatidylcholine (PtdCho) is the most abundant phospholipid in most eukaryotic membranes, and its synthesis is critical for maintaining membrane integrity and biophysical properties. The genes encoding enzymes of PtdCho biosynthesis and their regulation are largely known, but a precise description of the molecular mechanisms of PtdCho regulation and other lipid biosynthesis pathways remains incomplete. Genetic screening on the Saccharomyces cerevisiae knockout mutant collection using the Phosphatidylethanolamine (PtdEtn) methyltransferase inhibitor 2-Hydroxyethylhydrazine (2-HEH) identified potential regulators among processes that interact genetically with the methylation pathway of PtdCho biosynthesis. 2-HEH functions by selectively targeting and inhibiting the methylation pathway enzymes pem1/cho2 and pem2/opi3, through incompletely understood actions. S. cerevisiae gene deletion mutants were identified that exhibit 2-HEH sensitivity and genetically interact with the methylation pathway. Of 4800 strains tested, 410 experienced complete loss of growth, of which 371 encoded functional genes and the remainder “dubious” genes that often overlap or are very close in position to functional genes. Genetic analysis narrowed the list to 21 candidate genes that are negative genetic interactors when combined with pem1Δ and pem2Δ alleles. This gene set was especially enriched in functions relating to the cell membrane as well as nuclear function. Addition of extracellular choline or lyso-Phosphatidylcholine recovers 2-HEH-mediated cell death due to loss of methylation pathway-derived PtdCho biosynthesis, suggesting that PtdCho produced by either the Kennedy or Acyltransferase pathways can maintain necessary cellular PtdCho levels in the absence of the methylation pathway. These results collectively confirm the use of 2-HEH as a specific chemical inhibitor of the methylation pathway. This research will further the knowledge surrounding genetic involvement in PtdCho biosynthesis, and has potential applications in the creation of antifungal drug targets and treatment of pathogenic fungi.

MMoD Trainee Abstracts

Afternoon Session #2

NEW STRUCTURAL APPROACHES TO UNDERSTANDING METHYLGLYOXAL DETOXIFICATION

Cole Dolamore[1], Kara Zielinski[2], Lois Pollack[2], Dr. John Termini[3], Dr. Jiusheng Lin[1], David Berkowitz[1], Dr. Mark Wilson [1]

[1]University of Nebraska - Lincoln

[2]Cornell University

[3]Beckman Research Institute of City of Hope

Glycation stress occurs when cells accumulate covalent damage to macromolecules from the electrophilic compounds glyoxal and methylglyoxal, which are produced by central metabolism and contribute to various diseases. The dominant method for removing methylglyoxal and glyoxal from cells is the two-enzyme glutathione-dependent system comprising glyoxalase I and II (GLO I and GLO II). The recent discovery of a glutathione-independent glyoxalases/deglycases has added a new biochemical pathway for MG detoxification. All known glutathione-independent glyoxalases are in the DJ-1 superfamily, and yet fundamental aspects of their substrate, mechanism, and physiological relevance are intensely debated. We have used time-resolved mix-and-inject serial crystallography (MISC) to directly observe formation of a catalytic intermediate of methylglyoxal and the human protein DJ-1. The type of crystallographic data generated in this experiment are challenging to process and thus we applied new processing software that produced electron density maps of excellent quality. The observation of a methylglyoxal-cysteine adduct in the DJ-1 active site is inconsistent with a popular but controversial deglycase model for DJ-1 action and instead supports a model where DJ-1 is a slow glyoxalase acting directly on methylglyoxal.

EXPLORATION OF CYSTEINE MODIFICATIONS IN HUMAN Δ1-PYRROLINE-5-CARBOXYLATE REDUCTASE 2 (PYCR2)

Tyrell Rossman[1], Donald Becker[1]

[1]Department of Biochemistry, Redox Biology Center, University of Nebraska-Lincoln, NE.

Humans have three different PYCR isoforms; PYCR1, PYCR2, and PYCRL which all catalyze the NAD(P)H-dependent reduction of L-P5C into proline. PYCR1 and PYCR2 are found in the mitochondrion while PYCRL is found in the cytosol. PYCR1 and PYCR2 share 85% sequence identity and are active as dimers. Oxidative PTMs of Cys are an important redox signaling mechanism that impacts different cellular processes such as energy metabolism, apoptosis, and

insulin signaling. Cysthiols react with reactive oxygen species (e.g., H2O2) to form Cys-SOH (sulfenic acid) or Cys-SO2H (sulfinic acid), and with electrophilic compounds such as malondialdehyde. The mitochondrion is a major source of reactive oxygen species and the reactivity of the sulfur in Cys is higher in the alkaline environment of the mitochondria, thus increasing the likelihood of oxidative Cys PTMs in mitochondrial proteins. Oxidized Cys can be reduced back to the thiol form via glutathione/thioredoxin systems. The central hypothesis of this research is that PYCR1 and/or PYCR2 are regulated via PTM of key Cys residues. PYCR1 and PYCR2 contain four and six cysteine residues, respectively. The critical role of PYCR1 and PYCR2 in mitochondrial redox metabolism suggests Cys residues in PYCR1/2 could be susceptible to oxidation. The oxidative modification of Cys residues is proposed to down-regulate PYCR1/2 activity, and thus the proline cycle.

ADVANCING THE AUTOMATED CREATION OF CONSTRAINT-BASED METABOLIC MODELS OF THE HUMAN IMMUNE SYSTEM

Josh Loecker[1], Tomas Helikar[1]

[1]Department of Biochemistry, University of Nebraska-Lincoln, Lincoln, NE

Integrating multi-omics datasets to identify potential drug targets through metabolic modeling can be difficult without advanced tooling. I have been improving and advancing a software, Constraint-based Optimization of Metabolic Objectives (COMO), a user-friendly, jupyter-notebook-based pipeline that integrates multi-omics processing, context-specific metabolic model creation, gene knock-out simulations, and drug targeting/repurposing. COMO is installed as a Docker container and provides documentation in the browser-based environment and the online GitHub page. Bulk and single-cell RNA-seq, microarray, and proteomics can be integrated to create context-specific metabolic models. Creating a computational model provides an easy and low-cost alternative for predicting repurposable drugs through its use of public, open-source databases and software. This pipeline was used to create a model of naive B cells; simulation and analysis predicted 25 and 23 metabolic drug targets against rheumatoid arthritis and systematic lupus erythematosus, respectively. These targets were validated through literature research. COMO can be used to construct a model for any cell or tissue type and identify drug targets for any human disease. The use of this pipeline can greatly improve the health of the global community through cost-effective, high-confidence drug targets to pursue in preclinical and clinical studies.

Poster Presentations

1) The Gut Microbiota Modulates the Severity of Experimental Autoimmune Myocarditis

Xu Shi[1], Paul Velander[1], Robert Schmaltz[1], Jeff Price[1], Amanda Ramer-Tait[1]

[1]University of Nebraska-Lincoln, Lincoln, NE.

2) Investigating the Effects of the Infant Probiotic Bififdobacterium infantis and Human Milk Oligosaccharides on the Severity of peanut allergy

Morgan Cade[1], Tasneem Ali[1], Anthony Juritsch[2], Kristin Beede[2], Robert Schmaltz[2], Bethany Henrick[3], Amanda Ramer-Tait[2]

[1]School of Biological Sciences, University of Nebraska-Lincoln, Lincoln, NE.
[2]Department of Food Science and Technology, University of Nebraska-Lincoln, Lincoln, NE.
[3]Evolve Biosystems, Davis, CA.

3) Proteomics & Metabolomics Facility: Nebraska Center for Biotechnology

Sophie Alvarez[1], Mike Naldrett[1], Anne Fischer[1], Lori Loucks[1.]

[1]University of Nebraska-Lincoln, Lincoln, NE.

4) Evaluation of Commensal E. coli Outer Membrane Vesicles as Bioactive Vehicles for Oral Gene Delivery

Kari Heck [1], Amanda E. Ramer-Tait [2], Angela K. Pannier [1]

[1]Department of Biological Systems Engineering – University of Nebraska-Lincoln, Lincoln, NE.
[2]Department of Food Science and Technology – University of Nebraska-Lincoln, Lincoln, NE.

5) Identifying Genes Involved in Pyrroline-5-Carboxylate Transport in Saccharomyces Cerevisiae

Oseeyi Daudu[1], Lu Zhang[1], and Donald Becker[1]

[1]Department of Biochemistry, Redox Biology Center, University of Nebraska-Lincoln, Lincoln, NE.

6) Understanding the Stabilin Receptor Mediated Endosomal Escape Mechanism of Phosphorothioate Antisense-Oligonucleotide

Ekta Pandey[1], Edward Harris[1]

[1]Department of Biochemistry, University of Nebraska-Lincoln, Lincoln, NE.

7) Multifaceted Mitochondrial Ion Homeostasis Factor Mdm38/ LETM1

Iryna Bohovych[1], Oleh Khalimonchuk[1,2,3]

[1]Department of Biochemistry, University of Nebraska-Lincoln, Lincoln, NE.
[2]Nebraska Redox Biology Center, University of Nebraska-Lincoln, Lincoln, NE.
[3]Fred & Pamela Buffett Cancer Center, University of Nebraska Medical Center, Omaha, NE.

8) CAR-Signaling Resistance Exacerbated by Matrin-3 Deficiency Reveals Protective Roles of Matrin-3-CAR Axis in Nonalcoholic Fatty Liver Disease

Xiao Cheng[1], Vijaya Bhaskar Baki[1], Matthew Moran[1], Baolong Liu[2], Jiujiu Yu[2], Qingsheng Li[3], Xinghui Sun[1,4]

[1]Department of Biochemistry, University of Nebraska - Lincoln, Beadle Center, 1901 Vine St, Lincoln, Nebraska 68588, USA;
[2]Department of Nutrition and Health Sciences, University of Nebraska - Lincoln, 230 Filley Hall, Lincoln, Nebraska 68583-0922, USA;
[3]Vascular Biology Center, Medical College of Georgia, Augusta University, Augusta, GA 30912, USA; Department of Pharmacology and Toxicology, Medical College of Georgia, Augusta University, Augusta, GA 30912, USA;
[4]Nebraska Center for the Prevention of Obesity Diseases through Dietary Molecules, University of Nebraska - Lincoln.

9) Development of DJ-1 Affinity Microcolumns Utilizing Protein Entrapment

Jacob Jones[1], David S. Hage[1], Mark Wilson[2]

[1]Department of Chemistry, University of Nebraska-Lincoln, Lincoln, NE
[2]Department of Biochemistry, University of Nebraska-Lincoln, Lincoln, NE

10) Inhibition of Neddylation in the Vascular Endothelium Causes Reductive Stress and Death of Mice

Vijaya Bhaskar Baki[1], Xiao Cheng[1], Jiyao Zhu[1], Fatema Yeasmin Tanni[1], Rachelle Nelson[1], Huabo Su[2], Xinghui Sun[1,3]

[1]Department of Biochemistry, University of Nebraska - Lincoln, Beadle Center, 1901 Vine St, Lincoln, Nebraska 68588, USA;

[2]Vascular Biology Center, Medical College of Georgia, Augusta University, Augusta, GA 30912, USA; Department of Pharmacology and Toxicology, Medical College of Georgia, Augusta University, Augusta, GA 30912, USA;

[3]Nebraska Center for the Prevention of Obesity Diseases through Dietary Molecules, University of Nebraska - Lincoln.

11) Identification of Cell-Cell Signaling Peptides Within the Central Nervous System of Thirteen-lined Ground Squirrels During Hiber

Somayeh Mousavi[1], James Checco[1]

[1]Department of Chemistry, University of Nebraska-Lincoln, Lincoln, NE

12) Dietary Fiber from Sorghum Flour Protects Mice Harboring Human Gut Microbiotas Against Chemically-Induced Colitis

Anthony F. Juritsch[1], Kristin Beede[1], Morgan Cade[1], Sukaina al-Hamedi[1], Dulcie Achuleta[2], Qinnan Yang[1], Robert Schmaltz[1], Jeff Price[1], Devin Rose[1], Stephen Kachman[1], Scott Sattler[3], Andrew Benson[1], Amanda Ramer-Tait[1]

[1]University of Nebraska-Lincoln, Lincoln, Nebraska;

[2]Nebraska Wesleyan University, Lincoln, Nebraska;

[3]United States Department of Agriculture – Agriculture Research Service (USDA-ARS), Lincoln, Nebraska

14) The Role of AAA-ATPase ATAD3A in Regulating Mitochondrial Architecture and Its Implications in Disease

Alondra Torres[1], Oleh Kahlimonchuk[1,2]

[1]Department of Biochemistry, University of Nebraska-Lincoln, Lincoln, NE

[2]Nebraska Redox Biology Center, University of Nebraska-Lincoln, Lincoln, NE

15) Analysis of ALS-linked Mutations in the Mitochondrial Protease OMA1

Polash Ghosh[1], Gunjan Purohit[1], Martonio Ponte Viana[1], Colton Roessner[1], Oleh Khalimonchuk[1,2,3]

[1]Department of Biochemistry, University of Nebraska-Lincoln, NE
[2]Nebraska Redox Biology Center, University of Nebraska-Lincoln, Lincoln, NE
[3]Fred & Pamela Buffett Cancer Center, University of Nebraska Medical Center, Omaha, NE

16) Development of a Chromatographic Displacement Assay for Measurement of Trace Amounts of Free Drug Fractions

Kyei Isaac[1], David S. Hage[1]

[1]Department of Chemistry, University of Nebraska-Lincoln, Lincoln, NE

17) Thermodynamic Studies of Leonardite Humic Acid - Drug Binding

Kyungah Suh[1], David S. Hage[1]

[1]Department of Chemistry, University of Nebraska-Lincoln, Lincoln, NE

18) LACTB Deletion Alters Mitochondrial Metabolism and Impacts Intermembrane Contact Sites

Gunjan Purohit[1], Oleh Kahlimonchuk[1,2]

[1]Department of Biochemistry, University of Nebraska-Lincoln, Lincoln, NE
[2]Nebraska Redox Biology Center, University of Nebraska-Lincoln, Lincoln, NE

19) Identification of a Small Signal Molecule Involved in the Interaction Between the Biocontrol Agent Lysobacter sp. 3655 and Fungal Pathogens

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20) N6-methyladenosine Dynamics and Differential Methylation of Maternal and Zygotic mRNAs During the Early Stages of the Maternal to Zygotic Transition

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21) Combining L. taiwanensis and G. urolithinfaciens Decreases Body Weight Gain and Increases Lean Mass in Mice

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22) Multisite Interactions of Thiazolidinedione Drugs with Human Serum Albumin by Using High-performance Affinity Microcolumns

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23) Investigating the Mechanism of L-Proline Excretion Under Hypoxic Conditions

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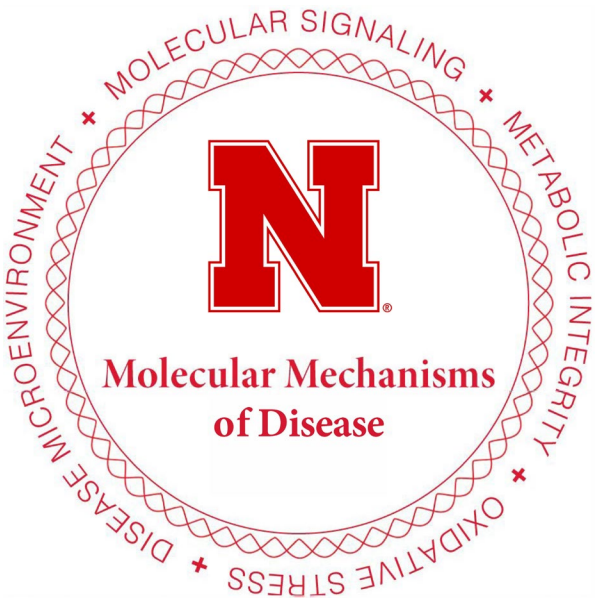

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