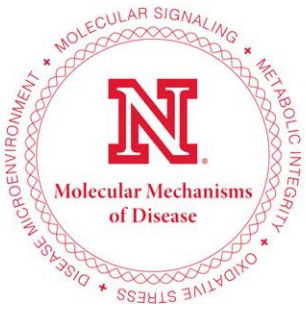

MOLECULAR MECHANISMS OF DISEASE

Predoctoral Training Program

Annual Symposium

April 20, 2016

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

Program Contacts

Director
Melanie Simpson
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Co-director
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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.

Molecular Mechanisms of Disease Internal Advisory Committee

Prem Paul
Vice Chancellor for Research and Economic Development

Ronnie Green
NU Vice President, Chancellor-elect
Harlan Vice Chancellor, Institute of Agriculture and Natural Resources

Brian Larkins
Associate Vice Chancellor for the Life Sciences

Laurie Bellows
Associate Dean of Graduate Studies
Director, McNair Program

Charles Wood
Lewis Lehr Professor of Biological Sciences and Biochemistry
Director, Center for Virology

Theresa Welbourne
FirsTier Banks Distinguished Professor of Business
President and CEO, eePulse, Inc.

If you require special accommodations, please contact Melanie Simpson at msimpson2@unl.edu.

Molecular Mechanisms of Disease Mentoring Faculty

Jiri Adamec, Biochemistry
Mitochondrial dysfunction biomarkers link to environmental stressors

Joseph Barycki, Biochemistry
Structural insights into redox homeostasis

Don Becker, Biochemistry
Role of proline in redox homeostasis and apoptosis

David Berkowitz, Chemistry
Chemical biology and synthetic organic chemistry

Paul Black, Biochemistry
Fatty acid transport in eukaryotes

Deb Brown, School of Biological Sciences
Vaccine strategies that target cytolytic CD4 T cells to the lung

Nicole Buan, Biochemistry
Contribution of methanogenic bacteria to gut function

Andrea Cupp, Animal Science
Role of VEGF in testis morphogenesis

Concetta DiRusso, Biochemistry
High throughput screens for fatty acid uptake inhibitors

Eric Dodds, Chemistry
Chemical glycobiology

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

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

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

Sri Kidambi, Chem & Biomol Engineering
Tissue engineering, stem cells, and liver reconstruction

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

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

Robert Powers, Chemistry
NMR metabolomics

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

Wayne Reikhof, Biological Sciences
Lipid distribution and storage mechanisms

David Ro, Biochemistry
Sestrins and TORC signaling in metabolism

Melanie Simpson, Biochemistry
Steroid elimination, hyaluronan signaling and turnover in prostate cancer progression

Cliff Stains, Chemistry
Novel fluorescent protein design and protein activity switches

James Takacs, Chemistry
Catalytic asymmetric hydroboration and "green chemistry" synthesis

Bill Velander, Chem & Biomol Engineering
cGMP recombinant factor IX for IV and oral hemophilia B therapy

Matthew Wiebe, Vet & Biomedical Science
Immunology and host-pathogen interaction

Mark Wilson, Biochemistry
Redox regulation of DJ-1 function

Jennifer Wood, Animal Sciences
Steroid hormone sensing and signaling

Limei Zhang, Biochemistry
Structural biology, Mycobacterium tuberculosis infectivity

Schedule of events

WEDNESDAY, April 20, 2016

8:00 AM TO 5:30 PM

University of Nebraska Alumni Champion's Club

8:00-8:45 AM Poster set-up and continental breakfast

8:45-9:00 AM Morning session welcome and kickoff presentation

Dr. Melanie Simpson, Program Director

9:00-10:00 AM MORNING PLENARY PRESENTATION

Dr. Stephen Haggarty, Harvard University,
Massachusetts General Hospital
Chemical Genomics of Neuropsychiatric Disorders

10:00-11:00 AM STUDENT PRESENTATIONS I: SENIOR TRAINEES

Christina Davis, Biochemist and Chemical Engineer
Meth dysregulation of function and autophagy in primary astrocytes

Shelbi Christgen, Biochemist and Food Science Technologist
Exploring Helicobacter proline metabolism and substrate channeling

Michelle Palmer, Biochemist and Cancer Biologist
Altered glucuronidation deregulates androgen dependence in prostate tumor cells

11:00-12:00 SESSION I POSTER VIEWING:

Molecular Signaling and Disease Microenvironment

12:00-12:15 PM EXECUTIVE OVERVIEW AND WELCOME

Dr. Ronnie Green, University of Nebraska Chancellor-elect, NU Vice President, Harlan Vice Chancellor of the Institute of Agriculture and Natural Resources, and Interim Senior Vice Chancellor for Academic Affairs

12:15-1:15 PM LUNCH

1:15-2:15 PM AFTERNOON PLENARY PRESENTATION

Dr. Paul Trainor, Stowers Institute for Medical Research,
University of Kansas School of Medicine
The Molecular Mechanisms of Craniofacial Development and Disease

2:15-3:45 PM STUDENT PRESENTATIONS II

Abby Gelb, Mass Spectrometrist and Cancer Biologist
Glycomic profiling of human hyaluronidase 1

Ed Germany, Biochemist and Biomolecular Engineer
Conserved AAA+ ATPase Afg1/LACE1 is a novel factor involved in mitochondrial proteostasis

Travis Nelson, Chemist and Structural Biologist
Probing protein aggregation with sensitive and rapid self-reassembling nanoluciferase fragments

Poster # Shulei Lei, Graduate student in Chemistry
The blockade of glycolysis by α-synuclein over-expression potentiates manganese neurotoxicity

Poster # Fang Xie, Graduate student in Animal Science
Oxidative stress during oocyte in vitro maturation increases the abundances of DPPA3 and POU5F1 maternal effect gene transcripts in matured oocytes and 2-cell embryos, indicative of altered post-transcriptional regulation of maternal mRNAs

3:45-4:45 PM SESSION II POSTER VIEWING:

Oxidative Stress and Metabolic Integrity

5:00 PM AWARD PRESENTATIONS

5:15-6:00 PM Poster take-down

PLENARY SPEAKER PROFILES

Dr. Stephen J. Haggarty is an Associate Professor of Neurology at Harvard Medical School, an Associate Neuroscientist at Massachusetts General Hospital, and Director of the MGH Chemical Neurobiology Laboratory. Dr. Haggarty's research program focuses on dissecting the role of neuroplasticity in health and disease using a combination of chemical genomics, biochemistry, proteomics, and translational neurobiology. A major emphasis of his research has become the use of reprogramming technology to create patient-specific induced pluripotent stem cell (iPSC) models of neuropsychiatric disorders. The ability to differentiate these human iPSCs into electrically active, neural networks with the capacity to form synapses and regulate genes in an activity-dependent manner provides powerful *ex vivo* model systems that enable new avenues for studies of neuroplasticity, for understanding the neurobiology of human disease, as well as for addressing the challenging goal of discovering novel targets and next-generation, disease-modifying, neuro-pharmacological therapies.

Website: www.hms.harvard.edu/dms/bbs/fac/Haggarty.php

Dr. Paul A. Trainor is an Investigator in the Stowers Institute for Medical Research and a Professor in the Department of Anatomy and Cell Biology at the University of Kansas School of Medicine. Research in the Trainor laboratory centers on neural crest cell development, evolution and disease. His group seeks to understand the mechanisms that regulate neural crest cell formation, migration, survival and differentiation. The goal is to apply this basic knowledge to better understand vertebrate evolution and the etiology and pathogenesis of birth defects, as well as to potentially develop therapeutic avenues for their prevention.

Website: http://www.stowers.org/faculty/trainor-lab

Oral Presentation Abstracts
Morning Session

Trainee presentation #1

METH DYSREGULATION OF FUNCTION AND AUTOPHAGY IN PRIMARY ASTROCYTES

Christina Wilson[1], Rick Bevins[2], Rodrigo Franco[3], Oleh Khalimonchuk[4] and Srivatsan Kidambi[1]

[1] Department of Chemical and Biomolecular Engineering, University of Nebraska-Lincoln
[2] Department of Psychology, University of Nebraska-Lincoln
[3] Department of Veterinary and Biomedical Sciences, University of Nebraska-Lincoln
[4] Department of Biochemistry, University of Nebraska-Lincoln

Methamphetamine (METH) is a highly addictive synthetic psychostimulant drug which has been linked to a number of psychological and physiological changes in the brain of chronic abuse cases. Recent studies have indicated the potential role of astrocytes in METH addiction, sensitization and neurotoxicity; however, the underlying molecular mechanisms of the astrocyte mediated effect of METH is not clear. The aim of our study is to understand how METH affects astrocyte health and function. We observed that METH decreased viability and function in a concentration dependent manner but did not induce astrogliosis. Furthermore, we observed that METH induced vacuole accumulation by dysregulation of autophagy flux. These observations indicate that the METH influence on astrocytes results in a concentration dependent decreased in viability and function which could contribute to neurodegeneration in chronic METH abuse cases.

Trainee presentation #2

EXPLORING HELICOBACTER PROLINE METABOLISM AND SUBSTRATE CHANNELING

Shelbi Christgen[1], Amanda Ramer-Tait[2] and Donald Becker[1]

[1] Department of Biochemistry, University of Nebraska-Lincoln [2] Department of Food Science and Technology, University of Nebraska-Lincoln

Proline is one of the preferred respiratory substrates of Helicobacter species; a type of bacteria involved in inflammatory bowel disease, peptic ulcer disease, and cancer. In gram negative bacteria like Helicobacter, the oxidation of proline to produce glutamate is performed by a multifunctional enzyme called proline utilization A (PutA). In gram positive bacteria and eukaryotes, this process is catalyzed by two separate enzymes, proline dehydrogenase (PRODH) and pyrroline-5-carboxylate dehydrogenase (P5CDH). PutA enzymes have two catalytic domains, a PRODH domain and a P5CDH domain, joined by a hollow cavity. This cavity allows for the translocation of the intermediate P5C without equilibration into the surrounding environment. Though the physiological importance of substrate channeling has yet to be shown, recent evidence suggests that monofunctional PRODH and P5CDH enzymes also interact to channel P5C, indicating a potentially important role of channeling. We seek to explore the role of substrate channeling in Helicobacter pathogenicity. It has previously been shown that PutA is required for Helicobacter pylori infection. In addition, the proline transporter PutP and the secreted peptidase collagenase have been shown to be necessary for infection. We hypothesize that collagenase is required because as it breaks down host collagen, free proline is released. This extracellular proline is then utilized by the pathogen as an energy source. A chronic state of inflammation caused by Helicobacter is thought to lead to the development associated host diseases. We hypothesize that the inflammation experienced by the host is the result of proline acquisition by Helicobacter through collagen breakdown.

Trainee presentation #3

ALTERED GLUCURONIDATION DEREGULATES ANDROGEN DEPENDENCE IN PROSTATE TUMOR CELLS

Michelle Palmer, Linlin Ma, Eileen G. Loughman, Trevor Romsdahl, Jonathan E. Markham, Javier Seravalli, Joseph J. Barycki, and Melanie A. Simpson

Department of Biochemistry, University of Nebraska, Lincoln, NE 68588-0664

Prostate epithelial cells control the potency and availability of androgen hormones in part by inactivation and elimination. UDP-glucose dehydrogenase (UGDH) catalyzes the NAD+-dependent oxidation of UDP-glucose to UDP-glucuronate, an essential precursor for androgen inactivation by the prostate glucuronidation enzymes UGT2B15 and UGT2B17. UGDH expression is androgen stimulated, which increases the production of UDP-glucuronate. This multipotent precursor is then directed into three downstream fates: 1) hyaluronan (HA) production, 2) proteoglycan and glycosaminoglycan production, and 3) UGT-catalyzed glucuronidation. Here, we knocked down expression of UGDH in androgen dependent cells to test the role of UGDH in driving cellular glucuronate levels, intratumoral steroid availability, and response to androgen availability. Selection of UGDH-deficient cells in decremental DHT levels that simulated androgen deprivation was found to elevate the potential for UGT-driven steroid clearance, as reflected in de-repression of UGT2B17 and increased secretion of androgen-glucronides. Restimulation of control transfectants with physiological levels of DHT could only maintain full androgen sensitivity in cells that had experienced complete androgen ablation. In contrast, the knockdown of UGDH permitted cells to diminish UDP-glucuronate pools, while maintaining UGT-driven glucuronidation of both exogenously added and cell-synthesized DHT. Androgen-independent cells were also manipulated genetically and chemically to characterize the partitioning of UDP-glucuronate into the alternate downstream fates. This comparison revealed a prioritization of precursor use for HA and PG production, rather than steroid glucuronidation. These results support a model in which maintenance of partial UGDH activity through gradual androgen deprivation facilitates loss of sensitivity in androgen elimination pathways and suggests adjuvant UGDH inhibition could prevent or delay onset of recurrence.

Oral Presentation Abstracts
Afternoon Session

Trainee presentation #4

GLYCOMIC PROFILING OF HUMAN HYALURONIDASE 1

Abby S. Gelb[1], Christine Booth[2], Melanie A. Simpson[2], and Eric D. Dodds[1]

[1]Department of Chemistry and [2]Department of Biochemistry, University of Nebraska, Lincoln, NE 68588

Hyaluronidase 1 (Hyal1) is a key glycoprotein noted at elevated levels for both breast and prostate cancers. Human Hyal1 (hHyal1) is an approximately 51 kDa enzyme containing 435 amino acid residues and three potential N-linked glycosylation sites (Asn 99, Asn 216, and Asn 350). These glycosylation sites have been confirmed as occupied but the glycans and site heterogeneity have not been fully characterized. Nevertheless, a point mutation of Asn 350 to Ala diminishes the activity of the enzyme. Hyal1 is involved in angiogenesis and migratory pathways, as well as an accelerant for metastatic progression. Additionally, hHyal1 is trafficked out of cells via exosomes that have also been shown to be involved in the metastatic process. Here, we have expressed hHyal1 in HEK293F suspension cells to analyze active, physiologically relevant glycoprotein. The anticipated glycomic complexity of hHyal1 has the potential to introduce significant analytical challenges, yet these analyses are expected to provide a level of detail on hHyal1 glycosylation that has thus far remained obscure. hHyal1 has been noted to have three N-linked glycosylation sites and, when considering all types of known or suspected hHyal1 glycan compositions, the number of possible hHyal1 glycoforms totals 114. These compositional possibilities arise from variable degree of branching (di-, tri-, and tetra-antennary structures), the presence of sialylation (on one, some, or all of the branches), and the presence of fucosylation (whether on the chitobiose core or a branch). Therefore, a glycomic investigation of hHyal1 is of significant interest in the context of prostate cancer progression.

Trainee presentation #5

CONSERVED AAA+ ATPase Afg1/LACE1 IS A NOVEL FACTOR INVOLVED IN MITOCHONDRIAL PROTEOSTASIS

Edward Germany, Nataliya Zahayko, Iryna Bohovych, Oleh Khalimonchuk

University of Nebraska-Lincoln, Department of Biochemistry/Redox Biology Center

Mitochondria are complex organelles critical for cellular physiology. Failure to maintain proper mitochondrial function is associated with altered proteostasis, increased oxidative damage, misbalanced ion homeostasis, and decline in ATP production. Post-mitotic cells with high-energy demand, such as cardiomyocytes and neurons, are particularly sensitive to these perturbations. Insults to mitochondrial homeostasis accumulate with age and can lead to many disease states, including neurodegeneration. Mitochondrial integrity is maintained through an intricate network of highly conserved proteases and chaperons known as mitochondrial quality control (MQC). While the importance of MQC is commonly appreciated, the roles of many of its individual components remain unclear. Here, we characterize the conserved mitochondrial AAA+ ATPase Afg1/LACE1. Using the yeast model we show that loss of Afg1 leads to a gradual decline in mitochondrial health, which is associated with enhanced oxidative damage. Synthetic genetic interaction analysis of AFG1 further confirms its functional involvement with well-characterized MQC factors. Remarkably, overexpression of Afg1 can partially compensate for inactivation of the key mitochondrial chaperone, mtHsp70. Consistent with the role of Afg1 in mitochondrial proteostasis, we demonstrate that the depletion of its homolog in the roundworm metazoan model activates mitochondrial unfolded protein response. Animals deficient in Lace-1 exhibit locomotory defects, thereby highlighting physiological significance of this protein. Our results indicate that Afg1/LACE1 is a novel MQC factor, which plays an important evolutionary conserved role in preservation of mitochondrial protein homeostasis.

Trainee presentation #6

PROBING PROTEIN AGGREGATION WITH SENSITIVE AND RAPID SELF-REASSEMBLING NANOLUCIFERASE FRAGMENTS

Travis J. Nelson[1], Jia Zhao[1], Mark A. Wilson[2], & Cliff I. Stains[1]

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

Protein aggregation represents a clear biological consequence of some human diseases, but current fluorescent methods for quantifying these interactions are hindered in their usefulness by key limitations. Recent advancements in luciferase engineering have produced a small, highly active luciferase, dubbed nanoluciferase (NLuc) that we believe can be harnessed to interrogate protein aggregation through bio-luminescence. Using predictive modeling software, we identified key fragmentation sites that yielded rapidly self-reassembling fragment pairs with luminescence that corresponds to only reassembled fragments. The fragments retained this self-reassembling property in vitro and in vivo. Using this, we were able to develop an assay capable of accurately quantifying protein solubility, the aggregation potential of several amyloid-beta (Aβ) mutants (including those related to Alzheimer's disease), and the efficacy of small molecule aggregation inhibitors. Given then this accurate, genetically encodable luminescence assay capable of accurately reporting within intact cells, we predict that platform can be further exploited to discover new aggregation inhibitors through library screens, investigate the role of key amino acids in the aggregation of other amyloid peptides, and answer long standing biological questions about the subcellular localization disease relevant proteins.

Poster #26 Talk

THE BLOCKADE OF GLYCOLYSIS BY α-SYNUCLEIN OVER-EXPRESSION POTENTIATES MANGANESE NEUROTOXICITY

Shulei Lei[1], Jiahui Li[3], Reilly Grealish[3], Annadurai Anandhan[3], Rodrigo Franco [2,3] and Robert Powers[1,2]

[1]Department of Chemistry, [2] Redox Biology Center, [3] School of Veterinary Medicine and Biomedical Sciences. University of Nebraska-Lincoln. Lincoln, NE.

Parkinson's disease (PD) is a neurodegenerative disorder characterized by the loss of dopaminergic cells in the substantia nigra. In addition to genetic factors and aging, environment factors play a significant role on PD prevalence. It is important to understand the interactions between genetic factors and environment exposures, which may lead to a new understanding of the mechanisms of PD etiology. Manganese (Mn) is an essential metal required for various physiological processes by serving as a cofactor for many enzymes. Over-exposure to high level of Mn may cause Mn brain accumulation and result in neurodegeneration, referred to as manganism. Manganism shares many symptoms with idiopathic Parkinson's disease (IPD). Like PD, the neurotoxicity of Mn has been linked to oxidative stress, energy metabolism dysfunction and protein aggregation. α-synuclein mutant is one of four genes found in early-onset PD patients. The aggregation of α-synuclein in the form of Lewy Bodies is a pathological hallmark of PD. It has been reported that Mn can act jointly with α-synuclein to induce a synergistic effect on dopaminergic cell death. However, the primary molecular mechanisms of Mn neurotoxicity, functions of α-synuclein and their interaction remain elusive.

In this study, we observed a synergistic effect from Mn and α-synuclein on the cell death of N27. The N27 cell lines with over-expressed α-synuclein are significantly more susceptible to Mn, even at low concentration. Our results from 1H-13C HSQC with [U-13C] glucose / [U-13C] pyruvate /[U-13C] glutamine reveal upregulated glycolysis after Mn treatment. In contrast we observed downregulated glycolysis for α-synuclein over-expressed cells before and after Mn treatment compared to the control cells. The protective role of glycolysis in Mn toxicity was further demonstrated by our experiments with different carbon sources supplement. Our data showed that cell death induced by Mn is significantly higher when glucose was removed from media. As expected, slowing down glycolysis with galactose increased cell susceptibility to Mn as well. We also observed impaired mitochondrial activity induced by Mn treatment. By piecing together these data, we hypothesize a mechanism of Mn toxicity and the toxicity interaction between Mn and α-synuclein in terms of cellular energy metabolism perturbation. Mn treatment induces cellular energy depletion by impairing the TCA cycle. As a surviving strategy, cells upregulate glycolysis to compensate the energy deficit. However, in α-synuclein over-expressed cells, α-synuclein blocks glycolysis, thus resulting in the loss of this metabolism adaption to defend against Mn toxicity. Our study demonstrates the important and dynamic role of energy metabolism adaption utilized by cells as a survival strategy when exposed to Mn.

Poster #48 Talk

OXIDATIVE STRESS DURING OOCYTE IN VITRO MATURATION INCREASES THE ABUNDANCES OF DPPA3 AND POU5F1 MATERNAL EFFECT GENE TRANSCRIPTS IN MATURED OOCYTES AND 2-CELL EMBRYOS, INDICATIVE OF ALTERED POST-TRANSCRIPTIONAL REGULATION OF MATERNAL mRNAS

F. Xie[1], R. L. Krisher[2], and J. R. Wood[1]

[1] Department of Animal Science, University of Nebraska-Lincoln
[2] Colorado Center for Reproductive Medicine

Maternal mRNAs stored during oocyte growth play important roles in successful oocyte maturation and early embryonic development. In previous studies, we showed that transcript abundance of Dppa3 and Pou5f1 were significantly increased in growing and mature oocytes due in part to exposure to an obesity-dependent inflammatory environment. Oxidative stress and local, chronic inflammation are intimately linked and represent a classic characteristic of obesity. Therefore, our objective was to determine if exposure of oocytes to oxidative stress during maturation impaired post-transcriptional regulation of mRNA abundance. We developed a method to detect and quantify mRNAs in individual oocytes/embryos using single molecule fluorescent in situ hybridization (FiSH). Briefly, complementary DNA probes were hybridized to endogenous Dppa3 or Pou5f1 mRNAs. The mRNA-probe complexes were subsequently hybridized with amplifier reagents which enhanced the fluorescent signal and enabled detection of single mRNA molecules without amplification of mRNA. The analysis was performed using maximum projected images “stitched” together using Image J with a threshold of signal intensity determined using a two-dimensional Gaussian fit. To validate this method, FiSH was performed using in vivo derived immature, mature oocytes, and early embryos collected from 8-week old CD-1 mice specific for Dppa3 or Pou5f1 DNA probes. As expected, all three mRNAs abundances were corresponding with previously published data. To investigate if oxidative stress during maturation, immature cumulus oocyte complexes (COCs) were collected from 8-week-old CD-1 mice. The COCs were in vitro matured (IVM) in the absence (control) or presence of 100 μM H2O2 (H2O2). Both Dppa3 and Pou5f1 were increased in matured oocytes treated with H2O2 compared to control ($P<0.01$). Furthermore, Pou5f1 mRNAs continued to be increased in 2-cell embryos ($P<0.01$) generated from H2O2 treated oocytes. These data suggest that exposure to oxidative stress during oocyte maturation alters post-transcriptional regulation of mRNA abundance, which impacts mRNA storage and potentially protein translation and function in the early embryo.

Poster Presentations

Morning Session:
Molecular signaling and Disease microenvironment

1
INDIVIDUAL AND COMBINED ADMINISTRATION OF GALACTOOLIGOSACCHARIDE AND *BIFIDOBACTERIA ADOLESCENTIS* IVS-1 TO HIGH FAT-FED MICE CAUSES DIFFERENTIAL ADAPTATION OF THE MICROBIOME AND METABOLIC PROFILE
Rafael Segura, Laure Bindels, Janina Krumbeck, Carlos Gomes, Hatem Kittana, Maria Quintero, Elizabeth Cody, Jens Walter and Amanda E. Ramer-Tait.
Department of Food Science and Technology, University of Nebraska-Lincoln

2
PROBING PROTEIN AGGREGATION WITH SENSITIVE AND RAPID SELF-REASSEMBLING NANOLUCIFERASE FRAGMENTS
Travis J. Nelson[1], Jia Zhao[1], Mark A. Wilson[2], & Cliff I. Stains[1]
[1] Department of Chemistry, [2] Department of Biochemistry, University of Nebraska-Lincoln

3
HYALURONAN-BASED SIGNALS MEDIATE STROMAL CELL TRANSFORMATION IN A PROSTATE CANCER CELL MODEL
Christine S. Booth[1], Caitlin O. McAtee[1], Jeremy S. Payne[1], Teresa Fangman[2] and Melanie A. Simpson[1]
[1] Department of Biochemistry, UNL [2] Center for Biotechnology, UNL

4
LOSS OF NEUROPILIN-1 IMPAIRS VEGFA ANGIOGENIC SIGNALING AND ALTERS EXPRESSION OF SPERMATOGONIAL STEM CELL MAINTENANCE MARKERS
Kevin M. Sargent, John R. Essink, Meredith L. Bremer, William E. Pohlmeier, Melissa M. Laughlin, and Andrea S. Cupp
Department of Animal Science, University of Nebraska-Lincoln

5
EXPLORING HELICOBACTER PROLINE METABOLISM AND SUBSTRATE CHANNELING
Shelbi Christgen[1], Amanda Ramer-Tait[2] and Donald Becker[1]
[1] Department of Biochemistry, University of Nebraska-Lincoln [2] Department of Food Science and Technology, University of Nebraska-Lincoln

6
EXPRESSION AND PRELIMINARY CHARACTERIZATION OF HUMAN HYALURONIDASE HYAL2
Eryn Lee and Melanie A. Simpson
Department of Biochemistry, University of Nebraska - Lincoln

7
A ROLE FOR HYALURONIDASE HYAL1 IN THE AUTOPHAGY AND EXOSOME BIOGENESIS PATHWAYS OF PROSTATE TUMOR CELLS

Caitlin McAtee[1], Christine Booth[1], Teresa Fangman[2], Oleh Khalimonchuk[1], Steve Caplan[3,4], Michael Henry[5], Melanie Simpson[1,4]
[1]Department of Biochemistry, University of Nebraska
[2]Microscopy Facility, University of Nebraska
[3]Department of Biochemistry and Molecular Biology, University of Nebraska Medical Center
[4]Fred and Pamela Buffett Cancer Center
[5]University of Iowa Carver College of Medicine

8

ELIMINATION OF VEGFA IN GRANULOSA AND SERTOLI CELLS VIA SRY-CRE DECREASES FERTILITY IN ADULT MICE

Mariah L. Hart, Scott G. Kurz, Kevin M. Sargent, Napoleone Ferrera, Gerrit J. Bouma, and Andrea S. Cupp
University of Nebraska-Lincoln, University of San Diego, Colorado State University

9

COMBINATION PATTERN RECOGNITION RECEPTOR AGONISTS INDUCE DENDRITIC CELL ACTIVATION AND CYTOKINE EXPRESSION IN VITRO

Anna T. Lampe[1], Eric Farris[2], Angela K. Pannier[2], and Deborah M. Brown[1]
[1] School of Biological Sciences and Nebraska Center for Virology, University of Nebraska-Lincoln, NE 68583 USA
[2] Biological Systems Engineering, University of Nebraska-Lincoln, NE 68583 USA

10

HIGH-THROUGHPUT MONITORING OF PROTEIN KINASE PERTURBATIONS USING DIRECT ACTIVITY SENSORS

Garrett R. Casey[1], Xinqi Zhou [1], Jing Wang[2], and Cliff I. Stains[1]
[1] Department of Chemistry, University of Nebraska-Lincoln - Lincoln, NE
[2] Eppley Institute, University of Nebraska Medical Center - Omaha, NE

11

IMPACT OF ESCHERICHIA COLI COLONIZATION ON SUSCEPTIBILITY TO GASTROINTESTINAL INFLAMMATION

Hatem Kittana, João Carlos Gomes, Rafael Segura, Elizabeth Cody and Amanda Ramer-Tait
Department of Food Science and Technology, University of Nebraska-Lincoln, Lincoln, NE, USA

12

ENHANCING NONVIRAL GENE DELIVERY TO HUMAN MESENCHYMAL STEM CELLS (hMSCs) USING GLUCOCORTICOID-ASSOCIATED PATHWAYS

Andrew D. Hamann[1], Abby M. Kelly[2], and Angela K. Pannier[1]
[1]Department of Biological Systems Engineering, University of Nebraska-Lincoln, Lincoln NE
[2]Department of Bioengineering University of Washington- Seattle, Seattle, WA

13

GLYCOMIC PROFILING OF HUMAN HYALURONIDASE 1

Abby S. Gelb[1], Christine Booth[2], Melanie A. Simpson[2], and Eric D. Dodds[1]
[1]Department of Chemistry and [2]Department of Biochemistry, University of Nebraska, Lincoln, NE 68588

14

A COMPARISON OF OVARIAN GENE EXPRESSION PROFILES PROVIDES INSIGHT INTO CELLULAR IDENTITIES AND FUNCTIONS

Sarah M. Romereim[1], Adam F. Summers[2], William E. Pohlmeier[1], Xiaoying Hou[3], Heather A. Talbott[3], Robert A. Cushman[4], Jennifer R. Wood[1], John S. Davis[3], Andrea S. Cupp[1]
[1] University of Nebraska–Lincoln
[2] New Mexico State University
[3] University of Nebraska Medical Center
[4] USDA, ARS, U.S. Meat Animal Research Center

15

THE ROLE OF UDP-GLUCOSE DEHYDROGENASE IN PROSTATE CANCER DISEASE PROGRESSION

Brenna M. Zimmer and Melanie A. Simpson
University of Nebraska, Department of Biochemistry

16

IMAGING ELLIPSOMETRY OF CELLS CULTURED ON OPTICALLY ANISOTROPIC NANOSTRUCTURED SURFACES

Tadas Kasputis[1], Darin Peev[2,3], Albert Nguyen[2,3,4], Amy Mantz[3,4], Eva Franke-Schubert[2,3], Angela K. Pannier[3,4], Mathias Schubert[2,3]
[1] Department of Biomedical Engineering, University of Michigan, Ann Arbor, MI
[2] Department of Electrical and Computer Engineering, University of Nebraska-Lincoln, Lincoln, NE
[3] Center for Nanohybrid Functional Materials, University of Nebraska-Lincoln, NE
[4] Department of Biological Systems Engineering, University of Nebraska-Lincoln, Lincoln, NE

17

MANIPULATION OF UDP-GLUCOSE DEHYDROGENASE (UGDH) STRUCTURE AND FUNCTION BY UNNATURAL AMINO ACID PHOTOCROSSLINKING

George Grady[1], Ashley Thelen[1], Jaleen Albers[1], Tong Ju[2], Jiantao Guo[2], Joseph J. Barycki[1], and Melanie A. Simpson[1]
[1] Department of Biochemistry, University of Nebraska-Lincoln, Lincoln, NE, 68588-0664
[2] Department of Chemistry, University of Nebraska-Lincoln, Lincoln, NE, 68588-0664

18

DEVELOPMENT OF NOVEL FAR RED TO NEAR INFRARED FLUOROPHORES FOR BIOIMAGING

Xinqi Zhou, Rui Lai, Jon R. Beck, Hui Li and Cliff I. Stains
Department of Chemistry, University of Nebraska - Lincoln

19

ELUCIDATING THE CONTRIBUTION OF INTERFERON REGULATORY FACTOR 3 TO EFFECTOR AND MEMORY CYTOTOXIC T CELL RESPONSES AFTER INFLUENZA

Alexander J. Vogel [1,2], Thomas M. Petro [3], and Deborah M. Brown [1,2]
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20

"ZIPPED SYNTHESIS" PROVIDES A CBS INHIBITOR THAT ATTENUATES H_2S LEVELS AND REDUCES NEURONAL INFARCTION IN A RAT STROKE MODEL

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21

THE CANDIDA ALBICANS QSM, FARNESOL, REMODELS THE PERITONEAL CAVITY MICROENVIRONMENT TO PROMOTE INFLAMMATORY RESPONSES IN MICE.

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22

ALTERED GLUCURONIDATION DEREGULATES ANDROGEN DEPENDENCE IN PROSTATE TUMOR CELLS

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23

THE NOVEL DELTAB1 VIRUS AND B1 COMPLEMENTING CELL LINE DEMONSTRATE THAT B1 KINASE SUFFICIENTLY COUNTERS BAF ANTIVIRAL RESPONSE

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24

INTERROGATING THE TEMPORAL DYSREGULATION OF SIGNALING NODES FROM NAFLD TO HCC

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25

THREE-DIMENSIONAL IN VITRO CULTURE MODEL OF GROWTH PLATE CARTILAGE USING ALGINATE HYDROGELS

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Afternoon Session
Oxidative Stress and Metabolic Integrity

26

THE BLOCKADE OF GLYCOLYSIS BY α-SYNUCLEIN OVER-EXPRESSION POTENTIATES MANGANESE NEUROTOXICITY

Shulei Lei[1], Jiahui Li[3], Reilly Grealish[3], Annadurai Anandhan[3], Rodrigo Franco [2,3] and Robert Powers[1,2]
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27

A PORTION OF HEIFERS ATTAINING "EARLY PUBERTY" ARE ANOVULATORY AND HAVE ALTERED SEX HORMONE BINDING GLOBULIN CONCENTRATIONS

Sarah C. Tenley[1], Adam Summers[2], Renata Spuri-Gomes[1], Mohamed A. Abedal-Majed[1], Jeff Bergman[1], Scott Kurz[1], Jennifer Wood[1], Robert Cushman[3], Andrea S Cupp[1]
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28

METH DYSREGULATION OF FUNCTION AND AUTOPHAGY IN PRIMARY ASTROCYTES

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29

CHARACTERIZATION OF PROTEIN INTERACTIONS WITH FREE FATTY ACIDS AND MEASUREMENT OF UNBOUND FREE FATTY ACIDS BY USING HIGH-PERFORMANCE AFFINITY CHROMATOGRAPHY AND ULTRAFAST AFFINITY EXTRACTION

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30

CONSERVED AAA+ ATPase Afg1/LACE1 IS A NOVEL FACTOR INVOLVED IN MITOCHONDRIAL PROTEOSTASIS

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31

MODELING AND VERIFICATION OF A SYNTROPHIC RELATIONSHIP BETWEEN HUMAN GUT MICROBES

Jennie L. Catlett[1], Mikaela Cashman[2], Megan D. Smith[1], Mary Walter[1], Zahmeeth Sakkaff[2], Myra B. Cohen[2], Massimiliano Pierobon[2], Christine Kelley[3] and Nicole R. Buan[1]
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32

INTEGRATING NMR AND LC-MS DATASETS FOR METABOLOMIC PROFILING OF BIOLOGICAL ORGANISMS

Eli Riekeberg[1], Dr. Robert Powers[2], Dr. Mark Wilson[1]
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33

CHANGES IN MITOCHONDRIAL ULTRASTRUCTURE AND BIOENERGETICS UNDER STRESS ARE MITIGATED BY MITOCHONDRIAL METALLOPROTEASE OMA1

Roman M. Levytskyy, Martonio P. Viana, Colton Roessner, Oleh Khalimonchuk
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34

ANALYSIS OF DRUG BINDING WITH SOLUBLE PROTEINS BY USING ULTRAFAST AFFINITY EXTRACTION AND ALPHA1-ACID GLYCOPROTEIN MICROCOLUMNS

Sandya Rani Beeram, Xiwei Zheng and Dr. David Hage
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35

HUMAN SERINE RACEMASE (hSR): EXPRESSION, ACTIVE SITE MUTAGENESIS AND MECHANISTIC STUDIES

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36

STABILIN-MEDIATED CELLULAR INTERNALIZATION OF PHOSPHOROTHIOATE-MODIFIED ANTISENSE OLIGONUCLEOTIDES (ASOs)

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37

CROSSTALK BETWEEN THE INNER AND OUTER MEMBRANE-ASSOCIATED COMPONENTS OF MITOCHONDRIAL QUALITY CONTROL

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38

OPTIMIZATION OF ON-COLUMN ENTRAPMENT CONTAINING HUMAN SERUM ALBUMIN FOR THE STUDY OF DRUG-PROTEIN BINDING BY HIGH PERFORMANCE AFFINITY CHROMATOGRAPHY

Shiden Azaria[1], John Vargas[2] and David Hage[1]
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39

CYTOCHROME OXIDASE ASSEMBLY FACTOR PET117 MEDIATES OLIGOMERIZATION OF HEME A SYNTHASE

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40

DEVELOPMENT AND USE OF A SCANNING NANO-LC SYSTEM WITH SURFACE ENHANCED FLUORESCENCE DETECTION

Elliott Rodriguez[1], John Vargas[1], Ryan Matsuda[1], Benjamin Hage[1], Zhao Li[1], Erika Pfaunmiller[1], Michael Stoller[1], Abhiteja Konda[1], Matt Kottwitz[1], Stephen A. Morin[1], Stephen Gross[2] and David S. Hage[1]
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41

GLYCOFORM ANALYSIS OF ALPHA1-ACID GLYCOPROTEIN BY CAPILLARY ZONE ELECTROPHORESIS WITH FIELD ENHANCED SAMPLE INJECTION

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42

PROTEOMIC CHANGES IN EPILEPTIC PATIENTS INDICATE DISRUPTION OF ENERGY AND REDOX PATHWAYS AND LIPID METABOLISM IN SEIZURE ONSET

Ryan A. Grove[1], Cory Boone[1], Camila Braga[2], Hannah Kyllo[3], Kaeli Samson[4], Kristina Simeone[4], Timothy Simeone[4], Ron Cerny[5], Deepak Madhavan[3], Jiri Adamec[1]
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43

ANTIBIOTIC-ALTERED MICROBIOTA AND ADAPTIVE IMMUNITY INFLUENCE SUSCEPTIBILITY TO DIET-INDUCED OBESITY

Abby L. Geis[1], Hatem Kittana[1], Joao Carlos Gomes Neto[1], Liz Cody[1], Rafael Ricardo Segura Muñoz[1], Sudheer Menon[1], Robert Schmaltz[1], Nicholas Bokulich[2], Martin J. Blaser[2], Amanda Ramer-Tait[1]
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44

STUDIES OF DRUG INTERACTIONS WITH ALPHA1-ACID GLYCOPROTEIN BY USING ON-LINE IMMUNOEXTRACTION AND HPAC

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45

EFFECT OF DIET ON ABILITY OF VASCULAR ENDOTHELIAL GROWTH FACTOR A (VEGFA) ISOFORMS TO ALTER FOLLICULAR PROGRESSION IN BOVINE OVARY

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46

STRUCTURE-MECHANISM RELATIONSHIPS OF WHIB7 IN THE PATHOGENESIS AND ANTIBIOTIC RESISTANCE OF MYCOBACTERIUM TUBERCULOSIS

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47

EXAMINATION OF THE MECHANISM OF FUNCTIONAL SWITCHING IN PROLINE UTILIZATION A

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48

OXIDATIVE STRESS DURING OOCYTE IN VITRO MATURATION INCREASES THE ABUNDANCES OF DPPA3 AND POU5F1 MATERNAL EFFECT GENE TRANSCRIPTS IN MATURED OOCYTES AND 2-CELL EMBRYOS, INDICATIVE OF ALTERED POST-TRANSCRIPTIONAL REGULATION OF MATERNAL mRNAS

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49

GENOME MINING FOR LYSOBACTER NEW NATURAL PRODUCTS

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50

DECODING THE METABOLIC CROSSTALK BETWEEN CANCER-STROMAL CELL IN PANCREATIC CANCER

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51

BIOCHEMICAL AND STRUCTURAL STUDIES OF SESTRIN2 FOR IDENTIFYING COPPER BINDING SITES

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NOTES

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