**Poster Session 1 - July 26th 1:00-2:15**

**1. Name:** Hailey Davis

**Institution:**Turtle Mountain Community College

**Email:** lbest@restel.com

**Research area:** Other

**Title:** Maternal Risk of Pre-eclampsia and the *CRP* Variant, rs2808629

**Authors:** Hailey Davis,1 McKayla Gourneau,1 Tyler Parisien,1 Lyle G. Best,1

1Turtle Mountain Community College

**Abstract:** Background: The etiology of pre-eclampsia (PE) is unknown; but it is accepted that normal pregnancy represents a distinctive challenge to the maternal immune system. C-reactive protein (CRP) is a prominent component of the innate immune system; and we previously reported an association between PE and three *CRP* single nucleotide polymorphisms (SNP). Our aim was to explore the effects of an additional *CRP* variant.

Methods: This study genotyped 131 cases of PE and 267 controls, from an American Indian community. Genotyping of the CRP variant, rs2808629, was conducted by real-time PCR using a TaqMan assay. Potential association was analyzed by chi square and logistic regression.

Results: Genotypic distribution satisfied Hardy-Weinberg equilibrium and neither dominant nor recessive models were significant for association by chi square testing. Univariate analysis of dominant, recessive, or additive genetic models indicated no significant evidence of association between rs2808629 and PE. Nulliparity and body mass index (BMI) were significantly associated with PE, OR 2.96 (95% CI 1.92-4.56) p<0.001 and OR 1.053 (95% CI 1.023-1.085) p<0.001 respectively.

Conclusion: There is no apparent association between rs2808629, approximately 8Kb 5' of *CRP* and PE in this American Indian cohort. Associations were clearly identified between nulliparity and BMI.

**2. Name:** Ayotunde Paul Ikujuni  **Institution:** University of Kansas  **Email:** apikujuni@ku.edu **Research area:** Other

**Title:** Characterization of Folding Stages of TolC, an Outer-Membrane Component of Antibiotic Efflux

**Abstract:** Overexpression of tripartite efflux pump systems in gram-negative bacteria has been reported to be linearly correlated with antibiotic resistance in clinical isolates as well as in selected mutants of different bacterial pathogens. The outer membrane subunit of the efflux pump, TolC, is difficult to produce at a sufficient yield for biochemical characterization. We have developed a method of refolding by which TolC remains folded in SDS-PAGE, retains binding to an endogenous ligand, and recapitulates the known crystal structure by single particle cryoEM analysis. Using circular dichroism, thermal denaturation, and proteinase K digestion as well as assessment of insertion into biological membranes, we characterized the folding stages of TolC including periplasmic intermediates. We anticipate that our findings will help in developing better efflux pumps inhibitors.

**3. Name:** Carrie Olson-Manning **Institution:** Augustana University **Email:** colsonmanning@augie.edu **Research area:** Other

**Title:** Analysis of the leaf metabolome of Arabidopsis thaliana mutation accumulation lines reveals association of pleiotropy and fitness consequences

**Abstract:** Understanding the mechanisms by which mutations affect fitness and the distribution of mutational effects are central goals in evolutionary biology. Mutation accumulation (MA) lines have long been an important tool for understanding the effect of new mutations on fitness, phenotypic variation, and mutational parameters. However, there is a clear gap in connecting the effect of new mutations to fitness. Here we complete a metabolomics study on Arabidopsis thaliana MA lines to determine how mutations affect global metabolic output. Leaf tissue of MA lines with high and low fitness were analyzed for 386 metabolites and compared to the unmutated progenitor. We find that although they do not have an average difference in the number of mutations, low fitness lines have significantly more metabolic subpathways perturbed than high fitness lines. Consistent with this result, we also find that the set of genes in or near mutations in the high fitness MA lines are enriched in DNA-binding transcription factor activity. Taken together, these results suggest that the effect of a new mutation on fitness depends less on the specific metabolic pathways disrupted and more on the pleiotropic effects of those mutations.

**4. Name:** Aaron Mehus **Institution:** University of North Dakota **Email:** aaron.mehus@und.edu **Research area: other****Title:** Activation of PPARγ and inhibition of cell proliferation reduces key proteins associated with the basal subtype of bladder cancer in As3+-transformed UROtsa cells

**Abstract:** It is well-established that environmental exposure to arsenite (As3+) is associated with the development of human urothelial cancer (UC). Muscle invasive urothelial cancer (MIUC) are grouped into basal or luminal molecular subtypes based on their gene expression profile. The basal subtype is more aggressive and can be associated with squamous differentiation, characterized by high expression of keratins (KRT1, 5, 6, 14, and 16) and epidermal growth factor receptor (EGFR) within the tumors. The luminal subtype is less aggressive and is predominately characterized by elevated gene expression of peroxisome proliferator-activated receptor- gamma (PPARγ) and forkhead box protein A1 (FOXA1). We have previously shown that As3+-transformed urothelial cells (As-T) exhibit a basal subtype of UC expressing genes associated with squamous differentiation. We hypothesized that the molecular subtype of the As-T cells could be altered by inducing the expression of PPARγ and/or inhibiting the proliferation of the cells. Non-transformed and As-T cells were treated with Troglitazone (TG, PPARG agonist, 10 µM), PD153035 (PD, an EGFR inhibitor, 1 µM) or a combination of TG and PD for 3 days. The results obtained demonstrate that treatment of the As-T cells with TG upregulated the expression of PPARγ and FOXA1 whereas treatment with PD decreased the expression of some of the basal keratins. However, a combined treatment of TG and PD resulted in a consistent decrease of several proteins associated with the basal subtype of bladder cancers (KRT1, KRT14, KRT16, P63, and TFAP2A). Our data suggests that activation of PPARγ while inhibiting cell proliferation facilitates the regulation of genes involved in maintaining the luminal subtype of UC. In vivo animal studies are needed to address the efficacy of using PPARγ agonists and/or proliferation inhibitors to reduce tumor grade/stage of MIUC.

**5. Name:** Swojani Shrestha **Institution:** University of North Dakota **Email:** swojani.shrestha@und.edu **Research area:** Other**Title:** Down regulation of lysosomal and mTOR related genes in human renal tubular epithelial cells composed of the progenitor CD133+/CD24+ cells and CD24+ cells by elevated glucose

**Abstract:** Hyperglycemia is one of the major health concern in many parts of the world. One of the serious complications of high glucose levels is diabetic nephropathy. The preliminary microarray study performed on primary human renal tubular epithelial (hRTE) cells exposed to high glucose levels showed a significant downregulation of mTOR as well as its associated genes as well as lysosomal genes. Based on this preliminary data, the expression of various lysosomal genes as well as mTOR and its associated genes were analyzed in hRTE cells exposed to 5.5, 7.5, 11- and 16-mM glucose. The results validated the microarray analysis, which showed a significant decrease in the mRNA as well as protein expression of the selected genes as the concentration of glucose increased. Co-localization of lysosomal marker, LAMP1 with mTOR showed lower expression of mTOR as the glucose concentration increased, suggesting decrease in mTOR activity. Although the mechanism by which glucose affects the regulation of lysosomal genes is not well known, our results suggest that high levels of glucose may lead to decrease in mTOR expression causing the cells to enter an anabolic state with subsequent downregulation of lysosomal genes.

**6. Name:** Maia Bennett **Institution:** University of Nebraska at Omaha **Email:** maiabennett@unomaha.edu **Research area:** Other**Title:** Development of A Novel Flow Cytometry-Based Human NK Cell-Mediated ADCC Assay

**Authors:** Maia M.C. Bennett, Arriana D. Blackmon, Paul W. Denton
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**Abstract:** Our team is currently developing a flow-based protocol for human natural killer (NK) cell-mediated antibody-dependent cellular cytotoxicity (ADCC) against B-cell lymphoma. ADCC is one method by which NK cells can kill target cells. Target antibodies (e.g., α-CD20 in B cell lymphoma) are used to facilitate recognition and killing of target cells by NK effector cells. Developing a NK-mediated ADCC assay has involved: determining the optimal target cell discrimination strategy; testing various incubation conditions such as time and volume; incorporating relevant B-cell lymphoma target cell lines; assessing multiple effector to target ratios; and establishing an NK cell pipeline from human procurement to assay incorporation. Our aims are to use this ADCC assay in future combination immunotherapeutic testing and to adjust the protocol for use in various cancer types. Assay development progress to date will be presented and discussed.

**7. Name:** Michael Douchey **Institution:** UNMC **Email:** michael.douchey@unmc.edu **Research area:** Neuroscience

**Title:** Probing Astrocyte Structure in Fragile X Syndrome Using Human-Induced Pluripotent Stem Cells

**Abstract:** FXS is the most common form of inherited intellectual disability and the leading monogenetic cause of autism. FXS is caused by a mutation, a CGG repeat expansion, in the Fragile X Mental Retardation Gene 1 (FMR1) that results in a lack of the Fragile X Mental Retardation Protein. While much of the literature has centered on neuronal FMRP in FXS research, the role of the loss of FMRP in astrocytes has also been explored. FMR1 mouse models have demonstrated that the loss of FMRP in astrocytes impairs synaptic structure and function. However, human astrocytes differ from mouse astrocytes in size, complexity, gene expression and function. Therefore, to better understand the contribution of astrocytes to FXS analysis of astrocytes derived from human FXS cells is necessary. We have developed an in vivo model where astrocytes derived from human-induced pluripotent stem cells (hiPSCs) are engrafted into the brains of immunodeficient mice. Here, we evaluate morphology differences between astrocytes derived from isogenic pair of control and FXS hiPSCs in 3- and 9-month engrafted brains.

**8. Name:** Laura Cogua **Institution:** Creighton University  **Email:** lauracogua@creighton.edu  **Research area:** Other

**Title:** Synthesis and Antimicrobial Evaluation of Multivalent 1,2,3- Triazolium Salts

**Abstract:** Triazolium salts are quaternary ammonium compounds (QACs) that have been shown to exhibit antibacterial and antifungal properties, which vary depending on the identity of their substituents. The purpose of this study was to evaluate the antimicrobial activity of triazolium QACs as the charge of the salt is varied, and to optimize potency through variation of aryl substitution patterns. Aryl azide compounds were synthesized with varying methyl, methoxy, n-butyl, t-butyl and chloro substituents. These aryl azides were reacted with ethynylbenzene, m-diethynylbenzene, p-diethynylbenzene and 1,3,5-triethynylbenzene using a base-catalzyed click reaction to form 1,5-disubsituted-1,2,3-triazole analogs. A library of triazolium salts was prepared by the mono, di and tri-substitution of benzyl bromide, 1-bromobutane or 1-iodobutane groups at the N3 position of each triazole ring. A total of 180 molecules were made and analyzed for antimicrobial properties by performing microdilution minimum inhibitory concentration (MIC) assays against Gram-positive bacteria, Gram-negative bacteria, and yeast. MIC activity indicated a maximum potency of 0.2 μM against Gram-positive bacteria, 0.8 μM against Gram-negative bacteria and 0.4 μM against yeast. MIC potency was enhanced by the presence of benzyl bromide on the N3 position and a hydrophobic chain on the N1-phenyl group. Disubstituted salts with meta connectivity proved to have the highest MIC potency. No significant difference was found in between equivalent bromide and iodide salts. Details regarding the synthesis, characterization, and antimicrobial assays of these compounds will be presented. This publication was made possible by grants from the National Institute for General Medical Science (NIGMS) (5P20GM103427), a component of the National Institutes of Health (NIH), and its contents are the sole responsibility of the authors and do not necessarily represent the official views of NIGMS or NIH.

**9. Name:** Shane Scholten **Institution:** Augustana University **Email:** sscholten@augie.edu **Research area:** Clinical-translational

**Title:** Ischemic Preconditioning Increases Average Power Output Across a 3 Minute Cycling Time Trial: A Randomized Control Trial

**Abstract:** Initially used for cardioprotective measures, ischemic preconditioning (IPC) has transitioned into the athletic world as a noninvasive technique to improve one’s performance. Anaerobic cycling performance has shown to benefit from this technique; however, IPC’s effects on aerobic cycling performance has been inconclusive.
PURPOSE: To determine the acute effects of IPC on a 3-minute cycling time trial performance in active individuals.
METHODS: In a randomized, single-blind crossover study, 11 physically active individuals, nine men and two women (26.4 ± 10.7 yr), performed a 3 minute maximum effort cycling test preceded by alternating bilateral IPC (3 x 5 minute ischemia, 5 minute reperfusion) or SHAM (3 x 1 minute ischemia, 1 minute reperfusion). Both the IPC and SHAM protocols induced 100% occlusion using a Delfi Personal Tourniquet System (PTS). The PTS was used to individualize proper limb occlusion pressure for complete blood flow occlusion to the leg muscles distal to the blood pressure cuff.
RESULTS: Statistical significance was found between the type of treatment (SHAM vs IPC) and 3-minute time trial performance (p=0.03).
CONCLUSION: IPC was found to increase average power output across 3-minute time trial performance in active individuals.

**10. Name:** Samaya Kallepalli **Institution:** Creighton University **Email:** ska64537@creighton.edu **Research area:** Other

**Title:** Investigation of ruthenium complex electrochemiluminescence at gold electrodes modified with different chain length alkanethiols

**Abstract:** Alkanethiols are used in the fabrication of electrochemiluminescent DNA biosensors. This study investigated which conditions lead to the largest and most stable ECL signals when an electrode is modified with monolayers of various alkanethiol chain lengths and immersed in a pH 7.4 buffer solution containing 1-μM Ru(bpy)32+ and the co-reactant 100-mM tri-n-propylamine. Cyclic voltammetry-ECL traces were collected to determine the potential of maximum ECL, which was observed at around 0.65V for each electrode with a peak intensity around 0.85V. Chronoamperometry (CA)-ECL traces were collected in order to: investigate ECL as a function of potential, observe its temporal profile, and measure the reproducibility of its signal. Through CA, potentials 0.70V, 0.75V, 0.80V, and 0.85V were applied at each electrode while ECL was monitored. From the ECL-time traces, the longer alkanethiol lengths electrodes exhibited signals of similar intensities at the same respective potentials. We also observed the ECL signal intensity to increase with increasing applied potential until the threshold of 0.80 V, where the signal became unstable. The most reproducible signals were observed at 0.70V and 0.75V. Through this work, we will determine the optimal alkanethiol chain length and applied potential to use in folding-based ECL nucleic acid biosensors.

**11. Name:** Dustin Slivka **Institution:** University of Nebraska at Omaha **Email:** dslivka@unomaha.edu **Research area:** Other

**Title:** Effects of exercise training in the heat on females

 **Abstract:** Purpose: To determine the impact of three weeks of aerobic exercise training in the heat on thermoregulation, PGC1α mRNA response, and aerobic capacity in women. Methods: Twenty-three untrained college aged females (24±4 years old, 168±5 cm tall, and weighed 67.3±11.2 kg) were randomly assigned to 3 weeks of aerobic exercise training in either 20°C (n=12) or 33°C (n=11) environmental temperatures. Results: VO2max in 20°C increased with training (2.57±0.35 to 2.71±0.32 L·min-1, p=0.01), but was not different between temperatures (p=0.821). HR decreased with training (152±16 to 140±0.13 bpm, p<0.001), but was not different between temperatures (p=0.341). Sweat rate increased with training (0.655±0.192 to 0.775±0.212 L·hr-1, p=0.006) and was higher in 33°C (0.835±0.144 L·hr-1) than 20°C (0.605±0.132 L·hr-1, p<0.001). PGC1α increased with acute exercise before (1.01±0.10 to 4.96±2.08-fold, p<0.001) and after training (1.07±0.10 to 3.21±1.39-fold, p<0.001), but had a smaller response after training (p=0.005). There were no differences in PGC1α between groups (p=0.661). Conclusions: Women increase aerobic fitness and maintain their exercise induced PGC1α mRNA response in the heat equally to that of room temperature conditions. This response contrasts with the blunted PGC1α mRNA response and VO2 max alterations previously observed in men.

**12. Name:** Kurt Zimmerman **Institution:** OUHSC **Email:** kurt-zimmerman@ouhsc.edu **Research area:** Other

**Title:** Exploring the heterogeneity of kidney resident macrophages using single cell RNA sequencing

**Abstract:** Tissue resident macrophages are highly diverse, even when located within the same tissue. This diversity is thought to be driven by localization, ontological origin, time in tissue, and niche specific cues. Importantly, these underlying factors likely influence resident macrophage phenotype and function during disease initiation and progression. To understand the diversity of kidney resident macrophages (KRM), we performed single cell RNA sequencing, parabiosis, and fate mapping on kidneys isolated from wild type and transgenic knock-in reporter mice. Using single cell RNA sequencing, we identified three subpopulations of KRM including one with enriched expression of Ccr2. Using Ccr2-RFP knock-in mice and Ms4a3cre Rosa Stopf/f TdT mice, we confirm that these resident macrophages were derived from monocyte precursors and preferentially localize to the renal cortex. Based on our single cell RNA sequencing data, we propose that monocytes undergo a series of differentiation steps upon entering the kidney to become Ccr2+ KRM. Analysis of single cell data using RNA Velocity and Monocle suggest that monocytes require Cx3cr1 in order to differentiate into Ccr2+ KRM. Loss of Cx3cr1 prevented the accumulation of Ccr2+ KRM and resulted in a skewed macrophage profile that prevented disease progression in a mouse model of cystic kidney disease.

**13. Name:** Addison Grinnell **Institution:** Oklahoma State University **Email:** addison.grinnell@okstate.edu **Research area:** Other

**Title:** A density-dependent requirement of PBP1B for cells to grow after disruption of the MreB-PBP2 elongation complex requires a minimum cell density

**Abstract:** The peptidoglycan (PG) cell wall provides shape and structure to most bacteria. There are two systems to build PG in rod shaped organisms: the elongasome and divisome, which are made up of many proteins including the essential MreB and PBP2, or FtsZ and PBP3, respectively. The elongasome is responsible for PG insertion during cell elongation, while the divisome is responsible for septal PG insertion during division. We found that the main elongasome proteins, MreB and PBP2, can be inhibited without affecting growth rate in a quorum sensing-independent density-dependent manner. Before cells reach a particular cell density, inhibition of the elongasome results in different physiological responses, including intracellular vesicle formation and an increase in cell size. This inhibition of MreB or PBP2 can be compensated for by the presence of the class A penicillin binding protein, PBP1B. Furthermore, we found this density-dependent growth resistance to be specific for elongasome inhibition and was consistent across multiple Gram-negative rods, providing new areas of research into antibiotic treatment.

**14. Name:** Kristy Kounovsky-Shafer **Institution:** University of Nebraska - Kearney **Email:** kounovskykl@unk.edu **Research area:** Other**Title:** Acrylamide characterization to be used in the elution and concentration of Lambda concatemer DNA molecules

**Authors:** Samantha Rau, Alex Larsen, and Kristy Kounovsky-Shafer, Department of Chemistry, University of Nebraska - Kearney, Kearney, NE 68849

**Abstract:** In order to use physical mapping systems such as Nanocoding or Optical Mapping to discover variations among long DNA molecules, the molecules must be spread out to span a large enough region that there is enough unique information on either side of the aforementioned region so that the area of the genome can be assembled. The fragility of large DNA molecules prevents the molecules from remaining full length when routine molecular biology techniques are used to concentrate DNA. Thus, A 3D printed polyacrylamide (PLA) device was developed and affixed to a glass slide to concentrate lambda DNA concatemers. Acrylamide gels of various concentrations were polymerized in the device to act as a “roadblock” to slow down the progression of DNA through the device. Using a pulsed waveform, DNA molecules were concentrated at the acrylamide roadblock. DNA was stained with YOYO-1 dye so that the progression of DNA was monitored while varying the different acrylamide concentrations. The pore size of the acrylamide concentrations was determined using a 1 kb ladder to compare the pore size of the gel to the amount of DNA concentrated.

**15. Name:** Eliezer Lichter **Institution:** UNMC **Email:** eliezer.lichter@unmc.edu **Research area:** Neuroscience

**Title:** Investigating the role of mitochondrial DNA methylation in aging and in a mouse model of Alzheimer's Disease

**Abstract:** DNA methylation is a well-known epigenetic phenomenon regulating DNA transcription. There are variable methylation patterns associated with normal aging, and it was recently shown that the mitochondrial DNA (mtDNA) can be heavily methylated in a pattern that is distinct from genomic DNA. Levels of mtDNA 5-methylcytocine have been shown to be increased in the brains of humans with Alzheimer Disease (AD)-related pathology. However, the methylation patterns of the presynaptic mtDNA, or that related to tau pathology progression, such as AD, has not yet been characterized. Preliminary data obtained in our laboratory shows that there is a decrease in the expression of mtDNA-encoded proteins in the presynaptic terminals of hTau mice, a mouse model of AD. In contrast, our laboratory has shown, during normal mouse aging there are dynamic alterations associated with increased expression of mtDNA-encoded proteins in presynaptic mitochondria. Here we show that there are age-dependent sex differences in the methylation patterns in synaptic and non-synaptic mitochondria in the hippocampi of C57BL/6 mice. These data will be compared with the mtDNA methylation patterns in hTau mice to determine if methylation difference play a role in the protein expression differences seen in aged versus hTau mice.

**16. Name:** Kumi Nagamoto-Combs **Institution:** University of North Dakota School of Medicine & Health Sciences **Email:** kumi.combs@und.edu **Research area:** Neuroscience**Title:** Behavioral consequences of food allergy and associated intestinal dysbiosis

**Authors:** Kumi Nagamoto-Combs\*1, Nicholas Smith2, Danielle Germundson2, Afrina Brishti1
Department of Biomedical Sciences1 and Clinical and Translational Science Graduate Program2, University of North Dakota School of Medicine & Health Sciences, Grand Forks, ND

**Abstract:** Behavioral disorders are often reported in individuals with food allergies. Altered intestinal microbiota and increased epithelial permeability have been suggested to link these two distinct disorders involving the nervous and immune systems, respectively. However, the notion of food allergy as an etiology of behavioral disorders is still debated due largely to inconsistent results among clinical studies and the lack of neuropathologic evidence. To clarify the causative role of food allergy in behavioral changes among allergic individuals and assess behavior distinct from anaphylaxis, we developed a mouse model of non-anaphylactic cow’s milk allergy. Hypothesizing that behavioral changes are a neurological consequence of intestinal impairment due to allergen exposure in susceptible individuals, we compared anxiety- and depression-like behavior in sham and allergen-sensitized mice after an allergen challenge and examined intestinal, immunological, and brain pathologies in these mice. Allergic mice showed decreased intestinal tight junction protein levels, elevated plasma cytokine expressions, and a distinct fecal microbiome profile, as well as neuroinflammation and demyelination in the brains. These results substantiated the contribution of food allergy to intestinal and brain pathologies and identified potential neural substrates for behavioral symptoms. Additional investigations are underway to elucidate the effect of altered microbiota on the nervous system.

**17. Name:** Benjamin Nelson **Institution:** Oklahoma State University **Email:** benjamin.n.nelson@okstate.edu **Research area:** Other

**Title:** Interactions of *Cryptococcus neoformans* with Human Airway Phagocytes

**Abstract:** Cryptococcus neoformans is an opportunistic fungal pathogen that causes over 180,000 annual deaths in HIV/AIDS patients. Innate phagocytes such as dendritic cells (DCs) and macrophages are the first cells to interact with the pathogen. Six different subsets of airway phagocytes (three macrophage and three DCs) have been characterized in healthy human lungs. However, the specific subsets responsible for the fate of C. neoformans are unknown. We hypothesize there are differences with uptake and survival of C. neoformans among the subsets. Healthy human bronchoalveolar lavage (BAL) containing these six phagocytic subsets was incubated with mCherry-expressing C. neoformans for two hours. Cells were examined by flow cytometry to determine association with the fungus and by imaging flow cytometry to visualize intracellular cryptococcal morphology, indicating killing or replication. Single cell RNA sequencing (scRNA-seq) was used to determine relative gene expression following cryptococcal interaction. Results showed that all phagocyte subsets interacted with C. neoformans, and different fungal morphologies were observed. scRNA-seq revealed differential TNF-α signaling among subsets. These findings suggest the outcome depends on the specific phagocyte subset C. neoformans encounters. Future studies will examine correlations of anti-cryptococcal mediators such as ROS, iNOS, and cathepsin B to antifungal activity in these phagocyte subsets.

**18. Name:** Annemarie Shibata **Institution:** Creighton University **Email:** annemarieshibata@creighton.edu **Research area:** Neuroscience**Title:** Investigating mutations associated with mild-to-moderate Carnitine Palmitotransferase II Deficiency and Schizophrenic Psychosis to understand metabolic signaling in CNS development and function.

**Authors:** Aaron Marta, Rochelle Wickramaskara, Holly Stessman and Annemarie Shibata
Creighton University Omaha NE

**Abstract:** Congenital metabolic diseases are rare inherited genetic disorders associated with a wide range of neurodevelopmental etiologies. During early postnatal development and periods of starvation, neurons use ketone bodies produced by astrocytes or from the liver for beta-oxidation and ATP synthesis. Beta-oxidation depends upon carnitine transferase shuttle to mediate carnitine-dependent entry of long chain acyl-coenzyme A into the mitochondrial matrix. Carnitine palmitoyltransferase II (CPTII) deficiency is a metabolic disorder resulting in impaired transport of long-chain fatty acids from the cytosol to the mitochondrial inner membrane, where fatty acid takes place. We have recently published a case study report outlining CPTII deficiency in a proband also diagnosed with schizophrenia. We will present whole exome sequencing and clinical presentation of the proband. We will present our experimental design to study the proband specific mutations in the zebrafish model system. These studies in the zebrafish model system will inform our understanding of how specific mutations in metabolic signaling affect brain development and function and may potentially contribute to neurological disorders such as schizophrenia.

**19. Name:** Samantha Mercer **Institution:** University of Nebraska at Kearney **Email:** mercerse@lopers.unk.edu **Research area:** Other**Title:** Determining host-T4BSS interactions of *Coxiella burnetii* using proximity labeling

**Authors:** Samantha Mercer, Brandon Luedtke, Department of Biology, the University of Nebraska at Kearney, NE 68849

**Abstract:** Coxiella burnetii is a gram-negative intracellular pathogen that causes the respiratory disease Q-Fever. During host cell infection, C. burnetii forms a parasitophorous vacuole (PV) from where it manipulates host cell processes. To mediate the infection an essential type IVB secretion system (T4BSS) is utilized. Interestingly, C. burnetii was shown to secrete the T4BSS protein DotA, which localized to the PV membrane. However, it is unknown if DotA interacts with host cell proteins as a part of the infectious process. To determine any DotA-host interactions, we chose to use proximity labeling, which has been used to show interactions between Chlamydia trachomatis effector proteins and the inclusion membrane. Here we constructed two expression plasmids that fused DotA to the promiscuous biotin ligase TurboID on either the N or C terminal. Wild type C. burnetii and the DotA knockout were transformed with the respective plasmids and then used to initiate tissue culture infections. In addition, western blot analysis proved that the bacteria were expressing the full DotA and TurboID fusion protein. When the tissue culture infections start to mature, we hope to identify the first interactions between the C. burnetii T4BSS and host cell proteins.

**20. Name:** Erin Gross **Institution:** Creighton University **Email:** ErinGross@creighton.edu **Research area:** Other**Title:** Comparison of mobile phone and CCD cameras for detection of biogenic amines via Ruthenium complex electrogenerated chemiluminescence

**Authors:** Erin M. Gross, Nicolas Heckenlaible, Department of Chemistry, Creighton University, Omaha, NE 68178, Alyssa Kava, Charles S. Henry, Department of Chemistry, Colorado State University, Fort Collins, NE 80524

**Abstract:** Portable analytical devices are important to the development of point-of-care diagnostics and on-site safety testing. Electrogenerated chemiluminescence (ECL) is an analytical technique that combines features of both electrochemical and luminescence methods. It possesses electrochemical advantages such as ease of miniaturization and portability, along with the sensitivities of fluorescence methods. In this work, the ECL reaction between tris(2,2’-bipyridyl)ruthenium(II) and biogenic amines was used to develop a detection method for biogenic amines at screen-printed carbon ink electrodes. Both a mobile phone camera and CCD camera were used as the detectors. Each detector was aligned with the electrodes via 3D printed light-tight housings. Image analysis was performed using the free software ImageJ. The analytical figures of merit of the two methods were compared. The mobile phone’s optical sensor achieved limits of detection of 127, 195, and 421-μM for spermidine, putrescine, and histamine, respectively. These detection limits were slightly higher than that determined for the CCD but were acceptable for applications to food safety. The mobile phone was able to determine the content of amines in skim milk to within an 8.6% error.

**21. Name:** Alexander Kloth **Institution:** Augustana University **Email:** akloth@augie.edu **Research area:** Neuroscience**Title:** Sex-dependent impacts of environmental enrichment on Angelman Syndrome mice

**Author:** Alexander D. Kloth (Principal Investigator)
Department of Biology, Augustana University, Sioux Falls, SD
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**Abstract:** Angelman Syndrome (AS) is a rare neurodevelopmental disorder caused by mutations maternal allele of UBE3A and in humans is marked by intellectual disability, ataxia, autism, and a happy demeanor; in mice, these symptoms are displayed as deficits on a variety of behavioral assays including the open field test, marble burying, rotarod, novel-object recognition, and forced swim, as well as physiological defects associated with learning. Notably, there are no effective therapeutic approaches for treating AS. We investigated the ability of a behavioral manipulation known as environmental enrichment (EE)—post-weaning exposure of AS mice and wild-type littermates to increased space, toys, treats, and running wheels—to rescue these phenotypes. Importantly, we examined whether there are sex-dependent differences in treatment outcomes on behavioral tasks. We found that EE for male AS mice reduced the motor coordination deficits usually seen in rotarod performance and restored species-specific marble burying behavior. We also examined alterations of behavior in the open field test and in the forced swim task. EE also ameliorated the weight phenotype in AS mice. Interestingly, female AS mice did not respond to environmental enrichment in the same way as the male mice. Ongoing experiments are examining whether EE can rescue well-documented deficits in AS mice related to plasticity, including reduced spine density and dampened long-term plasticity.

**22. Name:** Gwyneth Velasco **Institution:** College of Saint Mary **Email:** gvelasco7313@csm.edu **Research area:** Other**Title:** Chemical attraction of ticks (*Parasitiformes*: Ixodidae) to decomposition volatile organic chemicals

**Authors:** Gwyneth Velasco, Amanda Roe, Ph.D., Biology Program, College of Saint Mary, Omaha, NE 68106

**Abstract:** For arthropods that require blood to complete their life cycle, finding an acceptable blood host is an integral component. One method to increase host-finding may be found in certain tick species: to preferentially move to carrion and/or be mechanically transported by necrophagous insects to carrion, which increases their chances of finding a host. Although ticks use a combination of senses to locate hosts, they depend the most on chemical cues. Knowing this, we investigated two major research questions: 1. are ticks attracted to animal decomposition, and 2. if so, which volatile organic compounds are they attracted to? The research questions were divided into individual experiment trials using the adult tick species Amblyomma americanum (Lone star tick) and Dermacentor variabilis (American dog tick). Both trials were conducted with a dual-choice olfactometer. Trial 1 was conducted using a dead fetal pig. Tick attractiveness to the remains was tested every 24 hours for 168 hours (7 days). Trial 2 was conducted using individual volatile organic compounds. These included: dimethyl disulfide, dimethyl trisulfide, trimethylamine, indole, and phenol. Compounds were determined based on previous decompositional VOC research. Carbon dioxide was used as a control. Knowing tick attractiveness to animal decomposition can lead to better understanding of their host-finding behaviors and can lead to better tick population control measures.

**23. Name:** Meera Cao **Institution:** College of Saint Mary **Email:** mcao7151@csm.edu **Research area:** Other

**Title:** Three Transketolases in Salmonella enterica Contribute to Defending Against Oxidative and Nitrosative Stresses

**Abstract:** Within the United States, Salmonella strains cause 1.35 million infections and 420 deaths every year. Salmonella enterica serovar Typhimurium is a facultative intracellular pathogen that has evolved an array of antioxidative and anti-nitrosative defenses to detoxify host-derived reactive oxygen species (ROS) and reactive nitrogen species (RNS) produced during the innate immune response to infection. Several of these mechanisms depend on the NADH and NADPH production from glycolysis and the oxidative branch of the pentose phosphate pathway. Recently, we have shown that transketolases, which are rate-limiting enzymes of the downstream nonoxidative branch of the phosphate pathway, are required for Salmonella virulence in a mouse model of infection. S. Typhimurium strains with mutations of the transketolases (TktA, TktB, and TktC) are hypersensitive to killing by ROS and RNS compared to wild-type controls. This sensitivity is independent of NADPH production in the oxidative branch of the phosphate pathway. In this study, we will test the hypothesis that transketolases are required for the expression of S. Typhimurium antioxidant defense genes. We will test this hypothesis by comparing the expression of characterized antioxidant defense genes in wild-type and transketolase-deficient S. Typhimurium exposed to ROS and RNS. This study will help us understand how transketolases contribute to S. Typhimurium pathogenesis by regulating defenses against host-derived ROS and RNS.

**24. Name:** Alexandra Van Cleave **Institution:** Creighton University **Email:** alexvancleave@creighton.edu **Research area:** Other

**Title:** Analysis of bacterial growth in the presence of glmS riboswitch ligand analogs

**Authors:** Alexandra Van Cleave, Dr. Juliane Soukup

**Abstract:** Antibiotic resistance is an increasingly concerning public health issue as bacterial genomes acquire new genes, preventing the debilitating antibiotic effects. Therefore, researchers are investigating the potential of riboswitches as antibacterial drug targets. Riboswitches are non-coding mRNA sections that affect downstream gene expression in response to ligand binding.
 The glmS riboswitch controls expression of fructose-6-phosphate amidotransferase which produces glucosamine-6-phosphate (GlcN6P), a bacterial cell wall precursor. This catalytic riboswitch demonstrates self-cleavage upon binding with GlcN6P. The cleavage inhibits glmS gene expression and prevents cell wall synthesis. The glmS riboswitch is identified in over 400 gram-positive bacteria strains. Due to its prevalence and ability to control cell viability, the glmS riboswitch is a potential antibiotic target.
 This project aims to identify an analog, with similar affinity for the glmS riboswitch as GlcN6P, which inhibits cell viability. Growth assays monitored the growth of Bacillus subtilis and Staphylococcus aureus in the presence and absence of potential analogs. Current studies suggest that L-serine inhibits bacterial growth at concentrations of 96 mM for B. subtilis and 24 mM for S. aureus. Future studies will verify that the analog functions via riboswitch interactions and will investigate analog effects on mutant strains of B. subtilis and S. aureus.

Special thanks to the Soukup lab, NIH INBRE grant and CURAS for supporting this project.

**25. Name:** Afrina Brishti **Institution:** University of North Dakota **Email:** afrina.brishti@und.edu **Research area:** Neuroscience**Title:** Effect of repeated allergen exposure on cognitive functions and behavior in a mouse model of cow’s milk allergy

**Authors:** Afrina Brishti1, Danielle L. Germundson2, Nicholas A. Smith2, Colin K. Combs1 and Kumi Nagamoto-Combs1
Departments of Biomedical Sciences1 and Pathology2, University of North Dakota School of Medicine & Health Sciences, Grand Forks, ND 58202.

**Abstract:** Food allergy is an immunological disorder typically recognized and diagnosed by the skin and gastrointestinal symptoms and anaphylaxis. In addition, many clinical observations have reported mood and behavior changes in some food-allergic patients after consuming offending foods without severe reactions. However, how allergic hypersensitivity influences brain function and alters mood and behavior is not well understood. We hypothesized that repeated consumption of an offending food by allergic individuals with mild reactions would result in chronic neuroinflammation, which in turn affects behavior. To test our hypothesis, we established a repeated allergen exposure paradigm with a mouse model of non-anaphylactic cow’s milk allergy, in which C57BL/6J mice were sensitized to a bovine whey protein, β-lactoglobulin (BLG), and subsequently placed on a whey-containing diet. After three months of allergen exposure, BLG-sensitized mice exhibited anxiety-like behavior and cognitive dysfunction with the elevated zero maze and cross maze tests, respectively. Furthermore, cytokine array analysis using brain lysates showed that several inflammatory cytokines were elevated in the hippocampus of BLG-sensitized mice compared to sham-sensitized mice or BLG mice placed on a whey-free diet. Our results provide evidence for profound region-specific neuroinflammation and behavioral changes associated with repeated allergen consumption.

**26. Name:** Jesse Bueno **Institution:** The University of Oklahoma Health Sciences Center **Email:** Jesse-Bueno@ouhsc.edu **Research area:** Other**Title:** Sensory nerve depletion aids tumor development while neuropeptide inhibition impedes further development of established tumors

**Authors:** Jesse Bueno, Deborah Samkutty, Lydia Burger, Annie Doyle, Erin Richardson, Richard Pippin, Abigael Williams, Juan Jose Macias, and Maureen Cox

**Abstract:** Breast cancer is one of the most prominent types of cancer in the world, indicating an area of study with high importance. Solid tumors are known to recruit nerves from the surrounding tissue, which can aid tumor development in certain tumor types. Greater innervation of tumor in breast cancer patients is associated with a poor prognosis, and it is believed that different nerve types that are present have different functions relating to tumor. Exact nerve types in breast cancer and the mechanism of specific impact on tumor development, however, is still unknown. We have found that when a subset of sensory nerves are depleted, transplanted breast tumor grows much more aggressively in mice compared to controls. The depletion of capsaicin-sensitive sensory neurons results in faster tumor growth in B6 mice, but not in Rag1-/- mice, suggesting that sensory nerves are acting on an adaptive immune function. Further investigation involving the inhibition of a neuropeptide produced by sensory nerves, calcitonin gene-related peptide (CGRP), shows impeded growth in established tumor, indicating both pro- and anti-tumor activity of sensory nerves. If current pre-clinical therapies prove sufficient, existing sensory nerve signaling related drugs can be repurposed for cancer treatment.

**27. Name:** Rajesh Kandel **Institution:** Washburn University of Topeka, KS **Email:** rajesh.kandel@washburn.edu **Research area:** Other**Title:** Constructing a Micro Fabry-Perot Cavity System for Raman Detection and Characterization of Chemical Properties of Nanoscale Materials

**Authors:** Rajesh Kandel and Hoang Long Nguyen
Washburn University, Topeka, KS 66614

**Abstract:** Raman spectroscopy is a powerful technique for obtaining structural information about the molecules inside a cell. However, commercial Raman instrument often requires high concentrations of analytes, making it unsuitable for studying biomolecular activities in vivo. Optical microcavities can significantly enhance the Raman signal by concentrating the optical power and increasing the density of states of the Raman scattered light. Microcavities can therefore lower the limit of detection of Raman signal to nanomolar concentrations of analytes. The simplest form of a microcavity is a micro Fabry-Perot cavity, which is formed by two highly reflecting mirrors situated micrometers apart. The distance between the mirror is designed to match the resonance condition of the cavity to the wavelength of the Raman light for maximum amplification. As a result, a precise control of the mirror position is essential to the cavity construction. In this research, the distance between these two mirrors will be controlled by a homebuilt piezo stage to match the resonance condition of the incident and Raman scattered light and maximize the light-particle interaction. We will combine our microcavities with chemically-specific Raman spectroscopy to detect and observe the biomolecular activities of low-concentration species inside a cell.

**28. Name:** Patricia Soto **Institution:** Creighton University **Email:** patriciasoto@creighton.edu **Research area:** Neuroscience**Title:** Impact of calcium binding on the residue connectivity of Tcb2

**Authors:** Armaan Kumar [1], Adina M. Kilpatrick [2], Patricia Soto [3]

[1] Department of Chemistry, Creighton University
[2] Department of Physics and Astronomy, Drake University, Des Moine, Iowa 50311
[3] Department of Physics, Creighton University

**Abstract:** Although substantial progress has been made to design computational algorithms that predict stable protein structures, a consistent workflow to mimic the mechanism of structure stabilization remains underdeveloped. To fill this gap, we chose Tcb2, a protein found in the cytoskeleton of the protozoan Tetrahymena thermophila, to examine a computational protocol that identifies the biophysical forces that drive three-dimensional structure stabilization. Because Tcb2 is a well-studied protein using solution NMR, a wealth of experimental data is available to validate our protocol. Tcb2 is a Calcium binding protein that shows two well defined conformations. The conformational changes, however, are distinct from other well-known Calcium binding proteins such as Calmodulin. Our structural bioinformatics protocol captures the trend in conformational compactness, local mobility, and relative orientation of a-helices detected in the NMR analysis. Although the order parameter S2 is well represented by the simulations for residues in a-helices, the calculation for residues in the linkers is less accurate. Our network analysis highlights how the residue connectivity changes upon Calcium binding. We argue, therefore, that our protocol can be utilized to study other a-helix rich proteins that are much more challenging to study in a wet lab, such as the C-terminus of prion proteins.

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**29. Name:** Patricia Soto **Institution:** Creighton University **Email:** patriciasoto@creighton.edu **Research area:** Neuroscience**Title:** Comparative study of residue dynamic coupling across prion proteins from species with distinct susceptibility to prion diseases

**Authors:** Garret Gloeb[1] and Patricia Soto [2]

[1] Department of Chemistry, Creighton University
[2] Department of Physics, Creighton University

**Abstract:** Transmissible spongiform encephalopathies (TSEs, commonly termed prion diseases) are invariably fatal neurodegenerative disorders in mammals, including humans. The molecular level hallmark of prion diseases is the autocatalytic misfolding and polymerization of the extracellular and glycophosphatidylinositol-anchored prion protein (PrPC) to its infectious form (PrPSc). Protein conformational conversion involves rearrangement of the disordered N-terminus and of the a-helix rich C-terminus. Although the prion gene and overall fold of the C-terminus of the prion protein are highly conserved across species, residue substitutions modulate susceptibility to TSEs. For example, residue Met129 in human prion protein correlates with higher propensity to sporadic Creutzfeldt-Jakob, a form of TSE in humans, than Val129. The mechanism by which such residue substitutions impact residue connectivity between the disordered N-terminus and the globularly folded C-terminus of the prion protein remains unknown. Our study focuses on the prion protein of three species each with distinct susceptibility to TSEs. Our structural bioinformatics analysis indicates that the trend in local structural mobility lies in an inverse order to susceptibility to the TSEs. Normal mode analysis and network analysis suggest that in prion protein structures from species with low susceptibility to TSEs, the shortest paths of communication between methionine residues are localized in the C-terminus. In contrast, in prion protein structures from species with high susceptibility to TSEs, the shortest paths between methionine residues involve the N-terminus and C-terminus. We propose a model that links residue connectivity between N-terminus and C-terminus to prion protein misfolding pathways.
Acknowledgment: This publication was made possible by grants from the National Institute for General Medical Science (NIGMS) (2P20GM103427), a component of the National Institutes of Health (NIH), and its contents are the sole responsibility of the authors and do not necessarily represent the official views of NIGMS or NIH.

**30. Name:** Benjamin Brandsen **Institution:** Creighton University **Email:** benbrandsen@creighton.edu **Research area:** Other**Title:** Production of highly diverse lasso peptides by a single biosynthetic pathway

**Authors:** Benjamin M. Brandsen\*, Kelly S. Johnson, Mia N. Morrissey, and Ethan Hills
Department of Chemistry and Biochemistry, Creighton University, Omaha, NE 68178

**Abstract:** Lasso peptides are ribosomally synthesized and post-translationally modified peptide natural products that exhibit a wide range of biological activities. Their threaded lariat structure provides excellent resistance to heat and proteases, making them attractive as potential inhibitors for enzymes and protein-protein interactions. Because of the threaded lariat structure, however, preparation of lasso peptides by traditional synthetic strategies is challenging. As a strategy to prepare diverse lasso peptides, we evaluated the biosynthetic pathway that produces klebsidin, a lasso peptide from Klebsiella pneumoniae, for its potential to convert highly diverse precursor peptides into mature lasso peptides. By exploiting an intracellular expression assay in Escherichia coli where production of a lasso peptide leads to inhibition of cellular growth, we evaluated approximately 10,000 variants with 1-4 amino acid insertions in the lasso peptide loop and tail regions. Using biological activity as a measure for lasso peptide production, we identified more than 1,000 peptide sequences that are processed into mature lasso structures. These data highlight the tremendous potential of the klebsidin biosynthetic pathway to produce diverse lasso peptides, and the results suggest the klebsidin biosynthetic pathway will be useful for generating libraries of novel lasso peptides to screen as inhibitors against important biological targets.

**31. Name:** Padmashri Ragunathan **Institution:** University of Nebraska Medical Center **Email:** p.ragunathan@unmc.edu **Research area:** Neuroscience**Title:** Behavioral alterations in a mouse model of Fetal Alcohol Spectrum Disorders

**Authors:** Tammy Chaudoin, Stephen Bonasera and Padmashri Ragunathan
Division of Geriatrics, Department of Internal Medicine
Department of Neurological Sciences
University of Nebraska Medical Center, Omaha

**Abstract:** Fetal alcohol spectrum disorders (FASD) is one of the leading causes of developmental abnormalities worldwide. Here, we use a model of maternal voluntary alcohol consumption throughout gestation to investigate the effects of prenatal alcohol exposure (PAE) on behavioral phenotypes in mice. Metabolic studies were performed to study the effects of PAE on body composition and energy expenditure. Baseline behaviors including feeding, drinking, movement and their circadian rhythm were examined by performing home cage monitoring in adult mice. We also evaluated a range of behaviors that examined motor function, motor skill learning, anxiety-related behavior and sensorimotor gating to study the long-lasting behavioral alterations. Label-free proteomic analysis of cortical synaptosomes was performed to understand whether PAE results in persistent alterations in the synaptic proteome. Our data show that PAE alters offspring growth and body weight and affects body composition. Male PAE mice show dysregulation of time budgets. Results from a battery of behavioral tests show long-lasting behavioral impairments in offspring prenatally exposed to alcohol. We also found that PAE altered proteins associated with synaptic function as well as astrocytic proteins. Our results show that PAE results in long-lasting behavioral impairments and alterations in the synaptic proteome.

**32. Name:** Matthew Osmanski, Kelly Kleekamp, and Josh Schumacher **Institution:** Augustana University **Email:** mjosmanski19@ole.augie.edu **Research area:** Other

**Title:** Inhibition of Biofilm Formation by Mammalian Placenta Extracts

**Abstract:** The placenta is an organ that is composed of both maternal and fetal tissues. One role of the placenta is to produce antibacterial factors and signalling molecules that are part of innate immunity. It has been demonstrated that whole-placenta extracts inhibit bacterial biofilm formation, but there is little information about the mechanism by which the placenta inhibits biofilm growth, or whether the factors involved are derived from maternal or fetal placenta tissues. To study the antimicrobial properties of placenta, Staphylococcus aureus and Escherichia coli biofilms were cultured with and without homogenized maternal and fetal placental extracts. We found that cultures treated with placenta extracts exhibit less biofilm formation than cultures without placenta. Future work will determine whether the placenta kills bacteria in the planktonic phase in this assay and will determine whether antibacterial factors are associated with the maternal or the fetal portion of the placenta.

**33. Name:** Taylor McDonald Hamilton  **Institution:** Creighton University  **Email:** taylorhamilton@creighton.edu  **Research area:** Other**Title:** Understanding the binding mechanisms between CAF-1 and PCNA mutants

**Authors:** Taylor McDonald Hamilton and Lynne Dieckman
Department of Chemistry and Biochemistry, Creighton University

**Abstract:** The process of packaging newly synthesized DNA into nucleosomes is known as replication-coupled nucleosome assembly. This process maintains genome stability by ensuring proper gene activation and silencing. Proliferating cell nuclear antigen (PCNA) and chromatin assembly factor 1 (CAF-1) are two important proteins involved in replication-coupled nucleosome assembly. PCNA is the sliding clamp protein that acts as a scaffold to recruit proteins to the replication fork during replication and repair. CAF-1 is a heterotrimeric protein that brings histones to DNA during nucleosome formation. During replication-coupled nucleosome assembly, PCNA binds and recruits CAF-1 to the replication fork, and the interaction between these proteins is essential for accurate gene silencing. The lack of binding between PCNA and CAF-1 results in the expression of genes that should be silenced. However, the binding mechanism of these proteins is not yet known. Interestingly, we have identified mutations in PCNA that yield increased affinity with CAF-1 compared to that of wild type PCNA. We are currently carrying out binding assays to determine whether this interaction is unique to the CAF-1-PCNA interaction or if the mutation confers higher affinity for all PCNA interacting proteins. Thus far, the results of these studies, suggest a novel interaction between PCNA and CAF-1.

**34. Name:** Keely Orndorff **Institution:** Creighton University **Email:** kso71670@creighton.edu **Research area:** Other**Title:** Using X-ray Crystallography to determine the mechanism of interaction between Gene Silencing Proteins

**Authors:** Keely Orndorff, Lynne Dieckman
Department of Biochemistry, Creighton University

**Abstract:** The accurate replication of DNA and the proper packaging of newly synthesized DNA into nucleosomes are two crucial processes for maintaining genomic stability. Nucleosome assembly occurs when the DNA behind the replication fork is immediately wrapped around an octamer of histone proteins that make up the nucleosome. This assembly of nucleosomes will dictate which genes will be expressed and which will be silenced. The interaction between two proteins, proliferating cell nuclear antigen (PCNA) and chromatin assembly factor 1 (CAF-1), is essential for this process of replication-coupled nucleosome assembly. PCNA is a homotrimeric sliding clamp required for all DNA-templated metabolic processes. PCNA recruits CAF-1, a heterotrimeric histone chaperone that deposits histone proteins onto DNA during nucleosome assembly, to sites of nucleosome formation. Disruption of the PCNA-CAF-1 interaction can lead to chromatin rearrangements, inhibition of heterochromatin silencing, and mutator phenotypes, which are common features of many human cancers. However, the precise mechanism of interaction between these two proteins remains unclear. This project aims to determine the interaction between PCNA and CAF-1 at the structural level using X-ray crystallography. Current progress has identified promising crystallization conditions that yield crystals of a PCNA-CAF-1 complex which diffract to a resolution of 4 Angstroms. Determination of the structure of this complex will lead to a better understanding of genetic inheritance and how disruptions in this interaction can lead to disease.

**35. Name:** Michelle Holt **Institution:** University of Oklahoma **Email:** mnholt1217@ou.edu **Research area:** Other

**Title:** Intersection of ParE toxin expression and antibiotic susceptibility in pathogenic bacteria

**Authors:** Michelle Holt#, Shengfeng Ruan#, Christina R. Bourne
#equal first authors
University of Oklahoma, Department of Chemistry and Biochemistry, Norman, OK

**Abstract:** ParDE is a type II toxin-antitoxin system encoded in many pathogenic bacteria including Burkholderia cenocepacia and Pseudomonas aeruginosa. This family of proteins consist of a toxin, ParE, that targets the DNA gyrase and a cognate antitoxin, ParD, that neutralizes the toxicity of toxin through protein-protein interactions. Upon ParE inhibition of gyrase, the SOS response becomes activated due to the accumulation of DNA breaks, and error-prone polymerases can repair these breaks, preventing cell death. We hypothesize that low amounts of ParE expression may increase antibiotic resistance due to mutations arising from the SOS response. Our study seeks to determine if this occurs by using different ParE toxins expressed in their host bacteria. We performed toxicity assays to determine the highest concentration of ParE induction that allows for cell survival, thus providing a window for potential mutations as part of a repair pathway. On-going studies will determine the mutation rate as a function of ParE expression using fluctuation assays. We will also evaluate impacts of ParE expression on susceptibility of bacterial hosts to antibiotics by assessing minimum inhibitory concentrations (MICs) with non-toxic levels of ParE toxin expression.

**36. Name:** Neal Sinha **Institution:** Creighton University **Email:** NealSinha@creighton.edu **Research area:** Other**Title:** Solving the Structure of the interaction between PCNA and CAF-1 via X-ray crystallography

**Authors:** Neal Sinha and Dr. Lynne Dieckman
Creighton University Department of Chemistry and Biochemistry
Omaha, NE

**Abstract:** Replication-coupled nucleosome assembly is the process by which newly synthesized DNA gets packaged around histone proteins to form nucleosomes. This process is crucial for determining whether DNA is accessible to transcription factors, which dictates gene expression. Replication-coupled nucleosome assembly involves two major proteins: Proliferating cell nuclear antigen (PCNA) and chromatin assembly factor 1 (CAF-1). PCNA is a ring-shaped protein that encircles DNA and recruits many proteins, including CAF-1, to the replication fork. CAF-1 is a chaperone protein that carries histones to newly replicated DNA. The interaction between PCNA and CAF-1 is required for proper gene silencing. The goal of my project is to determine the 3D structure of the interaction between PCNA and CAF-1 using X-ray crystallography. To do this, I have expressed and purified large amounts of PCNA-CAF-1 protein complexes and determined conditions under which these protein complexes crystallize. I plan to generate diffraction maps using these crystals and determine the 3D structure of the interaction between PCNA and CAF-1. This will provide valuable insight into the mechanism of replication-coupled nucleosome assembly and ultimately how issues in this process may lead to diseases related to improper gene silencing.

**37. Name:** Michael Kinter **Institution:** Oklahoma Medical Research Foundation **Email:** mike-kinter@omrf.org **Research area:** Other**Title:** IDeA National Resource for Quantitative Proteomics.

**Authors:** Michael Kinter, Sam Mackintosh, Rick Edmondson, Stephanie Byrum, and Alan Tackett. Oklahoma Medical Research Foundation, Oklahoma City, OK. University of Arkansas for Medical Sciences, Little Rock, AR.
NIGMS R24 funding established this National Resource in 2020, building on prior funding to the Oklahoma and Arkansas Inbre programs.

**Abstract:** The resource has two workflows; 1) high-throughput discovery proteomics using the expertise and instrumentation at University of Arkansas for Medical Sciences (UAMS) and 2) targeted validation proteomics using the expertise and instrumentation at the Oklahoma Medical Research Foundation (OMRF). Both workflows are backed by bioinformatics analyses provided by the group at UAMS which specializes in the evaluation of proteomics data. As an example of the costs, a 10-sample tandem mass tag (TMT) experiment costs $2000, a 30-sample data independent acquisition (DIA) experiment costs $2600, and a 10-sample targeted experiment costs $1000. The R24 grant funds a voucher system to provide fully subsidized experiments for selected projects. Deadlines for applications for these vouchers are 3 times per year in June, October, and February.
The goal of this resource is to provide cost effective access to state-of-the-art quantitative proteomics platforms and skilled bioinformaticians to IDeA state scientists. Ideally, this access will generate a corresponding increase in the capacity of IDeA states to perform cutting-edge research.

**38. Name:** Chun Wu **Institution:** Mount Marty University **Email:** cwu@mountmarty.edu **Research area:** autoimmune disease

**Title:** *In silico* and *In vitro* investigation of the role of *Streptococcus pneumoniae* and *Acinetobacter baumannii* in the pathogenesis of Multiple Sclerosis

**Authors:** Kieren Luellman, Makenzi Rockwell, Nichole Haag, Chun Wu

**Abstract:** Multiple sclerosis (MS) is an autoimmune disease, in which a patient’s immune system mistakenly attacks his/her own protective sheath of nerve cells, myelin. The cause of MS is unknown. Statistically, many MS patients have a chronic medical history of *Streptococcus pneumoniae* or *Acinetobacter baumannii infections.* We hypothesized that *Acinetobacter baumannii or Streptococcus pneumonia* debris serve as the antigens to induce antibody production. In this study, we determined the sequence similarity of *Acinetobacter Baumannii* and*Streptococcus Pneumonia* with five peptides from three myelin proteins, *i.e.* Proteolipid Protein (PLP), PLP 139-151, PLP 178-191, Myelin Basic Protein (MBP) 84-104, MOG 92-106 and Myelin Oligodendrocyte Glycoprotein (MOG) 35-55, respectively, which was previously reported to induce Experimental autoimmune encephalomyelitis (EAE). We found that there are 11 and 97 hits (*i. e.* five and more amino acid sequence similarity) from *Acinetobacter Baumannii* and*Streptococcus Pneumonia* genomes respectively which are identical or similar to PLP139-151. There are also 32 and 97 hits to PLP178-191, 35 and 91 hits to MBP 84-104. 41 and 99 hits to MOG 35-55 and 26 and 22 hits to MOG92-106 respectively. In addition, Western blotting was used to assess possible interaction between the bacterial protein components and rat anti-MBP, anti-MOG and anti-PLP antibodies, corresponding to MBP 84-104, MOG 92-106/35-55 and PLP 139-151/178-191 respectively. We found that anti-MOG antibody recognized a protein of 25 kDa in *Acinetobacter baumannii*. The results suggested that this protein might potentially serve as an antigen to induce anti-MOG antibody. The proteomic study is underway to identify the nature of this protein. Our findings might pave the road for understanding of the pathogenesis of MS.

**39. Name:** Srivatsan Kidambi **Institution:** University of Nebraska-Lincoln **Email:** skidambi2@unl.edu **Research area:** Neuroscience

**Title:**

**Abstract:** Aging is associated with tissue regeneration and significant loss of function in brain cells, including microglia. Microglia play a critical role in the primary immune response of the central nervous system which are highly active and motile cells interacting chemically and mechanically with their environment. Studies have shown that brain stiffness is significantly higher in aging brains compared to younger brains. The role of matrix stiffness as related to subtle but pivotal changes in microglia physiology and dysfunction is under explored. The overall goal of our study is the development and implementation of a platform that enables the convergence of engineered cell microenvironment with the phenotypic and functional analysis of microglia. Using our innovative biomimetic model that allows modulation of substrate stiffness (2 kPa, 8 kPa, 15 kPa, and 25 kPa mimicking young, mid-age, middle-age, and aged brain tissue, respectively), we investigated the role of matrix stiffness in modulating microglial phenotype and function. We demonstrated that stiffness increased microglial proliferation and migration, elevated expression of inflammatory markers, and a heightened inflammatory M1 profile. We also demonstrated that microglia cultured on aging-like stiffness showed 1) increased ROS production, 2) impaired mitochondrial respiration, and 3) increased lipid droplet accumulation and total intracellular cholesterol. We observed a significant increase in oxidized glutathione (GSSG) in microglia cultured on aging-like stiffness compared to young brain stiffness. A similar effect has been observed in microglia isolated from aging murine models indicating a correlation to physiological conditions. These data suggest a plausible mechanism that increased stiffness modulates microglial dysfunction. Understanding the impact of stiffness on microglial biology will provide significantly more nuanced data to intervene in an aging-related loss in brain function.

**40. Name:** Olivia Burleigh **Institution:** Creighton University **Email:** olb19378@creighton.edu **Research area:** Neuroscience

**Title:**

**Abstract:** Multiple Sclerosis (MS) is an autoimmune disease that causes demyelination in the CNS. One leading hypothesis is that MS progression may be triggered by certain viral infections. Theiler’s murine encephalomyelitis virus (TMEV) is a single-stranded RNA cardiovirus and is used to model MS. Long non-coding RNAs (lncRNAs) were recently discovered to play a role in inflammation. We hypothesized that after TMEV infection in microglia, certain lncRNAs are differentially expressed and alter the viral load of infected microglia. To test our hypothesis, we infected the BV2 microglial cell line, primary microglia, and FBV/nJ mice with TMEV. Following infection, RNA was isolated for RT-PCR analysis. Functional studies using knockdown and overexpression of lncRNA-Nostrill were performed in BV2s. TMEV infection significantly upregulated Nostrill expression compared to controls. Knockdown of Nostrill in BV2s significantly increased viral load, while its overexpression significantly reduced the viral load. TMEV infection in primary microglia significantly upregulated pro-inflammatory and anti-inflammatory cytokines. The induction of Nostrill in response to TMEV infection likely contributes to antiviral defense in microglia. Further work will investigate the underlying molecular mechanisms of Nostrill, its contribution to viral defense in the central nervous system, and its potential role in the development of a model for MS.

**41. Name:** Rajesh Kandel **Institution:** Washburn University of Topeka, KS **Email:** rajesh.kandel@washburn.edu **Research area:** Other**Title:** Constructing a micro Fabry-Perot Cavity System for Raman detection and characterization of chemical properties of nanoscale materials

**Authors:** Rajesh Kandel and Hoang Long Nguyen
Washburn University, Topeka, KS 66614

**Abstract:** Raman spectroscopy is a powerful technique for obtaining structural information about the molecules inside a cell. However, commercial Raman instrument often requires high concentrations of analytes, making it unsuitable for studying biomolecular activities in vivo. Optical microcavities can significantly enhance the Raman signal by concentrating the optical power and increasing the density of states of the Raman scattered light. Microcavities can therefore lower the limit of detection of Raman signal to nanomolar concentrations of analytes. The simplest form of a microcavity is a micro Fabry-Perot cavity, which is formed by two highly reflecting mirrors situated micrometers apart. The distance between the mirror is designed to match the resonance condition of the cavity to the wavelength of the Raman light for maximum amplification. As a result, a precise control of the mirror position is essential to the cavity construction. In this research, the distance between these two mirrors will be controlled by a homebuilt piezo stage to match the resonance condition of the incident and Raman scattered light and maximize the light-particle interaction. We will combine our microcavities with chemically-specific Raman spectroscopy to detect and observe the biomolecular activities of low-concentration species inside a cell.