4th Annual Meeting
The Michigan Physiological Society
Alma College

Date: Thursday - Friday, June 8th-9th, 2017
Location: Alma College – Alma, Michigan
MPS LEADERSHIP 2016-2017

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(Wayne State University)

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A SPECIAL THANK YOU to our Donors!!!

**Diamond Sponsor: $2,000**
Wayne State University, Office of the Vice President for Research (Dr. Stephen Lanier-Vice President)

**Platinum Sponsors: $1,000**
Henry Ford Health System (Dr. Margot LaPointe-Vice President for Research)
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And thank you to the American Physiological Society for ongoing support of MPS!!
Thanks to the following who donated funds to support MPS programming:

Karen Ball
Susan Barman
Kevin Gordish
Robert Lasley
Donal O’Leary
Zhiying Shang

Assia Shisheva
Erica Wehrwein
Jeffrey Ram
Pat Mueller

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Naveen Sharma
Harold Bell
Qinghui Chen
Steve DiCarlo

John Durocher
Steven Elmer
Greg Fink
Nitin Kumar
Suresh Palaniyandi
Pat Mueller

Thanks to the following judges for Oral and Poster awards:

Nour-Eddine Rahlab
Zhiying (Jenny) Shan
Qinghui Chen
Naveen Sharma

Harold Bell
Greg Fink
John Durocher

...and special thanks to Mohamad El Chami for on-site photography, Margaret Shain from the APS for support of the Teachers’ Workshop, the Alma College Department of Integrative Physiology and Health Science faculty and Alma College administrative staff for donating time and effort toward meeting logistics.

Special thanks to the following:

John Davis
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Alex Montoye

Cristy Calhoun, Integrated Health Studies Institute Coordinator
Jodie Reeves, Provost’s Office
Tammy Rees, Events Coordinator
Keynote Speaker
June 8, 2017

J. Kevin Shoemaker, Ph.D.
Professor, and Canada Research Chair,
School of Kinesiology, The University of Western Ontario, London, Ontario Canada

Title: Forebrain Circuitry Associated with Autonomic Function in Humans

Abstract: The framework of homeostasis relies on the intricate communication between the body organs. Generally considered to be driven by brainstem networks, the autonomic nervous system forms a fundamental communication pathway affecting homeostatic adjustments to stress. However, early clinical evidence of neurally-mediated cardiac arrhythmias following brain damage, or severe emotional stress, led to systemic studies of the supramedullary sites, including the forebrain, as a modulator of the sympathetic and parasympathetic nervous systems with important consequences for cardiovascular control. The advent of functional neuroimaging methods enabled Dr. Shoemaker’s exploration of the conscious human cortex within the context of autonomic stress responses. Using isometric handgrip, and lower body negative pressure, as models of exercise and orthostatic stress, respectively, along with functional magnetic resonance imaging methods, our studies have exposed a cortical autonomic network in the human brain that relates to cardiovagal control and efferent sympathetic nerve activity. This network includes the insula cortex, dorsal anterior cingulate cortex, medial prefrontal cortex, hippocampus and posterior cingulate cortex. Both functional activation patterns and cortical thickness studies reinforce the idea that these sites relate specifically to cardiac and muscle sympathetic nerve activity outcomes. Importantly, the cortical autonomic network receives modulatory control from muscle sensory nerves leading to further hypotheses regarding the role of this network to integrate central and peripheral neural signals to enable a highly coordinated cardiovascular response.

Biography: Dr. Kevin Shoemaker is a Professor of Kinesiology with specific training in Exercise Physiology and Neurophysiology. He holds a Canada Research Chair in the Integrative Physiology of Exercise and Health. He received his B.A. in 1990 (Wilfrid Laurier University), and did his graduate training at the University of Waterloo in Kinesiology and Dr. Richard Hughson was his doctoral supervisor from 1992-1996. Following a three-year postdoctoral period at Pennsylvania State University, Division of Cardiology, he took up his current position at the University of Western Ontario in 1999. He currently directs the Neurovascular Research Laboratory and the Laboratory for Brain and Heart Health, the latter focusing on clinical applications. Overall, Dr. Shoemaker studies the neural control of the cardiovascular system in health and disease. Working at the interface of neurophysiology and vascular physiology, his specific programs of research include recruitment strategies of the sympathetic nervous system, brain regions related to autonomic cardiovascular control, and brain blood flow control. These programs overlap in the theme of vascular disease and its impact on neural function and how exercise interventions improve brain health. His research programs are funded by the Canada Research Chair program, the Natural Sciences and Engineering Research Council of Canada, Canadian Institutes of Health Research, and donor-supported project. He currently serves as Associate Editor for Frontiers in Neuroscience, and guest Editor for Autonomic Neuroscience; Basic and Clinical. He has received the Distinguished University Professor award at the University of Western Ontario 2016 and has acted as President of the International Society for Autonomic Neuroscience over 2016-2017.
Thursday, June 8

12:30 – 2:30 pm  **REGISTRATION** - Alma College, [Heritage Center, Lobby]
Pick up meeting materials and lodging information
Representatives from institutions within Michigan and out-of-state will be available during this time to handle inquiries and questions about undergraduate and graduate level programs.

2:30 - 3:15 pm  **WELCOME AND MEETING OPENING**, [Heritage Center, Presbyterian Hall]
- Patrick Mueller, PhD (President) and Karen Ball, PhD (President-Elect)
- Michael Selmon, PhD (Provost and Vice President for Academic Affairs, Alma College)
- Bruce McAttee (Mid-Michigan Regional Director for Senator Gary Peters)

3:15 – 4:15 pm  **KEYNOTE ADDRESS**: [Heritage Center, Presbyterian Hall]

Introduction: Patrick Mueller, PhD (Wayne State University School of Medicine)

**J. Kevin Shoemaker, PhD**
Western University, Ontario, Canada
Department of Kinesiology

**FOREBRAIN NEUROCIRCUITRY ASSOCIATED WITH AUTONOMIC FUNCTION IN HUMANS**

4:30-5:45 pm  **ORAL SESSION #1** (Cardiovascular/Renal)

**Sponsored by MSU Graduate School ******

**Sponsored by MSU College of Natural Sciences ******
[Heritage Center, Presbyterian Hall]

*Session Co-Chairs:*
Amelia Glazier (University of Michigan)
Guodong Pan (Henry Ford Hospital)

4:30-4:45  **Enshe Jiang** (Postdoctoral Fellow)
Michigan Technological University
HIGH SALT INTAKE ACTIVATES OREXIN-TNF SIGNALING IN HYPOTHALAMIC PARAVENTRICULAR NUCLEUS TO INCREASE SYMPATHETIC OUTFLOW IN DAHL SALT-SENSITIVE RATS

4:45-5:00  **Janice Diaz-Otero** (Graduate Student)
Michigan State University
TRANSCIENT RECEPTOR POTENTIAL VANILLOID 4 CHANNEL MEDIATES ENDOTHELIUM-DEPENDANT VASODILATION IN PARENCHYMAL ARTERIOLES
5:00-5:15  **Timothy Bryson** (Graduate Student)
Wayne State University/Henry Ford Hospital
OVEREXPRESSION OF PROSTAGLANDIN E2 EP4 RECEPTOR IMPROVES CARDIAC FUNCTION AFTER MYOCARDIAL INFARCTION

5:15-5:30  **Keyona King-Medina** (Graduate Student)
Wayne State University/Henry Ford Hospital
EXAGGERATED SALT-SENSITIVE HYPERTENSION IN THE ALMS1 (ALSTROM SYNDROME 1) KNOCKOUT RAT

5:30-5:45  **Melanie Flaherty** (Undergraduate Student)
Northern Michigan University
UNLOCKING THE MECHANISM OF RESOLVIN D2 IN VENOUS THROMBOSIS

5:45 – 6:00 pm  Break

6:00 – 7:00 pm  **Strolling Dinner and Poster Viewing** [Hogan Center, Lobby]

7:00 – 8:00 pm  **POSTER SESSION A**, [Hogan Center, Second Floor Lobby]
(Index of presenting authors is on page 26; poster abstracts are on pages 34-66)
*Presenters eligible for awards need to be at or near their posters so that the judges are able to score your presentation.
Representatives from institutions within Michigan and out-of-state will be available during this time to handle inquiries and questions about undergraduate and graduate level programs.

8:00 – 10:00 pm  Welcome Reception  [Hogan Lawn]
Featuring lawn games and local music from the “10-centers”.

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**Friday, June 9**

7:30 – 8:30 am  **REGISTRATION**, [Heritage Center, Lobby]
Continental Breakfast [Heritage Center, Lobby]
Representatives from institutions within Michigan and out-of-state will be available during this time to handle inquiries and questions about undergraduate and graduate level programs.

8:25 – 8:30 am  **MORNING ANNOUNCEMENTS**, [Heritage Center, Presbyterian Hall]

8:30 – 10:15 am  **ORAL SESSION #2 (Endocrinology/Physiological Genomics)**
*****  **Sponsored by MSU Office of Regulatory Affairs ******
*****  **Sponsored by Univ of MI - Dept of Mol & Integr Physiology ******
[Heritage Center, Presbyterian Hall]

**Session Co-Chairs:**
- **Ian M. Greenlund** (Michigan Technological University)
- **Nguyen Truong** (Michigan State University)

8:30-8:45  **Leena Kadam** (Postdoctoral Fellow)
Wayne State University
PPAR-γ RESCUES ENDOTOXIN MEDIATED EFFECTS ON INFLAMMATION AND TROPHOBLAST PHYSIOLOGY IN HUMAN PLACENTA

8:45-9:00  **Xingfa Han** (Graduate student)
University of Michigan
TRANSCRIPTOME ANALYSIS OF HYPOTHALAMIC NUCLEI DURING LEPTIN-INDUCED PUBERTAL DEVELOPMENT
9:00-9:15 **Naiomy Rios-Arce** (Graduate student)
Michigan State University
INTERLEUKIN-10 DEFICIENCY IN TYPE 1 DIABETES-INDUCED BONE LOSS IN MICE

9:15 – 9:30 am Break (Coffee and beverages available – Heritage Center, Lobby)

9:30-9:45 **Anne Tonson**
Michigan State University (Postdoctoral Fellow)
MUSCLE ACETYLCARNITINE: INFLUENCE OF EXERCISE AND GLUCOSE HOMEOSTASIS

9:45-10:00 **Juliette Brown** (Graduate Student)
Michigan State University
DISTINCT SUBPOPULATIONS OF NEUROTENSIN NEURONS IN THE LATERAL HYPOTHALAMIC AREA CONTRIBUTE TO ENERGY BALANCE BY DISCRETE MECHANISMS AND PROJECTIONS

10:00-10:15 **Joseph Bires** (Undergraduate student)
Oakland University
REPROGRAMMING SOMATIC CELLS INTO INDUCED ROD PHOTORECEPTORS

10:20 - 11:00 am Concurrent Breakout Sessions and Poster Viewing

**Session A: Joining a Professional Society**

***** Sponsored by MSU College of Medicine *****
[Swanson Academic Center – Room 110]
Chair: Sue Barman (MSU)

**Session B: “Elevator Pitches for Trainees: Implementing the 3 Minute Thesis in Our Department”** [Swanson Academic Center – Room 113]

***** Sponsored by WSU School of Medicine - Dean’s Office *****

Chair: Charles Chung (WSU),
Panelists: Tim Bryson (WSU); Joseph Mannozzi (WSU)

**Session C: Searching for a “Postdoc”**

***** Sponsored by Michigan Technological University *****
[Swanson Academic Center – Room 109]
Chair: Isola Brown (MSU)
Panelists: Leena Kadam (WSU); Vlado Grubisic (MSU);
Anne Dorrance (MSU); Harold Bell (CMU),

**Session D: Poster previewing**, [Hogan Center, Second Floor Lobby]

11:00 am -11:15 pm Break
11:15 am - 12:15 pm  ORAL SESSION #3 (Neuro/Respiratory Physiology)  
***** WSU SOM Vice Dean for Research *****  
[Heritage Center, Presbyterian Hall] (see list below)  

Session Co-Chairs:  
Mohamad El Chami (Wayne State University)  
Alexandra Cara (University of Michigan)  

11:15 - 11:30 Cameron Kortes (Medical student)  
Central Michigan University  
THE EFFECTS OF FIVE CLINICALLY RELEVANT NARCOTIC DRUGS ON THE GENERATION OF SPONTANEOUS AUGMENTED (SIGH) BREATHS  

11:30 - 11:45 Ninotchska Delvalle (Graduate Student)  
Michigan State University  
THE NEUROKININ-2 RECEPTOR ANTAGONIST GR 159897 PROTECTS AGAINST NEUROINFLAMMATION IN THE MOUSE ENTERIC NERVOUS SYSTEM DURING COLITIS  

11:45 - 12:00 Yuan Fan (Graduate Student)  
Michigan Technological University  
OREXIN A CAN INCREASE PROINFLAMMATORY CYTOKINE EXPRESSION IN PC12-OX1 CELLS  

12:00 - 12:15 Vladimir Grubisic (Postdoctoral Fellow)  
Michigan State University  
ENTERIC GLIAL CELLS ACUTELY REGULATE SECRETOMOTOR FUNCTION IN THE MOUSE COLON  

12:15 - 12:30 Isola Brown (Graduate Student)  
Michigan State University  
DYSREGULATION OF GLUTATHIONE SYNTHESIS BY ENTERIC GLIA DURING GASTROINTESTINAL INFLAMMATION  

12:30 - 1:30 pm Lunch for Main Meeting and Workshop Attendees (Hogan Center, Lobby)  
*Representatives from institutions within Michigan and out-of-state will be available during this time to handle inquiries and questions about under-graduate and graduate level programs.  

1:30 - 2:30 pm POSTER SESSION B for Main Meeting and Workshop Attendees  
[Hogan Center, Second Floor Lobby] (see list below)  
(Index of presenting authors is on page 26; poster abstracts are on pages 34-66)  

2:30 - 3:30 pm ORAL SESSION #4 (Physiology Education)  
***** WSU Office of Graduate Programs *****  
[Heritage Center, Presbyterian Hall]  

Session Co-Chairs:  
Ross Michaels (Michigan Technological University)  
Trevor G Gohl (Michigan State University)  

2:30 - 2:45 pm Christine Klingert (Medical Student)  
Wayne State University  
EFFECT OF EARLY PEDIATRIC DISABILITY EXPOSURE IN MEDICAL EDUCATION
2:45-3:00 pm  *Soumya Kulkarni* (High School Student)  
Northville High School  
FOSTERING INTEREST AND UNDERSTANDING OF NEUROPHYSIOLOGICAL CONCEPTS THROUGH THE USE OF INTERACTIVE DEMONSTRATIONS

3:00-3:15 pm  *Valerie VanRyn* (Post Baccalaureate)  
Michigan State University  
BUILDING COMMUNITY BY SERVING THE COMMUNITY – PHUN DAY

3:15-3:45 pm  *Keynote Speaker - Steve Elmer, PhD* (Assistant Professor)  
Michigan Technological University  
THE TWO HOUR MARATHON: WHAT DO STUDENTS THINK?

3:45-4:00 pm  **Break** (Beverages and snacks) / **Judges Convene**

4:00-4:10 pm  **Group Picture, [Heritage Center, Front Steps]**

4:15-5:00 pm  **BUSINESS MEETING & AWARDS PRESENTATION**  
[Heritage Center, Presbyterian Hall]

**Trainee Awards**  
*Sponsored by WSU Office for the Vice President of Research*  
Oral Presentations - awards given to the top rated oral presentations  
Poster Presentations - awards given to the top rated poster presentations

Please mark your calendars for MPS 2018!!

Michigan Technological University, Houghton, MI  
Michigan Physiology Quiz Bowl - June 13, 2018  
Annual Meeting - June 14-15, 2018
REGISTRATION, [Heritage Center, Lobby]
Continental Breakfast [Heritage Center, Lobby]
Representatives from institutions within Michigan and out-of-state will be available during this time to handle inquiries and questions about undergraduate and graduate level programs.

MORNING ANNOUNCEMENTS, [Heritage Center, Presbyterian Hall]

Welcome and Introductions, [SAC 110]

Keynote Address: Dr. Kevin Shoemaker, [SAC 110]
Western University, Ontario Canada
Department of Kinesiology
Effect of Exercise Training on Brain Health

Break [refreshments available in the Heritage Center Lobby]

Concurrent Breakout Sessions and Poster Viewing

Breakout Session #1 (10:20-10:40): Career Panel, [SAC 108]
Travis Wakeham, Graduate Student, Michigan Technological University
Naveeen Sharma, PhD, Assistant Professor, Central Michigan University

Breakout Session #2 (10:40-11:00): Select from the following
(Please refer to page 14 for summaries of each session)

Session A: Joining a Professional Society
Sue Barman (MSU), [Swanson Academic Center – Room 110]

Session B: Elevator Pitches for Trainees: Implementing the 3 Minute Thesis in Our Department
Charles Chung (WSU) and Tim Bryson (WSU), [Swanson Academic Center – Room 113]

Session C: Searching for a Postdoc
Isola Brown (MSU) and Leena Kadam (MSU), [Swanson Academic Center – Room 109]

Session D: Poster previewing, [Hogan Center, Second Floor Lobby]

Hands-on Learning Activity [Human Performance Lab]
Sue Speirs, Grosse Pointe North
Steward Jensen, Department of Physics, Alma College
Blood Flow Dynamics

Lab Demonstration – CV control during Lower Body Negative Pressure; forearm blood flow and blood pressure regulation. John Davis, Integrative Physiology and Health Science, Alma College [Human Performance Lab]
12:45 – 1:30 pm  **Lunch for Main Meeting and Workshop Attendees** [Hogan Center, Lobby]

1:30 -2:30 pm  **POSTER SESSION B for Main Meeting and Workshop Attendees**
[Hogan Center, Second Floor Lobby] (see list below)
(Index of presenting authors is on page 26; poster abstracts are on pages 34-66)

2:30-3:30 pm  **ORAL SESSION #4 (Physiology Education)**
[Heritage Center, Presbyterian Hall]

★★★★ **WSU OFFICE OF GRADUATE PROGRAMS ★★★★

*Session Co-Chairs:*
Ross Michaels (Michigan Technological University)
Trevor G Gohl (Michigan State University)

2:30-2:45 pm  **Christine Klingert** (Medical Student)
Wayne State University
EFFECT OF EARLY PEDIATRIC DISABILITY EXPOSURE IN MEDICAL EDUCATION

2:45-3:00 pm  **Neha Chava and Soumya Kulkarni** (High School Students)
Northville High School
FOSTERING INTEREST AND UNDERSTANDING OF NEUROPHYSIOLOGICAL CONCEPTS THROUGH THE USE OF INTERACTIVE DEMONSTRATIONS

3:00-3:15 pm  **Valerie VanRyn** (Post Baccalaureate)
Michigan State University
BUILDING COMMUNITY BY SERVING THE COMMUNITY – PHUN DAY

3:15-3:45 pm  **Keynote Speaker - Steve Elmer, PhD** (Assistant Professor)
Michigan Technological University
THE TWO HOUR MARATHON: WHAT DO STUDENTS THINK?

3:45-4:00 pm  **Break** (Beverages and snacks)/Judges Convene

4:00-4:10 pm  **Group Picture,** [Heritage Center, Front Steps]

4:15-5:00 pm  **BUSINESS MEETING & AWARDS PRESENTATION**
[Heritage Center, Presbyterian Hall]

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**Mark your calendars and see you next year for MPS 2018!!**

**Michigan Technological University, Houghton, MI**

**June 14-15, 2018**
4th Annual Meeting of the Michigan Physiological Society  
Alma College, Alma Michigan

HIGH SCHOOL STUDENT PROGRAM  
Friday June 9, 2017

7:30 – 8:30 am  REGISTRATION, [Heritage Center, Lobby]  
Continental Breakfast [Heritage Center, Lobby]

8:25 – 8:30 am  MORNING ANNOUNCEMENTS, [Heritage Center, Presbyterian Hall]

8:35 – 9:00 am  Welcome and Introductions [SAC 110]

9:00 – 10:00  Keynote Address: Dr. Kevin Shoemaker [SAC 110]  
Effect of Exercise Training on Brain Health

10:00 – 10:15  Break [refreshments available in the Heritage Center Lobby]

10:20 - 11:00 am  Concurrent Breakout Sessions and Poster Viewing

Breakout Session #1 (10:20-10:40): Career Panel, [SAC 108]  
Travis Wakeham, Graduate Student, Michigan Technological University  
Naveeen Sharma, PhD, Assistant Professor, Central Michigan University

Breakout Session #2 (10:40-11:00): Select from the following  
(Please refer to page 14 for summaries of each session)

Session A: Joining a Professional Society  
Sue Barman (MSU), [Swanson Academic Center – Room 110]

Session B: Elevator Pitches for Trainees: Implementing the 3 Minute Thesis in Our Department  
Charles Chung (WSU) and Tim Bryson (WSU), [Swanson Academic Center – Room 113]

Session C: Searching for a Postdoc  
Isola Brown (MSU) and Leena Kadam (MSU), [Swanson Academic Center – Room 109]

Session D: Poster previewing, [Hogan Center, Second Floor Lobby]

11:00 am -11:15 pm  Break

11:15 am -12:15 pm  ORAL SESSION #3 (Neuro/Respiratory Physiology)  
***** WSU SOM Vice Dean for Research *****  
[Heritage Center, Presbyterian Hall] (see list below)
Session Co-Chairs:
Mohamad El Chami (Wayne State University)
Alexandra Cara (University of Michigan)

11:15-11:30 **Cameron Kortes** (Medical student)
Central Michigan University
THE EFFECTS OF FIVE CLINICALLY RELEVANT NARCOTIC DRUGS ON THE GENERATION OF SPONTANEOUS AUGMENTED (SIGH) BREATHS

11:30-11:45 **Ninotchka Delvalle** (Graduate Student)
Michigan State University
THE NEUROKININ-2 RECEPTOR ANTAGONIST GR 159897 PROTECTS AGAINST NEUROINFLAMMATION IN THE MOUSE ENTERIC NERVOUS SYSTEM DURING COLITIS

11:45-12:00 **Yuan Fan** (Graduate Student)
Michigan Technological University
OREXIN A CAN INCREASE PROINFLAMMATORY CYTOKINE EXPRESSION IN PC12-OX1 CELLS

12:00-12:15 **Vladimir Grubisic** (Postdoctoral Fellow)
Michigan State University
ENTERIC GLIAL CELLS ACUTELY REGULATE SECRETOMOTOR FUNCTION IN THE MOUSE COLON

12:15-12:30 **Isola Brown** (Graduate Student)
Michigan State University
DYSREGULATION OF GLUTATHIONE SYNTHESIS BY ENTERIC GLIA DURING GASTROINTESTINAL INFLAMMATION

12:30 – 1:30 pm **Lunch for Main Meeting and Workshop Attendees** (Hogan Center, Lobby)
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1:30 -2:30 pm **POSTER SESSION B for Main Meeting and Workshop Attendees**
[Hogan Center, Second Floor Lobby] (see list below)
(Index of presenting authors is on page 26; poster abstracts are on pages 34-66)

2:30 – 3:45 pm **Lab Demonstration** – CV control during Lower Body Negative Pressure; forearm blood flow and blood pressure regulation. *John Davis, Integrative Physiology and Health Science, Alma College* [Human Performance Lab]

3:45-4:00 pm **Break** (Beverages and snacks)/**Judges Convene**

4:00-4:10 pm **Group Picture**, [Heritage Center, Front Steps]

4:15-5:00 pm **BUSINESS MEETING & AWARDS PRESENTATION**
[Heritage Center, Presbyterian Hall]
2017 MPS Breakout Session Summaries

Session A: Joining a Professional Society
Sue Barman (MSU), [Swanson Academic Center – Room 110]

This session will discuss benefits of joining a professional society like the American Physiological Society as well as MPS and being an active participant. That is, it is not enough to just pay dues; rather, you should be actively engaged in committees and meetings. You will learn how you can make a difference to your profession.

Session B: Elevator Pitches for Trainees: Implementing the 3 Minute Thesis
Charles Chung (WSU) [Swanson Academic Center – Room 113]

There is an increasing need and demand for trainees to practice delivering presentations and speaking to public audiences. The Three Minute Thesis is a global program that provides an opportunity for trainees to engage in both activities. We’ll discuss this need, how we set up for the program in our Department, and provide a demo with current graduate students.

Session C: Searching for and transitioning to a Postdoc
Isola Brown (MSU) and Leena Kadam (WSU), [Swanson Academic Center – Room 109]

This breakout session will offer advice on navigating the search for a postdoc and how to successfully transition from graduate student to postdoc. Panelists will include junior and senior level postdocs, as well as faculty who all have a diverse training background. This breakout session would be particularly valuable to senior level grad students, and faculty who would like to gain more information on training postdocs.
Increased sympathetic nerve activity (SNA) plays an important role in the development of salt-sensitive hypertension (SSHTN). However, the detailed mechanisms underlying high salt (HS) induced increase in SNA are not well understood. In this study, eight-week-old male Dahl salt-sensitive (Dahl S) rats and Sprague Dawley (SD) rats were grouped and fed either a HS (8% NaCl) or normal salt (NS, 0.4% NaCl). Four weeks following different diets treatment, animals were euthanized, and PVN tissues were punched out for the real time PCR assay. The results showed that HS diet resulted in significant increase in mRNA levels of prepro-orexin, orexin receptor I (OX1R), TNFα and Fra1. These results suggest that a HS diet triggers orexin-OX1R-TNFα signaling in the PVN which may contribute to increased SNA and development of SSHTN.

CHRONIC HYPERTENSION INCREASES THE RISK OF VASCULAR DEMENTIA

In this study, eight-week-old male Dahl salt-sensitive (Dahl S) rats and Sprague Dawley (SD) rats were grouped and fed either a HS (8% NaCl) or normal salt (NS, 0.4% NaCl). Four weeks following different diets treatment, animals were euthanized, and PVN tissues were punched out for the real time PCR assay. The results showed that HS diet resulted in significant increase in mRNA levels of prepro-orexin, orexin receptor I (OX1R), TNFα and Fra1. These results suggest that a HS diet triggers orexin-OX1R-TNFα signaling in the PVN which may contribute to increased SNA and development of SSHTN.
OVEREXPRESSION OF PROSTAGLANDIN E2 EP4 RECEPTOR IMPROVES CARDIAC FUNCTION AFTER MYOCARDIAL INFARCTION

Timothy Bryson1,2, Xiaosong Gu1, Liping Zhu1, Jiang Xu1, Edward Peterson3, Xiao-Ping Yang1 and Pamela Harding1,2.
1Hypertension & Vascular Research Division, Dept. Internal Medicine; 2Dept. of Physiology, Wayne State University School of Medicine, 3Dept. of Public Health Sciences Henry Ford Hospital, Detroit, Michigan, USA.

Prostaglandin E2 (PGE2) signals through 4 receptor sub-types (EP1, EP2, EP3 and EP4) to elicit a variety of biological effects. Our laboratory has reported that male mice with cardiomyocyte-specific deletion of EP4 KO mice develop dilated cardiomyopathy and that PGE2 via EP3 reduces cardiac contractility. We thus hypothesized that overexpression of EP4 improves cardiac function. This was tested in a mouse model of myocardial infarction (MI) with the use of AAV9-EP4 driven by the myosin heavy chain promoter. MI was produced in 10-12 wk. old C57/Bl6 male mice by ligation of the left anterior descending coronary artery and mice received either 1x10^{12} viral particles of AAV9-EP4 or the same dose of control virus (AAV9-luc) into the left ventricle (LV) free wall. Sham-operated mice also received viral injections. At 2 wk. post-surgery, echocardiography was performed and hearts were harvested. Overexpression of EP4 improved shortening fraction after MI (40.3 ± 2.5 vs 28.2 ± 2.9%, p<0.005) and ejection fraction (47.7 ± 2.4 vs 37.8 ± 2.2%, p<0.05) with a reduction in left ventricular dimension at systole (2.13 ± 0.16 vs 2.98 ± 0.26 mm, p<0.01). Myocyte cross-sectional area (MCSA) was increased after MI (p = 0.005) and this increase was attenuated by administration of AAV9-EP4 (p<0.01). Similar changes were also observed in interstitial collagen fraction. Macrophage migration was also increased in the LV of mice subject to MI (74.7 ± 5.4 cells/mm² for sham operated mice vs 283.5 ± 15.1 for MI mice receiving AAV9-luc) and was reduced to 122.2 ± 5.9 cells/mm² for MI mice receiving AAV9-EP4 (p<0.0001). In conclusion, our results support an anti-inflammatory role for PGE2 via EP4 in the myocardium and suggest that overexpression of the EP4 receptor protects against the decline in cardiac function post MI.

EXAGGERATED SALT-SENSITIVE HYPERTENSION IN THE ALMS1 (ALSTROM SYNDROME 1) KNOCKOUT RAT

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We recently found that a protein named ALMS1 (Alstrom syndrome 1) is expressed in the kidney thick ascending limb (TAL) where it mediates endocytosis of the renal Na/K/2Cl cotransporter termed NKCC2. To study the role of ALMS1 in renal physiology we generated ALMS1 knockout (KO) rats in a Dahl salt-sensitive genetic background via zinc-finger nuclease targeting. We previously found that the amount of NKCC2 in the apical surface is higher in the TALs from ALMS1 KO rats compared to WT- Salt-Sensitive (SS) rats. In order to determine the role of NKCC2 on blood pressure (BP) we utilized both noninvasive tail cuff measurements and invasive radio-telemetry to study the effects of dietary sodium (.22% Na chow and 4% Na chow) on the systolic blood pressure (SBP) of the ALMS1 KO rats and WT-SS rats. We hypothesized that deletion of the ALMS1 gene will increase SBP and enhance salt-sensitivity of BP, in part due to higher NKCC2-mediated Na reabsorption. First, we used tail cuff measurements to obtain the SBP in both groups of rats. We found that with normal Na intake (0.22% Na chow), the ALMS1 KO rats had a higher SBP than the WT-SS rats (KO: 136±3 and WT-SS: 125±3 mmHg, p=0.0461). To further explore the salt-sensitive response of these rats we utilized radio-telemetry monitoring for 4 weeks. We found the ALMS1 KO rats to have a higher baseline SBP on .22% Na chow compared to the WT-SS rats (KO: 145±2 and WT-SS: 134±1 mmHg, p=0.0009). After 2 weeks of high Na intake (4% Na chow) the SBP in ALMS1 KO rats increased to 181±1 mmHg, a 35±3 mmHg increase, whereas the SBP increased to 159±2 mmHg in WT-SS rats, a 25±1 mmHg increase (p<0.01 vs ALMS1 KO). Thus, the SBP was higher in ALMS1 KO rats fed a high salt diet (p=0.0001). We then explored the involvement of NKCC2 in the hypertension observed in ALMS1 KO rats. A daily dose of the loop diuretic bumetanide (3mg/kg), an NKCC2 inhibitor, decreased the SBP in both groups (KO: -23±4 and WT: -30±6 mmHg, p=0.40). After 7 days of treatment with bumetanide, the SBP was normalized only in the WT-SS rats while the SBP remained elevated in the ALMS1 KO rats (KO: 131±3 and WT: 115±4 mmHg, p<0.025). Therefore, we conclude that the ALMS1 KO rats have a higher salt-sensitivity of BP than the WT- (Dahl) SS rats and this in part is mediated by enhanced renal sodium reabsorption in the TAL. Furthermore, our data suggest that additional mechanisms are likely involved in the hypertension observed in ALMS1 KO rats fed a high salt diet.
Deep vein thrombosis (VT) is a serious condition that affects nearly one million people every year. Current treatment for VT is anticoagulation medication, but even with the standard treatment of care only a third of patients experience complete resolution of the thrombus. Therefore, a more effective methodology for the treatment of VT is necessary. Resolvin D2 (RvD2) is an anti-inflammatory molecule derived from Omega 3. RvD2, although studied in a number of cardiovascular diseases, has yet to be looked at as a possible treatment for VT. The objective of this work was to develop an in vitro model using murine vein endothelial cells (B6MPVE) in order to investigate the effects of RvD2 on the resolution of VT. Preliminary in vivo testing done by the Diaz labs shows a significant decrease in thrombus weight following RvD2 administration. Following the successful creation of the model, key proteins identified through primary literature were studied to ascertain the role they played. B6MPVE cells were cultured in one of four different conditions 1) no treatment, 2) thrombin only, 3) RvD2 only or 4) thrombin and RvD2. RNA was extracted from the B6MPVE cells and their relative expression of numerous thrombotic and inflammatory markers were quantified via qRT-PCR. Following analysis of the data, it was concluded that RvD2 decreases pro-inflammatory proteins while increasing anti-inflammatory proteins through the GSK3-β pathway.
PPAR-γ RESCUES ENDOTOXIN MEDITATED EFFECTS ON INFLAMMATION AND TROPHOBLAST PHYSIOLOGY IN HUMAN PLACENTA
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Approximately 27% of pregnant women suffer from pregnancy related disorders. Abnormal placental function and inflammation are common features in the wide spectrum of pregnancy related disorders. In the current study, we aimed to evaluate the relationship between inflammation and trophoblast cell physiology. The transcription factor PPAR-γ is known to regulate gene expression in inflammation, cell differentiation and oxidative stress. The drug Rosiglitazone is a potent agonist for PPARγ. We hypothesized that endotoxin exposure induces an inflammatory response in the placenta and impairs trophoblast differentiation and induction of PPAR-γ activity will reverse these effects. 1st trimester placental villous explants were dissected out and exposed to 1µg/ml of bacterial endotoxin Lipopolysaccharide (LPS) +/- 10µM Rosiglitazone (PPAR-γ agonist) for 24 hrs at 37°C, 8% O2. Exposure to LPS significantly upregulated the expression of inflammatory cytokines (n=12, ≥ 1.7 fold) CCL5 (p= 0.03), IL-1β (p=0.01) and IL-10 (p=0.03). LPS exposure also induced apoptosis (≥2.1 fold, p= 0.02) and decreased proliferation of trophoblasts in treated explants (lowered by ≥40%, p= 0.03). It also upregulated the expression of the major LPS receptor TLR4. The expression of genes involved in trophoblast differentiation was also significantly downregulated (~40%) - β-hCG (p= 0.009) and Gcm1 (p=0.03). Rosiglitazone treatment reversed the effects of endotoxin exposure. It significantly downregulated the levels of inflammatory proteins and reduced apoptosis. It also rescued the expression of β-hCG and GCM1 and restored the proliferation rate to that of untreated controls. We report for the 1st time that exposure of 1st trimester human placenta to endotoxins, in addition to inducing an inflammatory response also regulates receptor expression and factors involved in trophoblast differentiation suggesting a link between inflammation and altered placental function. Further, these effects were rescued by PPAR-γ activation, suggesting a crucial role of PPRA-γ in regulating trophoblast differentiation and placental inflammatory response providing a potential target for future treatments.

TRANSCRIPTOME ANALYSIS OF HYPOTHALAMIC NUCLEI DURING LEPTIN-INDUCED PUBERTAL DEVELOPMENT
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The link between childhood obesity and the increasing rates of early puberty in girls is well documented. Early menarche is associated with increased risk of adult obesity, type 2 Diabetes and breast cancer. The adipocyte-derived hormone leptin is secreted in proportion to fat mass and is a permissive factor for the onset of puberty. Our laboratory has identified the hypothalamic ventral premamillary nucleus (PMV) as an essential relay of leptin action on reproduction. However, the underlying mechanisms associated with this physiological regulation are unknown. In this study, we aimed to identify key PMV genes associated with leptin action on pubertal maturation using RNA-seq analysis. Mice were divided into three groups: a) diestrous females treated with saline; b) leptin-deficient ob/ob females treated with ip. saline; and c) ob/ob females treated with ip. leptin. After 4 injections of leptin, puberty onset was observed by the occurrence of vaginal opening. All animals were euthanized one hour after the last saline or leptin injection (2nd day at 9:00 AM). Brains were quickly dissected out and blocks containing the PMV and the arcuate nucleus were micro-dissected and collected for RNA-seq analysis. We identified 563 differentially expressed genes (DEGs) comparing diestrous and ob/ob+saline mice. Gene ontology analyses showed these DEGs are mainly involved in developmental and reproductive processes. Following leptin administration, the expression of 334 (59%) DEGs was recovered to diestrous levels. These recovered DEGs are mainly involved in developmental and reproductive processes, and anatomical morphogenesis. Several key recovered DEGs (e.g., Shh, Wnt7a, Gipr, Ccl17, Shox2, Zgrp1 and Pla2g3) were validated and defined as part of PMV or arcuate nucleus transcriptome using qPCR from punches. Our results provide molecular insights into the metabolic control of pubertal maturation. Further studies will be conducted to assess the role of these candidate genes in reproductive function.
INTERLEUKIN-10 DEFICIENCY IN TYPE 1 DIABETES-INDUCED BONE LOSS IN MICE
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The prevalence of diabetes is increasing worldwide. In addition to the commonly known complications of this disease, it is well known that Type 1 diabetes (T1D) can have a detrimental effect on bone health. During T1D onset there is an increase in systemic and local cytokine production, suggesting that cytokines could play a regulatory role in bone loss during diabetes. Role of the anti-inflammatory interleukin 10 (IL-10) in bone physiology is well known. However, the role of IL-10 in bone health during short- and long-term diabetes has not been studied. We hypothesized that deficiency of IL-10 in mice will lead to exacerbated bone loss in T1D. To test this, IL-10 knockout and the corresponding C57BL/6J wild-type mice were subjected to streptozotocin injection for 5 days to induce T1D. Diabetes was confirmed two weeks after the first injection by a blood glucose level of >300 mg/dL. Bones were collected at 4 weeks and 12 weeks post-diabetes induction. Bone volume fractions (BVF) of femur was analyzed using microcomputed tomography system. As expected T1D induced significant trabecular bone loss in WT mice at both 4 and 12 weeks after induction (BVF @ 4 weeks: WT control 29% VS WT STZ 15.9%; @12 weeks: WT control: 34.6% VS WT STZ: 9.6%; n=8-10). However, deficiency of IL-10 exacerbated bone loss at the 4-week time point (BVF @ 4 weeks WT STZ: 15.9% VS KO STZ: 8.7%; n=7), but by 12 weeks T1D BVF did not differ from WT mice. We further found that cortical bone was affected in a comparable manner. Bone effects corresponded to changes in osteoblast RNA markers but not osteoclast RNA markers, suggesting that IL-10 primarily affects anabolic events in the T1D bone. Taken together, these results suggest that IL-10 is required for osteoblast regulation during early diabetes-induced bone loss.

MUSCLE ACETYL Carnitine: INFLUENCE OF EXERCISE AND GLUCOSE HOMEOSTASIS
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Skeletal muscle acetylcarnitine (AC) is reportedly involved in glucose homeostasis and metabolic flexibility. Muscle AC content is also positively associated with muscle oxidative capacity and whole body insulin sensitivity. However, the acute effects of insulin-stimulated glucose uptake on muscle AC are still unknown. In this study, we evaluated the influence of an overnight fast and oral glucose challenge on muscle AC as well as the effect of exercise. Muscle AC was measured in the medial gastrocnemius using localized proton magnetic resonance spectroscopy (MRS) at 3T (PRESS, TR/TE=4000/350ms, voxel=30ml, time=5.30min). Eleven healthy participants (7 males) were tested (age=28±3 years old (±SE), BMI=25±1). Acetylcarnitine to creatine ratio (AC/Cr) and blood glucose (BG) were measured after an 8h fast and every 15-30min over 2h following ingestion of 50g of glucose. On a separate day, fasting AC/Cr was measured before and after 5min of strenuous rhythmic plantar flexion (PF) and plantar flexor muscles oxidative capacity was determined by Phosphorus MRS. Physical activity (PA) was measured by accelerometry for 7 days. Fasting AC/Cr ranged from 0.85 to 0.16 with an average of 0.39±0.08. Following glucose ingestion, BG significantly increased with a peak at 30min and returned to basal levels by 90min (p≤0.001). Meanwhile, AC/Cr declined over time between 0 and 45min (p=0.005); between 60 and 120min postprandial there was no detectable AC. Five minutes of PF increased fasting AC/Cr by 40% (p=0.029). Fasting AC/Cr was correlated with PA (r=0.733, p=0.010) and oxidative capacity (r=0.813, p=0.002). Initial postprandial BG increases were consistent with decreases in muscle AC content. However, the return of BG to basal levels was not concomitant to AC formation. These results suggest that muscle AC accumulation with fasting is associated with systemic glucose availability and insulin-stimulated glucose uptake. The exact underlying mechanisms are to be determined and might involve glycogen synthesis and pyruvate dehydrogenase activity. Support:NIH/DK095210
DISTINCT SUBPOPULATIONS OF NEUROTENSIN NEURONS IN THE LATERAL HYPOTHALAMIC AREA CONTRIBUTE TO ENERGY BALANCE BY DISCRETE MECHANISMS AND PROJECTIONS

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Some neurons in the lateral hypothalamic area (LHA) contain the neuropeptide neurtensin (Nts) and regulate feeding, drinking and physical activity. Indeed, many LHA Nts neurons contain the inhibitory neurotransmitter GABA, but some express the excitatory neurotransmitter glutamate (glut), supporting the possibility of different subtypes of LHA Nts neurons. The observation of LHA Nts neuronal terminals within both the Ventral Tegmental Area (VTA) and Substantia Nigra Compacta (SNC) suggests that subpopulations of LHA Nts neurons have distinct midbrain projection targets and mechanisms of action. Furthermore, some LHA Nts neurons co-express the long form of the leptin receptor (LepRb) and are activated by the anorectic hormone leptin (NtsLepRb neurons) while others are activated by dehydration (NtsDehy neurons). I examined the hypothesis that NtsLepRb and NtsDehy neurons are distinguishable from each other based on their expression of GABA or glut and whether they project preferentially to the VTA or SNC. Previously the lack of reagents to simultaneously identify Nts, GABA and glutamate prevented this analysis. To overcome this, we designed a dual genetic recombinase approach to simultaneously label Nts and GABA or glutamate-containing neurons. First, we generated mice that express FlpO recombinase specifically in Nts neurons (NtsFlpO mice) which we crossed with mice that express Cre recombinase in GABA or glutamate neurons. The resulting mice expressed FlpO recombinase in Nts neurons and Cre in GABA/Glutamate Neurons; NtsFlpO/vGatCre or NtsFlpO/vGlutCre mice respectively. Injecting these mice in the LHA with vectors for FlpO-inducible green fluorescent protein (GFP) and Cre -inducible red fluorescent protein (RFP) causes GFP expression only within LHA Nts neurons and RFP expression only in GABA/Glut neurons enabling the simultaneous detection of Nts and GABA and/or glutamate containing neurons via fluorescent microscopy. To examine the midbrain projections of subtypes of LHA Nts neurons, Fluoroagold (FG) retrograde tract tracer was injected into the VTA or SNC of mice that express GFP only in Nts neurons (NtsGFP mice) and followed with leptin, or dehydration treatment. Brains were analyzed via immunostaining to label NtsLepRb, or NtsDehy neurons and FG.

REPROGRAMMING OF SOMATIC CELLS INTO INDUCED ROD PHOTORECEPTORS

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Age-related macular degeneration (AMD) is a progressive disease in which patients experience deterioration of their visual acuity, which leads to irreversible blindness. There are two forms of AMD. Exudative AMD is also known as wet AMD and the non-exudative AMD is also called atrophic and dry AMD. The primary cause of AMD is retinal atrophy due to the death of retinal pigment epithelial (RPE) cells and their overlying photoreceptors (PRs): rods and cones. As greater portions of the retina atrophies, scar tissue forms and vision deteriorates. To reverse this process, we attempted to convert the fibroblasts that make the scar tissue into rod PR cells. Our hypothesis is that mature and functional rod PRs are derivable in vitro by directly reprogramming fibroblasts via forcing the expression of transcription factors (TFs) necessary for PR development. Our methodology consists of overexpressing TFs in fibroblasts obtained from Nrl-GFP positive transgenic mice and using immunocytchemistry for the analysis of NEUROD1 and CHX10 expression. We tested thirty-two combinations formulated by mixing of the following TFs: OTX2, CRX, NR2E3, NRL, and NEUROD1. Our results showed different rates in NRL-GFP-, CHX10-, and NEUROD1-positive cells among the 32 diverse groups of TF combinations. The highest rates of NRL-GFP-, CHX10-, and NEUROD1-positive cells were in the groups where OTX2 and CRX were overexpressed alone and together. This result suggests that OTX2 and CRX may have multiple regulatory roles in the expression of genes related to early PR development and might be necessary components in the reprogramming process of fibroblasts into rod PRs. Although NEUROD1 contributes to several differentiation pathways, including early retinal ganglion cell formation, we found that when overexpressed alone and in different combinations with the other TFs, the expression of NRL and CHX10 decreased, indicating a less efficient transformation. To improve the reprogramming efficiency, we will investigate the effect of RAX and PAX6 overexpression in combination with OTX2 and CRX. Furthermore, we will recreate the conditions the retinal environment, in vitro, by co-culturing the fibroblasts with RPE cells derived from embryonic stem cells. This environment will be used in combination with the overexpression of the above-identified TFs to determine if the reprogramming of induced rod cells can be enhanced. Our data shows that many LHA Nts neurons contain GABA and a come express glutamate. Additionally, both LHA NtsLepRb and NtsDehy neurons project to the VTA but LHA NtsDehy neurons also project to the SNC. Leptin and Dehydration treatment will used to determine the neurotransmitter content of these cue-distinct subpopulations too. (1F31 DK107081-01A1, 5 T32 ES007255-27) and GML (RC105025).
THE EFFECTS OF FIVE CLINICALLY RELEVANT NARCOTIC DRUGS ON THE GENERATION OF SPONTANEOUS AUGMENTED (SIGH) BREATHS
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The prototypical narcotic morphine powerfully suppresses protective respiratory reflexes such as spontaneous augmented breaths (ABs). Based upon the heterogeneity of pharmacological properties among commonly prescribed opioid medications, we hypothesized that the suppression of ABs would be significantly different in the presence of opioid drugs other than morphine. Therefore, we evaluated the effects of 5 common narcotics on the rate of ABs during spontaneous breathing using 6 unanesthetized adult male rats (534±47g, range=402-637g). Drugs were administered at the following dosage ranges: Morphine: 0, 1.0, 2.0, 3.0 mg/kg; Fentanyl: 0, 0.02, 0.03, 0.04 mg/kg; Methadone: 0, 1.0, 2.5, 4.0 mg/kg; Buprenorphine: 0, 0.01, 0.03, 0.05 mg/kg; Oxycodone: 0, 0.5, 1.0, 1.5 mg/kg (0 mg/kg = saline sham). The study followed a 2-factor repeated-measures crossover design, necessitating a total of 120 randomized monitoring sessions. At least 48 hours separated these within-animal plethysmographic monitoring sessions. Control injections did not affect the number of ABs, ranging from 6.5±1.4 to 7.3±1.6 ABs/15mins, p=0.912. However, each narcotic significantly depressed the number of spontaneous ABs in a dose-dependent manner, with the number of ABs observed at the highest drug dosages being: 1.5±1.3 (mor), 1.3±1.0 (fen), 0.8±1.2 (met), 0.0 ± 0.0 (bup), and 1.7±1.2 (oxy) ABs/15mins (p<0.001 for all changes vs sham). The strongest relationship between analgesic effect and suppression of ABs existed within the buprenorphine condition (r=−.764, p<0.001), which also yielded the lowest analgesic effect required to produce a 50% suppression in ABs (observed at 16% prolongation in tail flick latency). We conclude that the suppression of ABs is a ubiquitous side effect of common narcotics across the low-moderate analgesic range, and is unlikely to be mitigated by selective avoidance of specific opioids/opiates. Further, the unique potency of buprenorphine in suppressing ABs suggests that complex opioid receptor interactions may be of key mechanistic importance in mediating this effect.

THE NEUROKININ-2 RECEPTOR ANTAGONIST GR 159897 PROTECTS AGAINST NEUROINFLAMMATION IN THE MOUSE ENTERIC NERVOUS SYSTEM DURING COLITIS
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Irritable bowel syndrome (IBS) is a functional gastrointestinal (GI) disorder affecting 20% of Americans. Motility changes in IBS are driven by changes in the enteric nervous system (ENS). IBS has no cure, but recently, a neurokinin-2 receptor (NK2R) antagonist has shown to improve overall symptoms in a clinical study. However, the effects of NK2R antagonists on the ENS are not understood. We hypothesize that the beneficial effects of this antagonist on IBS are, in part, due to effects on the ENS. We tested this by treating mice with the NK2R antagonist GR159897 in a dinitrobenzene sulfonic acid (DNBS) model of colitis. To assess changes, we used immunohistochemistry to quantify neuronal survival and performed a glial morphology analysis. Calcium imaging recordings were performed in tissue from mice expressing the genetically-encoded calcium indicator GCaMP5g expressed under the control of the SOX10 promoter (SOX10::creERT2/+; PC::G5-TdT+/-). Our results show that treatment with GR159897 prevents increases in glial fibrillary acid protein (GFAP) immunoreactivity (n= 3-4 mice; p= 0.0141), increases in glial process length (n= 9-12 glia; p= 0.013) and neurodegeneration (n = 3-4; p= 0.0028). Immunohistochemical and calcium imaging data show that enteric glia express NK2Rs that are activated by neurokinin-A (NKA). Glial responses to NKA were decreased in the presence of GR 159897 (n= 101-113 glia; p= 0.0001) or tetrodotoxin and were significantly reduced in samples from mice lacking glial connexin-43 hemichannels (SOX10::creERT2/+; Cx43f/f mice; n= 50-113 glia; p= 0.0001). Glial responses to NKA were completely abolished in tissue from SOX10::creERT2/+; Cx43f/f mice in the presence of GR 159897 (n= 34-113; p= 0.0001). In conclusion, our data demonstrate that GR159897 provides neuroprotection during colitis through mechanisms that involve decreasing glial activity driven by NKA. We speculate that these mechanisms could explain some of the clinical benefits of current NK2R antagonists in IBS.
OREXIN A CAN INCREASE PROINFLAMMATORY CYTOKINE EXPRESSION IN PC12-OX1 CELLS

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Previous studies have demonstrated that orexin receptor activation resulted in an increase in sympathetic activity and elevation in intracellular calcium levels. This observation coupled with the important role of calcium on the control of Ca2+/calmodulin-dependent protein kinase II (CamK2) activity have led us to hypothesize that orexin system may involve in increasing sympathetic nerve activity through stimulating the expression of CamK2. In this study, we test this hypothesis using the neuron-like PC12 cell which artificially expressing human orexin A receptor 1 (OX1R). Consistent with the previous finding, incubation of human orexin A (100 nM) dramatically increased the cytosolic calcium level measured with a fluorescent calcium probe (Fluo-4AM) in PC12-OX1R cells. In addition, orexin A treatment (100nM) for 6 hours resulted in significant increases in the mRNA levels of calcium dependent protein early growth response 1 (EGR1, 25-fold, P<0.05), CamK2a(2-fold, P<0.05), Camk2b (4-fold, P<0.05) as well as Fosl1 (9-fold, P<0.05), a chronic neuronal activation marker, compared to control group. These data suggest that binding of orexin A and OX1R increases calcium influx which may subsequently stimulate EGR1 expression, the EGR1, as a major transcription regulator, may modulate expression of many genes including the expression of CamK2, which eventually increase neural firing. Our further study will investigate whether or not blockage of CamK2 will attenuate the increase in sympathetic nerve activity.

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ENTERIC GLIAL CELLS ACUTELY REGULATE SECRETOMOTOR FUNCTION IN THE MOUSE COLON

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Enteric glial cells are implicated in the regulation of epithelial barrier and secretomotor functions of the intestines. But whether glial cell activity regulates these functions acutely under physiological conditions is not clear. We addressed this issue by using transgenic animal models to modify the activity of enteric glia, either reducing glial expression of connexin 43 in Sox10::CreER<sup>T2+/-</sup>/Cx43<sup>f/f</sup> mice or activating glial calcium responses in GFAP::hM3Dq mice, and tested the effects on colonic barrier function and electrogenic ion transport in Ussing chambers. We assess neuronal dependent and independent contributions by activating or inhibiting neurogenic activity with veratridine and tetrodotoxin, respectively. Our results show that the reduction of glial Cx43 expression in Sox10::CreER<sup>T2+/-</sup>/Cx43<sup>f/f</sup> mice significantly reduced neurogenic ion transport to 75±5% (mean±SEM) of the paired littermate controls (P=0.004, One-sample t-test, n=6 animals per group). The selective glial activation in tissues from GFAP::hM3Dq mice evoked electrogenic ion transport to an equal extent as the direct activation of neurogenic ion transport with veratridine (74±9 vs. 75±2% of the responses to secretagogue forskolin) and glial driven responses consisted of both tetrodotoxin sensitive and insensitive components. The glia-selective stimulation did not affect transmural ion conductance or cell-impermeant dye flux but the baseline ion conductance was more variable in Sox10::CreER<sup>T2+/-</sup>/Cx43<sup>f/f</sup> tissues. Together, our findings show that glial activity contributes to the regulation of electrogenic ion transport in the intestine through effects on neurons and possibly direct effects on epithelial cells. However, the influence of glia on gut barrier function seems to involve tonic, rather than acute, mechanisms. These findings provide novel insight into the cellular mechanisms that control fluid transport homeostasis in the intestine. Additionally, these observations raise the possibility that enteric glial activation plays a role in functional diarrheal diseases and that the selective manipulation of glia could improve patient quality of life.
Enteric glia, a unique class of peripheral neuroglia, are integral in the maintenance of enteric neuronal circuits and normal gastrointestinal (GI) function. *In vitro* studies have identified the antioxidant reduced glutathione (GSH) as a vital neuroprotective factor secreted by glia (Abdo et al 2010). However, little is known about the cellular mechanisms regulating GSH production and the role GSH plays during *in vivo* inflammation. Here, we identified the cellular components involved in GSH synthesis and investigated the effects of *in vivo* inhibition of GSH synthesis during GI physiology and pathophysiology. We used immunohistochemistry to localize GSH synthesis proteins and to quantify changes in neuronal density and other cellular markers. We inhibited GSH synthesis *in vivo* and *in vitro* with the selective inhibitor Buthionine Sulfoximine (BSO) and induced *in vivo* inflammation using the 2,4-dinitro benzene sulfonic acid (DNBS) model of colitis. All experiments used male C57Bl/6 mice, aged 6-8 weeks. Data were analyzed using a Student’s t-test or one/two-way ANOVA as appropriate. Glutathione is synthesized in a two-step process. The first and rate-limiting enzyme glutamate-cysteine ligase (GCL) is expressed in enteric glia and neuronal varicosities. The second enzyme, glutathione synthetase, is similarly found in both neuronal and glial cell bodies. Immunoreactivity for both enzymes is upregulated in response to *in vivo* inflammation (p < 0.005). Inhibition of GSH production with the GCL-inhibitor BSO is sufficient to drive neurodegeneration both *in vivo* and *in vitro* (p = 0.053 and p < 0.01 respectively). Mice drinking water supplemented with BSO were protected from colonic shortening (p = 0.054), inflammatory cell infiltration (p < 0.001) and neurodegeneration during colitis. Our work highlights a novel role for the endogenous antioxidant GSH and shows that GSH production by enteric glia is an important neuroprotective mechanism during GI (patho)physiology.
EFFECT OF EARLY PEDIATRIC DISABILITY EXPOSURE IN MEDICAL EDUCATION
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Introduction: Currently, there exists a deficit in quality of care for children with developmental disabilities (DD), calling for improved physician training. The Arie home-visit program provides first and second year medical students the opportunity to interact with children with DD and their families to gain insight into their home life, medical needs, and personal concerns. Ultimately, this will translate into greater student empathy and understanding of the challenges involved in the treatment of DD.

Description: Groups of two to four students made home-visits to families of children with DD. Students were assessed before and after participation using the Medical Students’ Perceptions of Disability and Definitions and Criteria Associated with Disabilities Assessments and the Jefferson Scale of Physician Empathy adapted for Health Profession Students. Families completed a survey of physician trust and were asked questions from a structured interview. Some students were assigned to the program while others volunteered.

Impact: Family interviews demonstrated a perceived shortcoming in care for patients with DD. While 48% were pleased with their physician’s behavior, 23% called for improvement in quality care, and 39% identified physician perception as an obstacle to care. When asked for suggestions for improvement, families were most concerned with physician character (58%) and knowledge of DD (32%). Students’ pre-survey results showed that assigned students had a significantly lower perception of patients with DD than volunteer students. Post-test results show both groups demonstrated significant improvement in student confidence, compassionate care, and comfort working with children with DD (p<0.05).

Discussion: Exposing medical students to patients with DD had a positive impact on their perception of and comfort around these individuals; improved understanding of the challenges related to raising children with DD should translate to better care. These results could guide the implementation of similar programs across the country.

FOSTERING INTEREST AND UNDERSTANDING OF NEUROPHYSIOLOGICAL CONCEPTS THROUGH THE USE OF INTERACTIVE DEMONSTRATIONS
Neha S Chava, and Soumya S Kulkarni.
Northville Neuroscience Club, Northville High School, Northville, MI, 48167.

Finding ways to communicate complex ideas to people of all ages is an important hurdle to overcome in making neurophysiological concepts more understandable. Northville High School’s Neuroscience Club aims to make neuroscience an interesting and approachable topic for children, adolescents, and adults. Neurophysiology is difficult to explain to students in a typical educational setting due to the difficulty in visualizing concepts such as neural pathways and action potentials. The Neuroscience Club used a simple demonstration to explain these complex concepts to children; the demonstration was an exhibit at the Michigan Science Center’s Brain Day, which featured neural activity in large Madagascar cockroaches. The club used Backyard Brain’s affordable SpikerBox™, which works by hooking up a detached hind leg of a cockroach to a machine that connects electrodes from a battery to muscles in the leg. As voltage passes through the electrodes, two events can occur: neurons can be stimulated to fire causing the leg to move or the muscle can contract independent of innervation. This was extrapolated to using music of different frequencies to control the movement of the leg. Children visiting the station discovered the anesthesia process through detachment of a cockroach leg, learned cockroach anatomy, listened to neuronal firings, and viewed the effect of varying the neuronal firings on the cockroach leg’s movement. The interactive demonstration with cockroaches fostered interest and curiosity in children, enabling connection of the complicated topics of neural pathways and cockroach physiology to the easy-to-digest concepts of movement and music. Volunteers from Neuroscience Club asked children questions to expand thinking on the concept such as “How does your brain tell your arm to move?” Not only was this exhibit informative and exciting for children and parents who visited, but it was also cost-effective (<$100) and incited long-term passion for neurophysiological concepts.
Teamwork is a professional skill that educators encourage students to cultivate throughout their college years. However, as institutions we often falter in demonstrating how to properly work together as a team and how valuable shared time and resources can be in accomplishing a common goal. At Michigan State University (MSU), we utilize our Physiology Understanding (PhUn) week outreach to build rapport not only in the community surrounding our school but also within the academic community found on campus. This practice was demonstrated in our 2016 PhUn Day, where our departmental outreach coordinators were assisted by research and teaching faculty and staff in the Department of Physiology along with more than 4 other departments, 5 student organizations, the Medical School, the Center for Service Learning and Civic Engagement, the MSU radio and newspaper, and both undergraduate and graduate students across numerous majors. Thus, by implementing the element of institution-wide teamwork to our event, we are able to model and inspire dynamic professional skill building to our students, be inclusive of all individuals on campus by inviting them to partake in outreach activities in any number of facets and help MSU achieve its mission of making a positive difference, both locally and globally, through educational tools.

KEYNOTE LECTURE FOR EDUCATIONAL SYMPOSIUM
THE TWO HOURS MARATHON: WHAT DO STUDENTS THINK?
Steven Elmer, Ian Greenlund, Michael Joyner, Jason Carter
Michigan Technological University, Department of Kinesiology and Integrative Physiology

For over 100 years, athletes, coaches, and scientists have endeavored to improve running performance. The marathon world record is 2:02:57, requiring a 2.4% improvement to achieve a sub-two hour marathon. Joyner (1991) used the main determinants of running performance (VO2max, lactate threshold, running economy) to model that a runner could theoretically run a 1:57:58 marathon. Twenty years later, Joyner and colleagues (2011) revisited this concept and asked the question - The two hour marathon: who and when? This question sparked enthusiasm as physiologists from around the world provided commentary, and industry lead initiatives set a new best marathon time (2:00:25). Given the widespread interest and uncertainty surrounding the two hour marathon, we used this as an opportunity to engage our students in the discussion, and challenged them to connect physiology to current events. Using problem- and project-based learning methods, exercise physiology students (n=48) explored what it would take to achieve the first sub-two hour marathon. Foundational content was off-loaded to video lectures to allow more class time for active learning. Undergraduate students completed a multi-day unit in which they: discussed the Joyner et al. (2011) and follow-up commentary, collaborated in groups to propose additional factors that would impact marathon performance, and presented their ideas to the class. Graduate students expanded upon this through a multi-week unit in which they discussed additional review papers, determined their own running economy, and presented their ideas at a department seminar. Students identified factors ranging from genetics, running economy, drafting, surface material, training volume/intensity, and psychology. Finally, students presented their ideas to Dr. Joyner through Skype and learned about the physiology and history of running. This two hour marathon activity required students to ask and refine questions, debate, make predictions, conduct experiments, analyze data, and communicate their findings to peers and experts in the field.
Poster #1
A novel role for NHERF1 in regulating human T cell responses
David Broadbent, Ananth Kumar Kammala, Rupali Das and Hariharan Subramanian
Department of Physiology, Michigan State University, East Lansing, MI-48824

Poster #3
Molecular gut contents of water mites from Belle Isle’s Blue Heron Lagoon, Detroit, Michigan
Adrian A. Vasquez, Tiffany Burris, Milad Qazazi, Jeffrey L. Ram
Department of Physiology, Wayne State University School of Medicine

Poster #5
Association Between Body Composition, Muscle architecture, Neuromuscular Function and Task Performance in Middle-Aged Women
Phillips, Kevin; Gage, Matt; Noh, Byungjoo; Yoon, Tejin
Michigan Technological University

Poster #7
Opposing roles of CD2 and 2B4 in iNKT cell cytotoxic responses
Hyun Hee Lee, Trevor G. Gohl, Ryan M. Mack, Rupali Das
Department of Physiology, Michigan State University

Poster #9
Decreased aldehyde dehydrogenase (ALDH)2 activity contributes to coronary endothelial dysfunction in diabetic cardiomyopathy
Gudong Pan1, Mandar Deshpande2, and Suresh S. Palaniyanand1
1Division of Hypertension and Vascular Research, Department of Internal Medicine, Henry Ford Health System, Detroit, MI 48202; 2Department of Physiology, Wayne State University, Detroit, MI, 48202

Poster #11
Identification of a novel immune checkpoint for iNKT cell anti-cancer response
Ryan M. Mack, Hyun Hee Lee, Trevor G. Gohl, Rupali Das
Department of Physiology, Michigan State University

Poster #13
The immune adaptor ADAP plays a pivotal role invariant natural killer T cell functions.
Trevor G. Gohl, Hyun H. Lee, Sami J. Abdelaziz, Ryan M. Mack, Rupali Das
Department of Physiology, Michigan State University

Poster #15
Acetate, the Metabolite of Ethanol, Increases Cytosolic Calcium and mRNA Expression Levels of EGR1 and TNFβ± in Dopaminergic Like PC12 Cells
Behnke, Jessica; Chapp, Andrew; Driscoll, Kyle; Shan, Zhiying; Chen, Qinghui
Michigan Technological University

Poster #17
The potential role of the adaptor protein SAP in CD2 signaling pathway
Shashank Chitta, Hyun Hee Lee, Rupali Das
Michigan State University
Poster #19
Effects of Glucocorticoids on Mucins Levels in the Small and Large Intestine
Shinouskis, Allison; Schepper, Jonathan; McCabe, Laura
Michigan State University

Poster #21
Tai Chi Intervention for Teenagers with Autism Spectrum Disorder
Dai, Yi Ling; Rohrbeck, Kristin; Ferrari, Thomas
Oakland University William Beaumont

Poster #23
Effects of Metabolic Remodeling on Mechanical Function in Heart Failure
Lopez, Rachel; Van den Bergh, Francoise; Gao, Xin; Michele, Daniel; Beard, Daniel
University of Michigan

Poster #25
In Vivo Characterization of Muscle Hemodynamic Recovery in Electrically–stimulated Rat Hindlimb Using Magnetic Resonance Imaging
Tonson, Anne; Kasper, Jonathan; Tanbakuchi, Daniel; Meyer, Ronald; Wiseman, Robert
Michigan State University - Physiology Department

Poster #27
A study of gender influence on the pattern of cardiorespiratory response to transient and unrelenting acute severe hypoxemia in adult rats
Van Maele, Nicholas; Hosner, Conner; Bell, Harold
Central Michigan University College of Medicine

Poster #29
THE PSYCHOPHYSIOLOGY OF SELF-FORGIVENESS
Eaton, Kyle; Ferrari, Thomas
Oakland University William Beaumont

Poster #31
Mechanisms for posterior cerebral artery remodeling during Angiotensin II-induced hypertension
Jessica Yen, Janice M. Diaz-Otero, Courtney Fisher, Anne M Dorrance
Department of Pharmacology and Toxicology, Michigan State University

Poster #33
The Development of a Co-Culturing System to Study the Phenomenon of Cancer-Associated Thrombosis
Dustin G. DeGrave[1], Nicholas A. Shortreed[1], Melanie M. Flaherty[1][2], Johnathan E. Lawrence, Ph.D.[1], Jose A. Diaz, M.D.[2]
Northern Michigan University

Poster #35
Early Metabolic Adaptation in the ZDF Rat Model of Type 2 Diabetes
Lewis, Matthew; Tonson, Anne; Kasper, Jonathan; Bazil, Jason; Meyer, Ronald; Wiseman, Robert
Michigan State University
Poster #37
The effect of Hibiscus sabdariffa tea on cardiovascular risk factors in prehypertensive adults

Greenlund, Ian; Wakeham, Travis; Nelson, Emily; Revoyr, Mikayla; Durocher, John
Michigan Technological University

Poster #39
Enhancing the anti-leukemia functions of invariant natural killer T cells using a CD1d-anti-CD19 scFV fusion protein

Gohl T., Guan, P., Mack R., Evans E., Zauderer M, Nichols K., Das, R.
Michigan State University

Poster #41
Hypoxia-regulated expression of GLUT1 in GBM Cell Lines

Kane, Marissa
Northern Michigan University

Poster #43
JAK inhibitors in allergy: a promising alternative for the treatment of severe asthma.

Bansal, P., Kammala, A.K., Subramanian, H., Das, R.
Physiology Department, Michigan State University, Michigan

Poster #45
Store operated Ca2+ mechanisms contribute to mast cell activation via MrgprX2

Occhiuto, Christopher; Subramanian, Hariharan
Michigan State University

Poster #47
Exercise-trained and long-lived Drosophila activate similar genetic programs

Maryam Safdar, Alyson Sujkowski, Robert Arking, Robert Wessells
Wayne State University

Poster #49
Assessment of cardiac function during the progression of heart failure induced by intracoronary embolizations in the canine model: monitoring in conscious and anesthetized states.

Krishnan, Abhina; Lovelace, Abe; Senador, Danielle; Kaur, Jasdeep; Rasaih, Megan; Alvarez, Alberto; Hanna, Hanna; Levanovich, Peter; Aung, Kimberly; Dombrowksi, Mary; Haddad, Samuel; Mannozzi, Joseph; Sala-Mercado, Javier; Levy, Phillip; O'Leary, Donal
Wayne State University

Poster # 51
Beta Cell ER Homeostasis

Cesar Barrabi, Xuequn Chen
Wayne State University

Poster #53

Elmer, Steven; Bye, Thomas; Hudak, Kirsen; Gabe, Alex; Carter, Kathyrn
Michigan Tech University
Poster #55
Blueberries Protect Pancreatic Beta Cell Function
Liu, Weixang; Schoenborn, Jacob; Tang, Xiaoqing
Michigan Technological University

Poster #57
Using Anisotropic Ultrasonic Backscatter to Determine Cardiac Fiber Maps in Small Animals
Alkhazal, Thamer; Milne, Michelle; Chung, Charles
Wayne State University

Poster #59
The Two Hour Marathon: What do Students Think?
Steven Elmer, Ian Greenlund, Michael Joyner, Jason Carter
Michigan Technological University, Department of Kinesiology and Integrative Physiology

Poster #63
Use of a Course-based Service Learning Assignment to Increase Understanding of Physiology in Local Schools
Bye, Thomas; Carter, Kathryn; Elmer, Steven; Carter, Jason
Michigan Tech University

Poster #65
Berberine hydrochloride improves insulin resistance induced by cytokines in C2C12 myoblast cells
Anil Poudel, DVM., PhD and Lixin Li, MD., PhD
Physician Assistant Program, College of Health Professions, Central Michigan University, Mt. Pleasant, Michigan 48859
**POSTER SESSION B - Friday, June 9th 1:30-2:30 pm**

**Poster #2**  
A novel role for the adapter molecule NHERF1 in regulating mast cell mediated anaphylactic response  
*Ananth Kumar Kammala, Hariharan Subramanian*  
Department of Physiology, Michigan State University, East Lansing, MI

**Poster #4**  
Differential mast cell response between Th1 (B6) and Th2 (Balb/c) mice is regulated by G-Protein Coupled Receptor Kinase 2  
*Canchai Yang, Christopher Occhuito and Hariharan Subramanian*  
Department of Physiology, Michigan State University, East Lansing, MI-48824

**Poster #6**  
G protein receptor kinase 5 and 6 contribute to Mas-related gene receptor X2-induced responses in human mast cells  
*Natasha Dighe* and Hariharan Subramanian  
Department of Physiology, Michigan State University, East Lansing, MI

**Poster #8**  
Gender Differences Involving Serotonin (5-HT) Receptors in Type I Diabetic Rats  
*Moldovan, Tudor; Cervantes, Marisa; Bucan, Jonathan; LePage, Bernard; Walker, Zachary; Bhaskaran, Subha; Banes-Berceli, Amy*  
Oakland University

**Poster #10**  
Investigating The Phenomenon of Cancer Associated Thrombosis in vitro Utilizing A Conditioned Media Model  
*Shortreed, Nicholas; Flaherty, Melanie; DeGrave, Dustin; Lawrence, Johnathan; Diaz, Jose*  
Northern Michigan University

**Poster #12**  
Activation of FceRI modulates GRK2-6 expression in mast cells  
*Mohammad Mustafa Ahmadzai, Canchai Yang, Christopher Occhiuto, Rupali Das, Hariharan Subramanian*  
Department of Physiology, Michigan State University, East Lansing, MI

**Poster #14**  
Novel therapeutic intervention to modulate allergen-induced iNKT cell-mediated immune response in asthma  
*Sami J. Abdelaziz, Trevor G. Gohl, Christopher Occhiuto, Ryan M. Mack, Hariharan Subramanian, Rupali Das*  
Michigan State University

**Poster #16**  
High Salt Diet Plus Fructose Water Intake Induces Hypertension.  
*Taija M. Hahka, Yuanyuan Fan, Enshe Jiang, Qinghui Chen, Zhiying Shan.*  
Kinesiology & Integrative Physiology, Michigan Tech. Univ., Houghton, Mi. 49931.

**Poster #18**  
3-D Printing Open Source Click-MUAC Bands for Identification of Malnutrition  
*Ross E. Michaels & Joshua M. Pearce*  
Michigan Technological University
Poster #20
Effect of Sedentary Conditions on the Expression of GABAAα2 Receptor Subunits in Bulbospinal C1 and non-C1 Neurons along the Rostrocaudal Extent of Rat Rostral Ventrolateral Medulla (RVLM)
Bozena E Fyk-Kolodziej1, Toni A Azar1, Ida J Llewellyn-Smith1,2, and Patrick J Mueller1
1Dept. of Physiology, Wayne State Univ. School of Med., Detroit, MI, 48201 and 2Centre for Neuroscience, Flinders University, Adelaide, Australia

Poster #22
Androgen receptor expression in chemically-defined hypothalamic nuclei associated with energy balance and reproduction
Cara, Alexandra; Elias, Carol
University of Michigan

Poster #24
Pyruvate Dehydrogenase Activation follows the Cytosolic ATPase Activity during Contraction in Skeletal Muscle with High and Low Mitochondrial Density
Kasper, Jonathan Lewis, Matthew Gudziak, Greg Meyer, Ronald Wiseman, Robert
Michigan State University

Poster #26
Reliability of heart rate variability as an assessment of cardiac sympathetic activity in humans
Wakeham, Travis; Fonkoue, Ida; Durocher, John; Cooke, William; Carter, Jason
Michigan Technological University

Poster #28
The Characterization Of LN229/mKate Glioblastoma Multiforme Cells Cultured in vitro Utilizing Artificial 3D Chitosan-Alginate Scaffolds
Shortreed, Nicholas; Lawrence, Johnathan; Belton, Robert; Winn, Robert
Northern Michigan University

Poster #30
MILD INTERMITTENT HYPOXIA WITH SUSTAINED HYPERCAPNIA IMPROVES AIRFLOW, REDUCES THERAPEUTIC CPAP AND BLOOD PRESSURE IN PARTICIPANTS WITH OBSTRUCTIVE SLEEP APNEA.
Mohamad El-Chami1,2, Sukhesh Sudan1,2, Trevor McCreary1,2, Ho-Sheng Lin1,2,3, and Jason H. Mateika1,2
Wayne State University School of Medicine, Detroit, MI.; Research and Development, John D. Dingell VA Medical Center, Detroit, MI; Department of Otolaryngology, Wayne State University School of Medicine, Detroit, MI.

Poster #32
Imaging renin granule exocytosis in juxtaglomerular cells by Total internal reflection (TIRF) microscopy

Poster #34
Psychophysiological Effects of Acute Mindfulness Meditation
Marti, Hannah; Wakeham, Travis; Morin, Brigitte; Durocher, John
Michigan Technological University

Poster #36
Mathematical Modeling of Tissue Bioenergetics in Streptozotocin-Induced Type 1 Diabetic Rat Muscle
Lewis, Matthew; Bazil, Jason; Meyer, Ronald; Wiseman, Robert
Michigan State University
Poster #38
Interferon gamma increases lipolysis and fatty acid oxidation in pancreatic β cells.
*Nguyen Truong, Jason Bazil, L. Karl Olson*
Department of Physiology, Michigan State University, Michigan 48823, USA

Poster #40
Cardiac myosin binding protein C mutants interact with Hsp70-family chaperones and disrupt protein quality control
*Glazier, Amelia; Helms, Adam; Hafeez, Neha; Kotlo, Srisha; Yob, Jaime; Tang, Vi; Day, Sharlene*
University of Michigan

Poster #42
Is Donated Kidney A Source of Hypertension in Kidney Transplant Recipients?
*Tantisattamo, Ekamol* and Mopuru, Haritha
Oakland University William Beaumont School of Medicine

Poster #44
The effects of social shuffling during puberty on pubertal cytogenesis and behaviors in adulthood.
*Wilks, Kristian*
Michigan State University

Poster #46
Aging enhances atrial fibrillation inducibility in atherosclerotic hosts
*Daniel J. Tyrrell,* Roberto Ramos Mondragón, Guadalupe Guerrero-Serna, Héctor H. Valdivia, José Jalife, and Daniel R. Goldstein
University of Michigan

Poster #48
Initiating an Exercise Physiology-Themed PhUn Week Presentation for 4th graders
*Conner J. Steffke,* & Naveen Sharma
School of Health Sciences, Central Michigan University, Mount Pleasant, MI, 48859

Poster #50
Titin-dependent Auxotonic Cardiac Relaxation
*Schick, Brianna; Chung, Charles*
Wayne State University

Poster #52
Gender Differences in Auditory Response Time
*Sosnowski, Benjamin,* Speirs, Sue
Grosse Pointe North High School

Poster #54
Reflexes for A and B personality types
*Hedman, Casey; Haggerty, Erin*
Grosse Pointe North High School

Poster #56
Height Impacts Reflex Reaction Time
*Doherty, Melina; Rafael, Christina; Castronero, Carmen*
Grosse Pointe North High School
Poster #58
Music Impacts Amplitude, Not Reflex Time
*Badih, Aiyana; Sonaglia, Victoria; Lemanske, Elizabeth*
Grosse Pointe North High School

Poster #60
Dog Age and Auditory Response Time
*Dean, Alex; Yerramalli, Gowri; Fazekas, Nicole*
Grosse Pointe North High School

Poster #62
Distracted Driving Lengthens Reaction Time
*Levick, Steven; Leone, Francesca; Mourad, Rachel*
Grosse Pointe North High School

Poster #64
Characterization of the in vitro glycation of insulin, insulin receptor, and insulin-like growth factor-1
*Turkette, Thomas; Rhinesmith, Tyler; Root-Bernstein, Robert*
Michigan State University

Poster #66
Effects of Respiratory Muscle Fatigue on Upper-Body Exercise Tolerance
*Thomas Bye & Steven Elmer*
Michigan Technological University, Department of Kinesiology and Integrative Physiology
**Poster #1**

**A novel role for NHERF1 in regulating human T cell responses**

*David Broadbent, Ananth Kumar Kammala, Rupali Das and Hariharan Subramanian*

*Department of Physiology, Michigan State University, East Lansing, MI-48824*

Allergic asthma is estimated to affect 1 in 12 people in the US, with treatments costing up to $56 billion in medical costs, lost school, and work days. This phenotype is best described as a T helper 2 (Th2) cell driven response. Yet, despite increased understanding of asthma, treatment options remain limited. The G protein coupled receptor, C3aR plays a pivotal role in regulating allergic asthma in humans. C3aR has a class I PDZ binding domain in its cytoplasmic tail. Previous reports from our laboratory have demonstrated that the sodium anion exchanger regulatory factor 1 (NHERF1), a class I PDZ binding protein regulates C3aR responses in immune cells such as mast cells. In addition, our preliminary data suggests that NHERF1+/− mice have reduced pulmonary inflammation as compared to NHERF1+/+ mice in a house dust mite model of allergic asthma. These data indicate a possible role for NHERF1 in regulating the Th2-driven pathology in asthma. The objective of this study was thus to characterize the role of NHERF1 in T lymphocyte signaling and functions following activation via the T cell receptor (TCR). Our data suggests that NHERF1 is important regulator of TCR signaling in human T cells. Knockdown of NHERF1 in Jurkat T cells shows downregulation of key phosphorylation signals in proximal TCR signaling proteins, such as Zap and Lat. Canonical pathways such as the mitogen-activated protein kinase pathway are also affected. These data correlate with functional assays that demonstrate that depletion of NHERF1 attenuates cytokine release and T cell chemotaxis. Together, these data demonstrate a novel role of NHERF1 in T cell signaling and highlight its potential as a possible drug target for allergic asthma.

**Poster #2**

**A novel role for the adapter molecule NHERF1 in regulating mast cell mediated anaphylactic response**

*Ananth Kumar Kammala, Hariharan Subramanian*

*Department of Physiology, Michigan State University, East Lansing, MI*

Anaphylaxis is a rapidly developing, life threatening allergic reaction that is classically elicited by crosslinking of antigen-specific IgE bound to the high affinity IgE receptor FceRI on mast cells. Proteases released from FceRI-activated mast cells result in the accumulation of the anaphylatoxin, C3a that can further amplify the anaphylactic response. Mast cells express C3a receptor (C3aR), a G-protein coupled receptor (GPCR) for C3a. C3aR possess a class I PDZ binding motif on its cytoplasmic tail. Adapter proteins such as the Na+ /H+ exchanger regulatory factor 1 (NHERF1) have been implicated in regulating functions of certain GPCRs by binding to the class I PDZ motifs in these receptors. In our previous study we had demonstrated that although NHERF1 does not directly interact with the C3aR, it is required for C3a induced degranulation and chemokine production in human mast cells. The aim of this study was to test the in vivo relevance of these observations in clinically relevant murine models of passive systemic anaphylaxis (PSA) and passive cutaneous anaphylaxis (PCA). In agreement with our in vitro data, NHERF1 promotes PSA and PCA induced by IgE and antigen. Specifically, NHERF1+/− mice showed significant attenuation of hypothermia and a substantial reduction in inflammatory biomarkers such as histamine and prostaglandin D2 as compared to control NHERF1+/− mice. Furthermore, we observed a significant decrease in vascular permeability response in NHERF1+ /− mice following cutaneous sensitization with IgE and antigen. These results suggests that the NHERF1 plays an vital role in regulating mast cell -mediated anaphylaxis and that NHERF1 could be a potential target for anaphylaxis and allergic diseases.
Poster #3

Molecular gut contents of water mites from Belle Isle’s Blue Heron Lagoon, Detroit, Michigan

Adrian A. Vasquez, Tiffany Burris, Milad Qazazi, Jeffrey L. Ram
Department of Physiology, Wayne State University School of Medicine

Water mites are predatory arachnids that inhabit aquatic habitats and are found worldwide. Water mites are one of the most biologically diverse microinvertebrates but according to leading acarologists only about half have been properly described in North America. In this study we have looked at the populations of water mites found in Blue Heron Lagoon, a lagoon of Belle Isle, Detroit, MI that was recently connected to Lake St. Clair by an EPA-funded habitat restoration project. Our preliminary studies revealed the presence of at least 15 genera of water mites with potentially several species represented at each genus by our molecular COI barcode studies. Use of an 18S chironomid family-specific primer revealed that at least two genera were feeding on chironomids from as early as June to as late as October. Additionally, molecular gut content analysis revealed previously unknown evidence that water mite diets in the field includes the consumption of chironomids by Arrenurus sp. deuteronymphs which are not known to feed on Dipteran larvae as adults. These preliminary data have led to a greater understanding of the dietary habits of water mites. We are currently designing and testing other primers to establish baseline data that can be used for next generation sequencing of water mite molecular gut contents.

Poster #4

Differential mast cell response between Th1 (B6) and Th2 (Balb/c) mice is regulated by G-Protein Coupled Receptor Kinase 2

Canchai Yang, Christopher Occhitto and Hariharan Subramanian
Department of Physiology, Michigan State University, East Lansing, MI 48824

Allergic diseases such as asthma, rhinitis and food allergy affect millions of individuals and the prevalence of these diseases has been increasing worldwide. Individuals with a suppressive phenotype (Th2) are more prone to allergy as compared to those who are susceptible to a pro-inflammatory (Th1) response. Mast cells are tissue resident immune cells that play a critical role in mediating allergic responses. While it is known that mast cells cause allergic diseases, it is unknown if their responses are altered in Th2 susceptible individuals as compared to Th1 dominant humans. We hypothesized that Th2 skewed mast cells will respond more robustly as compared with Th1 type mast cells. Accordingly, our data suggested that mast cells from Balb/c (Th2) mice respond more robustly to allergen than mast cells from B6 (Th1) mice. We have previously demonstrated that G-Protein Coupled Receptor Kinase 2 (GRK2) enhances allergen responses in mast cells. To directly test whether the enhanced Balb/c mast response was attributable to GRK2 expression, we examined the levels of GRK2 in B6 and Balb/c mast cells following allergen stimulation. Preliminary analysis revealed that Balb/c mast cells demonstrated enhanced GRK2 levels compared to B6 mast cells. In conclusion, GRK2 expression levels may regulate mast cell responses and this might explain why Balb/c mast cells respond more than B6 mast cells.
**Poster #5**

*Association Between Body Composition, Muscle architecture, Neuromuscular Function and Task Performance in Middle-Aged Women*

**Phillips, Kevin; Gage, Matt; Noh, Byungjoo; Yoon, Tejin**

*Michigan Technological University*

Although excessive body fat has been viewed as having a negative effect on health and physical function, recent studies have shown positive adaptations related to muscle architecture and neuromuscular function. We examined the association between body fat and performance of functional tasks, muscle quantity and quality in middle-aged women. Sixteen middle-aged women (54 ± 5 years) participated in 2 sessions. Body fat percentage of each participant was recorded in both sessions and averaged (34 ± 6%, range: 15–40%), using a body composition analyzer. During the 1st session, *in vivo* muscle architecture measurements were made using a B-mode real-time ultrasound scanner. Muscle thickness and pennation angle was examined in the rectus femoris, vastus lateralis, gastrocnemius medalis, and gastrocnemius lateralis muscles. During the 2nd session, participants completed a six minute walk test and a sit to stand test. Next, participants performed maximal voluntary isometric contractions (MVIC) of knee extensor muscles on a dynamometer. Single electrical stimuli were applied over quadriceps muscles to elicit twitch torque during MVIC and upon relaxation (approximately 2 s) following the MVIC to assess voluntary activation [VA = (1-superimposed twitch/resting twitch) x 100]. Maximal torque and rate of torque development were calculated and normalized by weight. The associations between all variables were tested with Pearson product-moment correlation coefficient. The correlation between body fat percentage and normalized MVIC torque showed a significant negative relationship (r = -0.642, p = 0.007). Additionally, normalized rate of torque development trended towards a negative relationship (r = -0.486, p = 0.056). However, there was no significant association between body fat percentage and muscle architecture, voluntary activation, or task performance. Body fat percentage was not associated with performance of functional tasks or muscle architecture. However, body fat percentage was negatively associated with the quality of knee extensor muscles in middle-aged women.

**Poster #6**

*G protein receptor kinase 5 and 6 contribute to Mas-related gene receptor X2-induced responses in human mast cells*

**Natasha Dighe and Hariharan Subramanian**

*Department of Physiology, Michigan State University, East Lansing, MI*

Mast cells are important modulators of the innate and acquired immune response. They become activated during infection and release pre-formed granule-associated pro-inflammatory mediators via a process termed as “degranulation”. Mast cell activation occurs via either the high affinity IgE receptor, FceRI or via G protein-coupled receptors (GPCRs) expressed on the cell surface. Interaction of GPCR with its cognate ligand often results in activation of downstream signaling pathways that regulate mast cell responses. GPCR kinases (GRK2, 3, 5 and 6) negatively regulate signaling by phosphorylating the GPCR which leads to densensitization and internalization of the receptor. We recently identified a novel GPCR Mas-related gene-X2 (MrgprX2) on human mast cells and demonstrated that several host defense peptides promote mast cell activation via MrgprX2. Furthermore, GRK2 and GRK3 do not regulate MrgprX2; however it is unclear if MrgprX2 induced mast cell response is dependent on other GRKs such as GRK5 and GRK6. Our preliminary data suggests that GRK5 and GRK6 play a vital role in MrgX2-induced human mast cell responses. Specifically we observed reduced mast cell degranulation response in the absence of GRK5 or GRK6. Investigation of the role of GRK5 and GRK6 will therefore provide further insights into how mast cells play a protective role in innate immunity against pathogens.
**Poster #7**  
**Opposing roles of CD2 and 2B4 in iNKT cell cytotoxic responses**  
*Hyun Hee Lee, Trevor G. Gohl, Ryan M. Mack, Rupali Das*  
Department of Physiology, Michigan State University

Invariant natural killer T (iNKT) cells comprise a unique lineage of innate-type lipid-reactive T lymphocytes with important roles in host immunity, including defense against specific pathogens and cancers. Although it is well appreciated that iNKT cells rapidly produce cytokines and mount potent cytotoxic responses following T cell receptor (TCR) engagement, the mechanisms that control iNKT cell functions remain poorly understood. This gap in current knowledge restricts the development of effective iNKT cell-based therapies for human diseases such as cancer. CD2 and 2B4 are receptors on iNKT cells that both bind to CD48 on target cells and modulate immune activation by facilitating cell-cell interactions and transducing intracellular signals. To determine their roles during iNKT cell activation, we examined the TCR-induced killing activity of murine iNKT cells that lacked expression of CD2 or 2B4. Through these studies, we observe that Cd2−/− iNKT cells fail to kill target cells while 2b4−/− iNKT cells exhibit significantly enhanced cytolysis, both in vitro as well as in vivo. These novel findings suggest that CD2 is a positive and 2B4 a negative regulator of TCR-induced iNKT cell functions. Using biochemical, cellular and confocal microscopy approaches, we are currently investigating whether 2B4 exerts its inhibitory role by directly competing with CD2 for CD48 binding and/or by inducing a negative signal. As CD1d (the ligand for the invariant TCR) and CD48 (the ligand for CD2 and 2B4) are both widely expressed on hematopoietic tumors, further elucidation of the mechanisms by which CD2 and 2B4 regulate TCR-induced iNKT cell anti-tumor activity is of significant scientific and clinical importance. The results obtained from these studies will have an impact by establishing new paradigms for iNKT signaling and offering insights into how iNKT cells can be best activated to enhance host immunity and treat hematopoietic cancers such as leukemia and lymphoma.

**Poster #8**  
**Gender Differences Involving Serotonin (5-HT) Receptors in Type I Diabetic Rats**  
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*Diabetes mellitus* is an important topic of research with vascular and renal dysfunction being some of the many negative impacts observed. Increased plasma levels of 5-HT exist in male diabetic rodent models and previous data in male Japanese diabetic patients demonstrated that inhibition of 5-HT2A receptors reduced observed proteinuria.

Whether these are differences in males and females in 5-HT levels and function is unknown. We hypothesized that increased levels 5-HT receptors may be the cause of the vascular damage observed in diabetics; elevated levels of 5-HT and 5-HT receptors may lead to increased vasoconstriction. We used male and female Sprague-Dawley rats (300-325g) and induced diabetes with Streptozotocin (STZ). At 14 days and 28 days post-onset of diabetes we euthanized the animals then harvested tissues and blood vessels for Western blot and myograph analysis. At day 14, no significant differences in contractile responses in the blood vessels from either the male or female control as well as diabetic rats in the thoracic aorta, renal, superior mesenteric, and femoral arteries existed. At day 28, there was an increased contractile response to 5-HT in the aorta from the diabetic rats in both males and females. There was no change observed in the renal artery or femoral artery from either sex at 28 days. In the kidney cortex at day 14 we observed increased expression of both 5-HT2A and 5-HT2B levels. At day 28 only 5-HT2B in the kidney cortex from the diabetic rats was increased. In the cortex from the female rats we saw increased levels of 5-HT2A at both 14 and 28 days and increased levels of 5-HT2B levels at 28 days. These data clearly show altered expression of 5-HT receptor during the development of diabetes and clear sex differences as well.
Poster #9
Decreased aldehyde dehydrogenase (ALDH)2 activity contributes to coronary endothelial dysfunction in diabetic cardiomyopathy
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Background: Around 8% of Americans acquire diabetes mellitus (DM). Diabetics cause micro and macrovascular complications to develop, that lead to end-organ damage. However, microvascular damage is understudied in diabetic cardiomyopathy (DCM), despite needing extensive coronary perfusion. Hyperglycemia-mediated reactive aldehydes, like 4-hydroxy-2-nonenal (4HNE) are associated with cardiac damage. Aldehyde dehydrogenase (ALDH) 2, a mitochondrial enzyme which detoxifies 4HNE, is implicated in endothelial cell function in vasculature.

Hypothesis: DM-mediated 4HNE-induced coronary endothelial cell (CEC) injury potentiates DCM, which is further augmented by low ALDH2 activity.

Methods and results: Type-1 DM was induced in C57BL/6 wild-type (WT-DM) and ALDH2*2 mice (with intrinsic low ALDH2 activity due to E487K mutation; ALDH2*2-DM). After 3 weeks, in comparison to WT-DM, we found decreased fractional shortening (%FS), an index of cardiac function (55±4 vs 40±11%, p<0.05) and increased area of fibrosis (6±0.7 vs 9.5±2%, p<0.05) in ALDH2*2-DM. This indicates the augmentation of DCM in ALDH2*2-DM compared to WT-DM. Similarly, we found reduced CD31+ coronary endothelial cells (CECs): 2119±277 vs 478±62 cells/mm², p<0.001, increased coronary perfusion: 77±7 vs 94±7 mmHg, p<0.01, and reduced eNOS levels in CECs in WT-DM vs ALDH2*2-DM respectively, which indicates more coronary endothelial dysfunction in DCM in ALDH2*2-DM. Furthermore, to substantiate our findings on role of ALDH2 in endothelial dysfunction, we subjected cultured mouse CECs to high-glucose (HG, 33mM D-Glucose) stress with ALDH2 inhibition by disulfiram (DSF+) or without DSF (DSF-). We found DSF augmented HG-induced 4HNE adducts: 1.4±0.2 vs 4±0.8 folds of Ctrl, p<0.05, mitochondrial ROS: 1.6±0.2 vs 3±0.4 folds of Ctrl, p<0.05, cell death: 28.8±4.2 vs 57.5±8%, p<0.01, and decreased angiogenesis: 35.6±3.3 vs 16.7±3 branches/mm², p<0.05, in CECs (in DSF- vs DSF+ respectively).

Conclusion: ALDH2 plays an important role in protecting CECs from hyperglycemic stress. Decreased ALDH2 activity deteriorates coronary endothelial dysfunction in diabetic cardiomyopathy.

Poster #10
Investigating The Phenomenon of Cancer Associated Thrombosis in vitro Utilizing A Conditioned Media Model
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Venous thromboembolism (VTE) is the leading cause of death, behind the malignancy itself, in patients diagnosed with cancer. Statistically, patients with cancer are four times more likely to develop VTE. With over 1.7 million newly diagnosed patients in 2016, VTE and its associated complications create a challenge to physicians and their patients. First described by Trousseau in 1865, there is a well-documented link between VTE and cancer. Modern work such as the 2005 Multiple Environment and Genetic Assessment (MEGA) study has reiterated the connection between VTE and cancer. Although the risk of cancer associated thrombosis (CAT) is well established, the complex molecular mechanisms underlying this phenomenon have yet to be fully understood. The aims of this research were to develop and test an in vitro model of cancer associated thrombosis in order to better understand and isolate the molecular mechanisms involved. We hypothesized that various factors secreted by cancerous cells are directly responsible for the increased rates of VTE observed in cancer patients. Relative expression levels of thrombotic and inflammatory markers were monitored in murine venous endothelial cells (B6MPVE) treated with media conditioned by a murine glioblastoma multiforme (GBM) cell line (GL261) at time points of 1, 3, 6, 9, 12, 24, 48, and 72 hours. RNA was extracted from the B6MPVE cells and their relative expression of numerous thrombotic and inflammatory markers were quantified via qRT-PCR. Upon analysis, treating endothelial cells with conditioned media from GBM cells resulted in a significant upregulation of several biomarkers of interest. This in vitro model will help facilitate future studies to understand the CAT phenomenon.
In the last few decades, advances in our understanding of the immune system have lead to unprecedented treatments for various types of cancers. Invariant natural killer T cells (iNKTs) comprise a unique lineage of innate-like T lymphocytes with important roles in these developing anti-cancer immunotherapies. Although iNKTs are capable of rapidly producing cytokines and mounting potent cytotoxic responses following engagement by a tumor cell, the lack of knowledge about how these mechanisms are controlled greatly impedes their full potential in both drug and adoptive therapies. One of the most promising approaches to sustaining antitumor immunity is blocking of the immune checkpoints, which control inhibitory pathways that prevent the immune cells from executing desired antitumor actions. To that end, our recent studies demonstrate that 2B4; a cell surface receptor negatively regulates iNKT cell anti-tumor responses both in vitro as well as in vivo. Given that the surface expression of 2B4 is absent on resting iNKT cells but gets upregulated following activation, we postulate that 2B4 may be a novel immune checkpoint for iNKT cells. As CD48, the binding partner of 2B4 is widely expressed on tumors, further elucidation of the mechanisms by which 2B4 regulates iNKT cell anti-tumor activity is of significant scientific and clinical importance. Studies are also underway to examine whether 2B4 regulates the expression and/or functions of other inhibitory receptors that may regulate iNKT cell anti-tumor responses. Collectively, these studies will facilitate a better understanding of harnessing the anti-tumor activates of iNKTs in a clinically relevant manner.

**Poster #12**

**Activation of FceRI modulates GRK2-6 expression in mast cells**

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Mast cells are tissue-resident inflammatory cells that drive the anaphylactic response. Mast cell degranulation culminates in the release of inflammatory mediators consequent to cross-linking of antibody-bound high affinity IgE receptor (FceRI) by allergen/antigen. While the allergic response is largely IgE-mediated, this pathological event can be evoked by other redundant receptor signaling pathways. The G-protein-coupled receptor kinases (GRK) are cytosolic proteins that downregulate cell surface receptors. Previous reports from our laboratory have showed that GRKs, specifically GRK2 regulate signal transduction events that contribute to mast cell degranulation following stimulation via FceRI. The aim of the current study was to determine whether antigen stimulation induced changes in the expression of GRKs (GRK2, 3, 5, and 6) in bone marrow-derived mast cells (BMMCs) that were isolated from B6 and BALB/c mice, and rat basophilic leukemia (RBL, a mast cell line) cells using qRT-PCR and Western Blot. Stimulation of IgE-sensitized BMMCs and RBL cells with antigen for 0, 2, 5, 10, 15, 30, and 60 min had no effect on protein or mRNA expression levels at later time-points (30 and 60-min, n=3, p>0.05). At earlier time-points (5, 10 and 15 min), however, stimulation of FceRI evoked a robust decrease in GRK2, GRK5 and GRK6 gene expression that was greatest at 15 min post-stimulation (n=3, p<0.05). These preliminary findings suggest that GRK2-6 may sub-serve an important role in the intracellular signal transduction cascade that culminates in mast cell degranulation.
In the study of asthma, recalcitrant to conventional pharmacotherapies, resistant phenotype of these cells. The results obtained from these studies will provide insights into how the functions of critical regulator of Th2 allergic response and may thus serve as an attractive therapeutic target for asthma. To that end, we are investigating that role of CD2 in hu-iNKT cell response to allergens as well as its contribution to the corticosteroid-resistant phenotype of these cells. The results obtained from these studies will provide insights into how the functions of hu-iNKTs can be harnessed in a therapeutically relevant manner for the treatment of asthma patients, particularly those that are recalcitrant to conventional pharmacotherapies.
**Poster #15**

**Acetate, the Metabolite of Ethanol, Increases Cytosolic Calcium and mRNA Expression Levels of EGR1 and TNFα in Dopaminergic Like PC12 Cells**

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We have previously reported that acetate activates NMDAR in vivo and in vitro, increases pro-inflammatory cytokine mRNA expression, and cytosolic calcium levels, all of which may contribute to neurotoxicity. Neural growth factor (NGF) derived PC12 cells are a reasonable model used in substances of abuse and neurodegeneration studies as they are primarily dopaminergic in nature. Neurotransmitter release is governed in part from rises in cytosolic calcium levels which facilitate loading of synaptic vesicles and exocytosis from the synaptic cleft. We hypothesized that acetate may have similar effects in NGF derived PC12 cells which may facilitate increased dopamine release contributing to alcohol dependence. NGF derived PC12 cells exposed to (2 mM) acetate had a significant (p<0.05) increase in cytosolic calcium relative to baseline, measured in real-time with a fluorescent calcium probe (Fluo-4AM). Additionally, acetate (2 mM) significantly (p<0.05) upregulated mRNA expression levels of a calcium dependent protein, early growth response 1 (EGR1) at 15 and 30 minutes post acetate treatment, 5 and 6 fold respectively relative to control. EGR1 is an early response protein implicated to alterations in synaptic plasticity and also the immune response. Furthermore, 30 minute treatment with acetate also significantly (p<0.05) upregulated mRNA expression of tumor necrosis factor alpha (TNFα), 2 fold compared to control. This data suggests that physiological circulating levels of acetate (2 mM) following ethanol consumption and metabolism lead to increased cytosolic calcium levels, and mRNA expression levels of EGR1 and TNFα in dopaminergic like PC12 cells. These in combination may contribute to the development of alcohol dependence and alcohol associated neurotoxicity.

**Poster #16**

**High Salt Diet Plus Fructose Water Intake Induces Hypertension**

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Prior studies have indicated that a high salt (HS) diet in combination with high fructose intake results in hypertension. This study aims to tests the metabolism of rats with a HS diet or a normal salt (NS) diet with and without fructose drinking water. Detailed metabolic parameters weren’t measured. Seven-week-old, Sprague-Dawley (SD), male rats were divided into four groups (n=6) and fed the following diets: NS (control), HS (4% NaCl), NS diet with 20% fructose water (F), and HS (4% NaCl) diet with 20% fructose water (HS+F), respectively. Blood pressures were measured twice a week via tail-cuff method for two week, then they were transferred into individual metabolic cages. Rats were given 24 hours to acclimated to their metabolic cages and then their food intake, water intake, urine output, and fecal output was measured. The beginning-baseline blood pressure indicated no significant difference between all groups. A three-week diet of a HS+F treatment has confirmed an increase in mean arterial pressure (MAP) by 26 mmHg (p < 0.05) compared NS diet. No significant increase was observed in the other two groups (C: 89.43 ± 5.44, HS: 96.89 ± 4.14, F: 96.50 ± 7.09, HS+F: 126.63 ± 9.43; mmHg). Metabolic measurements have shown a significant increase in water intake for HS and HS+F (C: 27.88 ± 1.98, HS: 59.17 ± 6.40, F: 35.14 ± 5.23, HS+F: 54.75 ± 6.00, mL) and in urine output (C: 13.53 ± 1.73, HS: 39.67 ± 2.21, F: 13.27 ± 1.63, HS+F: 28.13 ± 1.42, mL) in comparison to NS diet. A diminish of food intake occurred in the HS+F group only (P<0.05) (C: 36.10 ± 3.60, HS: 25.11± 0.90, F: 21.67 ± 0.70, HS+F: 14.51 ± 1.02, mL); feces output (C: 4.10 ± 0.82, HS: 7.17± 1.09, F: 3.72 ± 1.28, HS+F: 3.92 ± 0.79, mL). Osmolary was taken via the osmometer (OSM) to further determine the osmotic strength of urine. Studies have revealed that the reduction in osmolality correlates to an increase in blood pressure. Osmolality of urine samples (n = 9) were tested and verified that urine osmolality in 4% NaCl high salt plus 20% fructose SD rat groups decreased by 85.24 mmol/kg (p < 0.0012) compared to the control group. Evidence indicates that the SD rats which have endured a 4% NaCl high salt food diet plus 20% fructose water have a mean arterial blood pressure increase of 26.3 mmHg (p < 0.005) at day 21. Four groups of SD rats were studied over 21 days (n = 3 each), three replicated trials were performed: control, 4% NaCl high salt food diet, 20% fructose, and 4% NaCl food diet plus 20% fructose water. No significant difference was found in the mean arterial blood pressure when only 20% fructose or only 4% NaCl food diet was given to SD rats independently. Metabolic measurements were taken on each SD rat; this includes inputs and outputs of water, urine, food, and feces which were obtained every 24 hours for three days (n=6), two replicated trials were completed. Evidence showed an increase in thirst for water by 7.26 mL (p < 0.01) in the 20% fructose SD rats and an increase of 26.87 mL (p < 0.00) in the 4% NaCl high salt plus 20% fructose SD rats. This research expressed that there is a decrease of 14.43 mL (p < 0.01) in food intake for the 20% fructose SD rats. The 20% fructose SD rats ingested 18.16 mL (p < 0.03) of additional food compared to the 4% NaCl high salt plus 20% fructose SD rats. An increase of 26.14 mL (p < 0.00) of urine output was demonstrated in 4% NaCl high salt SD rats. 4% NaCl high salt plus 20% fructose SD rat group showed an increase of 14.6 mL (p < 0.00) in urine output. The 4% NaCl high salt SD rats demonstrated a low fecal output of 13.29 mL (p < 0.00) in this study.
Poster #17
The potential role of the adaptor protein SAP in CD2 signaling pathway
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Invariant natural killer T cells (iNKTs) comprise a unique subset of T lymphocytes with important roles in host immunity including protection against specific pathogens and cancers. Most iNKTs express an “invariant” T cell receptor (TCR) that confers specificity for glycolipid antigens when presented by the MHC class I-like molecule CD1d. Following TCR engagement; iNKTs rapidly secrete cytokines, transactivate other immune cells as well as exhibit robust killing of cancer cells. However the signaling mechanisms that control iNKT cell functions remain poorly understood. Our recent studies demonstrate a novel cell intrinsic role for cell surface receptor CD2 in iNKT cell functions. Specifically, Cd2−/− iNKTs exhibit a significant 70-80% diminution in target cell killing. Additionally, the cytolytic activity of SAP (SH2 domain-containing adaptor protein)−/−deficient and Fyn (Src kinase)−/−deficient iNKTs is impaired to a similar extent to those observed for Cd2−/− iNKTs, suggesting that SAP and Fyn function downstream of CD2. Signaling through CD2 is dependent on its cytoplasmic tail that has no intrinsic protein tyrosine kinase activity and lacks tyrosine residues able to serve as docking sites for SH2 domains upon phosphorylation. Several SH3 domain-containing proteins interact with the cytoplasmic region of CD2, including Src kinases, Fyn and Lck. Using biochemical and overexpression studies, we are currently examining whether SAP and Fyn interact with the cytoplasmic tail of CD2. As SAP is a known binding partner for Fyn, we postulate that SAP interacts with CD2 via Fyn. Efforts are underway to manipulate iNKT cell functions therapeutically for cancer and other diseases. However, before these efforts can be fully realized, it is necessary to define how iNKTs recognize and respond to targets, including malignant or infected cells. It is likely that the pathways uncovered in these studies could be exploited as a novel therapeutic approach for patients with advanced malignancies, such as leukemia.

Poster #18
3-D Printing Open Source Click-MUAC Bands for Identification of Malnutrition
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Objective An effective method for the diagnosis of severe acute malnutrition is the measurement of the middle upper arm circumference (MUAC). Current methods for measure MUAC in a pre-hospital setting is through the use of measuring tape indicators, which require users to be trained in how to apply, adjust the tightness and read the device properly. This represents a challenge to using MUACs in many developing world contexts. This study explores the technical viability to overcome some of these challenges with conventional MUAC measurement methods using open source 3-D printable click-MUAC bands.

Methods: The dimensional accuracy of the open source 3-D printable click MUAC band was quantified with a digital micrometer to ensure reproducibility. The durability is evaluated by putting bands through a deformation test to simulate the use of the band in the field 500 times. The production costs are quantified using the mass of the filament and electricity consumed to manufacture each band.

Findings: The click MUAC bands are dimensionally accurate (inner circumferences of +/- 0.50 mm) and durable (surviving 500+ deformations with no notable residual deformation). The 3-D printable click MUAC bands are easier to use and provide a significant cost savings (93.85% to 97.98%) when compared to current MUAC measurement methods.

Conclusion: The open-source 3-D printed click-MUAC bands offer a viable alternative to the current methods of obtaining MUAC measurements.

Figure 1. The OpenSCAD code (left) and solid model (right) of the 115 mm 3-D printable click MUAC band
Effect of Sedentary Conditions on the Expression of GABAAα2 Receptor Subunits in Bulbospinal C1 and non-C1 Neurons along the Rostrocaudal Extent of Rat Rostral Ventrolateral Medulla (RVLM)

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Bulbospinal RVLM neurons play a crucial role in the regulation of sympathetic nerve activity and blood pressure, and can be characterized phenotypically as catecholaminergic (C1) or non-catecholaminergic (non-C1). Although excitatory in nature, the tonic activity of the RVLM is strongly influenced by the prevailing level of the inhibitory GABAergic tone. Following sedentary conditions, RVLM neurons also exhibit structural and functional plasticity in a region-dependent manner, with enhanced dendritic branching and increased glutamate responsiveness in rostral versus caudal regions. Because withdrawal of GABAergic tone produces net excitation of RVLM neurons primarily via GABAA receptors, it is possible that sedentary conditions also alter GABA receptor expression in a region-dependent manner. Thus, the purpose of the present study was to test the hypothesis that sedentary conditions reduce GABAA receptor expression in rostral regions of the RVLM. To this end, male Sprague-Dawley rats were divided into sedentary (no wheels) and physically active (running wheels) conditions for 12 wks (n=4 ea). Spinal cord injections of cholera toxin B (CTB) labeled bulbospinal neurons. We examined coronal sections processed for triple-labeling immunofluorescence using laser confocal microscopy, and quantified CTB-positive C1 and non-C1 neurons expressing the GABAAα2 receptor subunit. As expected, more bulbospinal C1 cells expressing GABAAα2 occurred in caudal versus rostral RVLM (p<0.05). Sedentary rats had fewer bulbospinal C1 cells that expressed GABA Aα2 only in the rostral most subregions of the RVLM when compared to active rats (p<0.05). The number of bulbospinal non-C1 neurons also varied rostrocaudally. However, there were more non-C1 neurons in rostral compared to caudal RVLM with no overt differences between sedentary versus active groups. Decreases in RVLM neurons expressing GABAA receptors in rostral RVLM are consistent with previous data demonstrating enhanced sympathetic outflow following sedentary conditions, which may in turn contribute to the increased incidence of cardiovascular disease in sedentary individuals. (R01-HL096787-06; NHMRC 1025031; AHA25810010).
**Poster #21**  
Tai Chi Intervention for Teenagers with Autism Spectrum Disorder  
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Oakland University William Beaumont

Individuals on the autism spectrum (ASD) often experience motor challenges. Dance/Movement Therapy (D/MT) has addressed this aspect of the disorder with moderate success. As Tai Chi involves following an instructor in a series of simple but controlled movements, it is easy to draw parallels between D/MT and Tai Chi. As part of OUCARES summer camp program, Tai Chi classes were offered for higher-functioning ASD teenagers to measure changes in motor attunement. We hypothesized that Tai Chi practice would result in improved motor following skills, as measured quantitatively with pressure sensor technology. The Tai Chi intervention sessions were 30 minutes, twice a week, eight sessions in total. In addition to psychometric data, we collected pressure sensor recordings (ADInstruments, Power Lab 26T) from the back of the hand while participants performed ‘playing hands’ for ten cycles. Playing hands is one of the fundamental movements of Tai Chi, performed between two people where one is to continuously move their hand in a horizontal circular motion without losing contact with the back of their partner’s hand. This motion not only requires some motor skill, but also self-other awareness and a willingness to engage. Through the pressure sensor, we recorded total contact duration (D), number of contacts and breaks, and contact force. The data show that the voluntary contact duration increased for all participants except one (n = 18, pre mean D = 27.8 sec, post mean D = 33.4 sec, p = .076), indicating increased engagement. For the two participants that engaged in all 8 sessions, the % contact increased significantly from 84.5% and 46.5% in session 1 to 100% for both by session 8. The data from this pilot study suggest Tai Chi is a useful intervention for developing motor attunement and self-other engagement.

**Poster #22**  
Androgen receptor expression in chemically-defined hypothalamic nuclei associated with energy balance and reproduction  
Cara, Alexandra; Elias, Carol  
University of Michigan

Androgens are important for both reproductive and metabolic physiology of males and females. Disorders of androgen imbalance, hyperandrogenemia in females or hypoandrogenemia in males, increases risk of visceral adiposity and type 2 diabetes. In females, androgen excess is present in polycystic ovary syndrome (PCOS), a highly prevalent disorder also associated with obesity and insulin resistance. Androgens act upon androgen receptors (AR), expressed in many metabolically relevant tissues. In the brain, AR is abundant in hypothalamic nuclei involved in energy balance. This is of particular interest due to sexually dimorphic differences in central response to metabolic hormones, such as leptin. While previous studies have focused on the contribution of estrogen receptor and estrogens on the central regulation of metabolism, the role of androgens acting on AR is largely unknown. In rats, AR is expressed in the arcuate (Arc), ventromedial (VMH), and ventral premammillary (PMv) nuclei. However, little is known about the distribution and chemical identity of AR neurons in male and female mouse brain. We have developed an in situ hybridization probe to target AR mRNA in mice. As described in rats, strong AR expression is observed in the medial pre-optic area, PMv, and ventro-lateral subdivision of the VMH. However, species differences were apparent. For example, AR is abundant in the dorsomedial VMH of rats but not mice. In the PMv and Arc, AR is expressed in leptin receptor neurons, which are important for energy homeostasis and fertility. Our findings suggest that AR in hypothalamic neurons contributes to metabolic dysfunction observed in states of androgen imbalance. Further studies will be necessary to assess if AR in metabolically relevant neural circuits is required for energy homeostasis in both sexes.
Poster #23
Effects of Metabolic Remodeling on Mechanical Function in Heart Failure
Lopez, Rachel; Van den Bergh, Françoise; Gao, Xin; Michele, Daniel; Beard, Daniel
University of Michigan

An estimated 6.5 million Americans are affected by heart failure, a condition in which the heart is unable to meet the blood and oxygen demands of the body. Pathophysiological changes that occur during heart failure include remodeling of the myocardium and changes in the metabolic state that are associated with the depletion of key metabolic pools. Our group has previously predicted that depletion of the myocardial adenine nucleotide pool results in an impaired ability for oxidative phosphorylation to maintain phosphate metabolite levels and ATP hydrolysis potential. We further hypothesize that the resulting alterations to phosphate metabolite levels impairs mechanical function of the heart, contributing to the heart failure phenotype. To test this hypothesis, 3-week-old Sprague Dawley rats underwent transverse aortic constriction (TAC) to induce heart failure. Hearts were excised after 15 weeks to measure mitochondrial oxidative capacity and cytosolic metabolites. Mechanical function was measured by echocardiography to understand its relationship with cytosolic metabolite levels. We observed a loss of fatty acid and carbohydrate oxidative capacity with impaired mechanical function in heart failure. Our preliminary results also show that the adenine nucleotide pool levels are depleted in the TAC rats. Taken together these results suggest that depletion of the adenine nucleotide pool and reduction in mitochondria oxidative capacity both contribute to metabolic dysfunction in this model, and that this metabolic dysfunction contributes to mechanical pumping failure. Future studies include investigating whether cardiac function can be improved in heart failure by targeting metabolic pool regulation. We will develop an inducible 5’nucleotidase (5’NT) knock-out rat. The 5’NT enzyme depletes the nucleotide pool by hydrolyzing AMP to adenosine. This will help determine if the pathological depletion of the nucleotide pool in a rat model of heart failure can be prevented by knocking out 5’NT, and if preservation of the nucleotide pool restores mechanical function.

Poster #24
Pyrurate Dehydrogenase Activation follows the Cytosolic ATPase Activity during Contraction in Skeletal Muscle with High and Low Mitochondrial Density
Kasper, Jonathan Lewis, Matthew Gudziak, Greg Meyer, Ronald Wiseman, Robert
Michigan State University

Pyrurate dehydrogenase (PDH) regulates mitochondrial glucose utilization and is activated during exercise to increase skeletal muscle glucose disposal. The activation status of PDH is regulated by reversible phosphorylation catalyzed by a set of pyruvate dehydrogenase phosphatases (PDP) and kinases (PDK) allosterically regulated by Ca2+ and the cellular energetic status respectively. During contraction these allosteric regulators covary therefore the relative contribution to PDH activity is difficult to discern. We hypothesized that by reducing mitochondrial density that the sustainable energetic load and calcium cycling could be temporally separated. To achieve this, adult male Wistar rats were treated with methimazole (MMI), a thyroid hormone synthesis inhibitor, for four weeks in their drinking water (0.025% w/v) to reduce skeletal muscle mitochondrial density by 50%. This allows for similar energetic steady states as control animals when stimulated at half of the contractile intensity, thus reducing the effective Ca2+ load with the same energetic demand as controls. Phosphorus nuclear magnetic resonance spectroscopy was used to determine the muscle energetic status in vivo and parallel benchtop experiments were performed to obtain muscle samples for determination of PDH activity using a radioisotopic tracer assay. Twitch stimulation intensities of the triceps surae group were controlled through electrode implantation at the sciatic nerve and force output was recorded. Control rats were stimulated at 0.5 and 1.0Hz intensities with MMI treated rats stimulated at 0.25 and 0.5Hz. Interestingly, when PDH activity was plotted against the steady state energetics (GATP) there was a rightward shift in this relationship for the MMI treated group. However, when plotted against the cytosolic ATPase activity (mM/s) or total force output (g*s), indicators of the effective Ca2+ load, no significant difference was found. These data suggest a primary role of Ca2+ and PDP activity in the control of PDH activation and regulation of glucose disposal during exercise.
Poster #25

In Vivo Characterization of Muscle Hemodynamic Recovery in Electrically–stimulated Rat Hindlimb Using Magnetic Resonance Imaging

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This study aimed to quantitatively resolve in vivo muscle blood flow (BF) dynamics in response to contractile activity and characterize the relationship between hyperemia and metabolic demand in O2 over the physiological range of muscle function in wistar rat (males, n=9). Leg muscle contractions were electrically-induced (sciatic nerve stimulation) for 3min at 0.5Hz, 0.75Hz, 1Hz, 2Hz, 3Hz, 4Hz, and 5Hz and randomly applied with 20min recovery period. Contraction-induced BF changes were quantified in the femoral vein from phase contrast velocity map images at 7T. Measurements were continuous over the 20min of recovery (time resolution=15s) and every 30s during stimulation (n=4). O2 delivery rate was determined assuming O2 arterial content = 7.60µmol (FiO2=100%). Contraction-induced BF increased with respect to stimulation intensity and peaked at 89.4±17.1 ml-min-1·100g muscle-1 post 5Hz. Assuming a constant ATP cost per twitch of 0.26 µmol ATP-g muscle-1 and a Phosphate to O2 ratio of 6, we showed that O2 delivery rate matches the corresponding O2 cost of the contraction up to 2Hz. Specifically, O2 delivery increased from 1.8±0.5 µmol-min-1·g muscle-1 at 0.5Hz to 6.3 ± 0.9 µmol-min-1·g muscle-1 at 2Hz intensity for a corresponding O2 cost ranged from 1.55±0.18 µmol-min-1·g muscle -1 to 6.4±0.5 µmol-min-1·g muscle-1. Beyond 2Hz, O2 delivery was no longer sufficient to meet the corresponding metabolic demand. Measurements during stimulation suggest that contracting muscle perfusion was compromised at those intensities. Regardless intensity, BF returned to baseline within 20min following an exponential decay. However, above 2Hz, recovery kinetics were characterized by an early delay that increased with increasing frequencies. These results suggest that within muscle aerobic range (≤2Hz) metabolic demand for O2 is the primary controller of contraction-induced hyperemia. Above this threshold, the systematic delay in BF recovery suggests that factors other than those sensing O2 demand might contribute to hyperemia potentially metabolic acidosis or elevated temperature. However, the relative increase in BF per gram force was significantly reduced following stimulations >2Hz compared to lower intensities presumably due to intramuscular pressure during the contractile phase. Support:NIH/DK:09521.

Poster #26

Reliability of heart rate variability as an assessment of cardiac sympathetic activity in humans

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Spectral analysis of heart rate variability (HRV) has been widely used as an indirect, non-invasive method for assessing cardiac autonomic function in numerous psychophysiological and clinical research studies. However, there is debate whether or not the low-frequency (LF) component of the analysis provides accurate insight into cardiac sympathetic activity in humans. The purpose of this study was to evaluate the reliability of the LF component of HRV compared to that of direct measures of peripheral muscle sympathetic nerve activity (MSNA) in humans. Sixteen adults (9 men and 7 women) participated in three experimental sessions across two laboratory visits. Experimental sessions one and two were conducted during the first laboratory visit and separated by 45 minutes of supine rest, while the third session was completed approximately one month after the first laboratory visit. Each session included a five-minute supine baseline that included recordings of MSNA via microneurography, R-R intervals using a three-lead electrocardiogram, and respiratory rate via a pneumobelt. Intraclass correlation analyses (Cronbach’s α coefficient) were performed to determine the consistency of measurements across the three sessions. Respiratory rates were consistent across the three sessions (Cronbach’s α = 0.90, P < 0.001). The spectral analysis revealed that the high-frequency (HF) component, which is primarily associated with cardiac parasympathetic activity, was highly reliable (Cronbach’s α = 0.90, P < 0.001). In contrast, the LF component was not significantly consistent across the three sessions (Cronbach’s α = 0.45, P = 0.078). As previously reported from our laboratory, MSNA was highly consistent across the three sessions (Cronbach’s α = 0.84, P < 0.001). In summary, our findings demonstrate that unlike MSNA, the LF component of HRV is not a stable and reliable measurement of cardiac sympathetic activity in humans.
**Poster #27**

A study of gender influence on the pattern of cardiorespiratory response to transient and unrelenting acute severe hypoxemia in adult rats

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Males are at greater risk of sudden death across the spectrum of life stages. Conditions such as sudden infant death during sleep (SIDS), sudden unexplained death in epilepsy (SUDEP), and sudden death in adulthood are all biased towards a preponderance of male victims. We hypothesized that gender-related differences exist in the pattern of cardiorespiratory responses to acute severe hypoxemia (asphyxia) are at least partly responsible for this gender disparity in incidence of sudden death. To test this, we studied pentobarb-anesthetized and age-matched adult male (n=9, 702±86g) and female (n=9, 342±49g) Sprague Dawley rats, instrumented to directly monitor respiratory and cardiovascular variables. In a first transient exposure to asphyxia, animals were switched back to room air exposure upon demonstrating hypoxia-induced respiratory arrest, allowing for the potential of a spontaneous autoresuscitation. In a second unrelenting exposure to asphyxia, animals were not returned to room air during respiratory arrest, and gasping. In response to transient asphyxia, we found that systolic blood pressure (SBP) was significantly lower in females (141±14mmHg) vs. males (162±29mmHg) at the point where the first gasping breath was observed (p=0.038), as well as at the point where eupneic breathing resumed (p<0.001). During this same autoresuscitation process, there were also gender differences observed in the duration of primary apnea (97.8±15.5 in males, vs. 81.8±8.0 sec in females, p<0.01). There were no other gender differences to transient or unrelenting asphyxia in the animals we studied. We conclude that adult male rats experience longer primary apnea in response to transient asphyxia, and sustain higher SBP during the gasping and recovery phases of autoresuscitation. These differences equate to a delay the process of reoxygenation upon gasping in male animals, and potentially less flexibility in their ability to alter blood flow distribution to facilitate oxygen delivery during respiratory autoresuscitation.

**Poster #28**

The Characterization Of LN229/mKate Glioblastoma Multiforme Cells Cultured in vitro Utilizing Artificial 3D Chitosan-Alginate Scaffolds

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Glioblastoma multiforme (GBM) is the most malignant primary brain tumor with a median life expectancy of 15-17 months. Studies suggest a population of cancer stem cells (CSCs) is directly responsible for tumor recurrence following initial treatment. Development of clinically effective therapies that target CSCs are desperately needed to improve existing treatments. There are significant limitations to research involving CSCs, including the costs of culture and a slow rate of growth for CSC cultures. Previous work demonstrated that GBM cell lines cultured on artificial three-dimensional (3D) chitosan-alginate scaffolds de-differentiate into CSC-like cells by increasing the expression of CSC biomarkers. Recently, we detailed a refined synthesis method for 3D chitosan-alginate scaffolds and demonstrated their efficacy for in vitro cell culture. Our previous work suggests that populations of CSC-like cells can be generated from GBM cell lines using 3D chitosan-alginate culturing techniques. The aim of this current project was to characterize the GBM cell line LN229/mKate following culture on 3D chitosan-alginate scaffolds. We hypothesized that cells cultured on 3D chitosan-alginate scaffolds would exhibit a CSC-like phenotype. After growth of the LN229/mKate cells on the scaffolds, the proliferation rates and relative expression levels of several CSC-markers were measured. These cells were then cultured using standard adherent and suspension conditions without chitosan alginate to determine whether the cell populations retained their CSC-like phenotype. For this work, cellular proliferation of adherent cultures was monitored by manual cell counting, and in suspension cultures via fluorescence microscopy at 1, 3, 5, and 7 days. Additionally, RNA was harvested from scaffold-cultured cells and assessed for relative expression levels of CSC-biomarkers using qRT-PCR. Our results suggest that LN229/mKate cells cultured on 3D chitosan-alginate scaffolds morphologically and phenotypically resembled CSCs. With additional testing, artificial 3D chitosan-alginate scaffolds may support and expand future studies involving CSCs.
Poster #29
THE PSYCHOPHYSIOLOGY OF SELF-FORGIVENESS
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We are interested in the physiological correlates of self-forgiveness (SF). First, we collected psychometric data on stress levels, trait anger, and trait and state forgiveness. Participants were then connected to a 3-lead ECG, frontalis EMG, and respiratory belt (ADI PowerLab 26T, LabChart v8) and listened to a 40 min SF audio based on the Internal Family Systems (IFS) model. Afterwards, we re-administered the three forgiveness instruments. Our hypothesis was that ratings of SF, and possibly other-forgiveness, would increase after listening to the IFS-based audio. Scores did increase very significantly for all three forgiveness instruments (p-level = 0.005, 0.00001, and 0.008, n = 15), confirming the effectiveness of the IFS-based imagery. HR did not vary during the recording, but heart rate variability (HRV-SDRR) were significantly different from baselines during a guided relaxation (p = .011) as well as the SF portion (p = 0.02). A similar pattern was observed for HRV total power and the LF/HF ratio, but not the VLF or LF bands. Power in the HF band dropped significantly during the guided relaxation and remained significantly lower for the remainder. A significant decrease in respiration rate (BPM) for most participants also occurred during guided relaxation (p = .001) and was correlated with SF (R = 0.56, p = 0.03). BPM variability, inspiration t, and expiration t were also significantly different during the guided relaxation. We are currently analyzing the frontalis EMG data and exploring correlations with the ECG and respiratory data, as well as any correlations with the psychometric data. Overall, the findings indicate the IFS-based SF intervention is highly effective and may be facilitated by enhancing parasympathetic tone.

Poster #30
MILD INTERMITTENT HYPOXIA WITH SUSTAINED HYPERCAPNIA IMPROVES AIRFLOW, REDUCES THERAPEUTIC CPAP AND BLOOD PRESSURE IN PARTICIPANTS WITH OBSTRUCTIVE SLEEP APNEA.
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Intermittent hypoxia (IH) elicits long term facilitation of ventilation and upper airway muscle activity. The latter form of plasticity could contribute to stabilizing the upper airway. Based on this possibility, we examined if administration of mild IH accompanied by sustained hypercapnia reduces the continuous positive airway pressure (CPAP) required to treat obstructive sleep apnea (OSA). In addition, we investigated if daily exposure to mild IH would elicit changes in the autonomic nervous system, leading to a decrease in blood pressure in individuals with OSA.

Methods: Ten male participants with OSA initially visited the laboratory to confirm the presence of sleep apnea, while a second visit was used to determine the therapeutic CPAP (TP). On a third visit, the participants completed a sham protocol (i.e. normoxic and normocapnic conditions), which mimicked the timeframe of the IH protocol that would be introduced in a subsequent visit, while being treated with TP. During the sham recovery period, the CPAP was reduced in a step-wise fashion to measure the change in flow and upper airway resistance. On a fourth visit participants were treated with TP. PETCO2 levels were increased and sustained 3 mmHg above baseline. Subsequently, twelve 2-minute episodes of hypoxia (PETCO2 = 50 mmHg) separated by 2-minute intervals of normoxia were administered. During recovery, the CPAP was reduced in a step-wise fashion until flow limitation was evident. In a separate set of experiments two subjects with OSA underwent daily exposure to IH on 15 occasions over 3 weeks.

Results: Following exposure to intermittent hypoxia minute ventilation (P < 0.001) and tidal volume (P < 0.001) during end-recovery and the step-downs in positive pressure were greater compared to B2 (P < 0.001). Inspiratory flow and resistance remained at baseline levels following exposure to intermittent hypoxia after TP was reduced by 5 cmH2O. In contrast, after completion of the sham protocol inspiratory flow was reduced (P < 0.001) and resistance was increased (P < 0.01) in response to the 5 cmH2O reduction in positive airway pressure. Preliminary data in two participants showed that daily exposure to IH over a 3 week period led to a decrease in systolic (144 ± 1 vs. 128 ± 5 mmHg) blood pressure and an increase in baroreceptor sensitivity (6.5 ± 0.06 vs. 17.9 ± 1.7 ms/mmHg). CPAP compliance data was retrieved from one individual. CPAP use increased from 2 h 52 minutes in the 1st week to 5 h 52 minutes in the 3rd week.

Conclusion: The administration of mild IH and sustained hypercapnia decreases the CPAP required to eliminate apneic events. Additionally, increases in resistance and decreases in flow following CPAP step-downs were significantly mitigated following IH with sustained hypercapnia. Preliminary data from 2 individuals showed that mild IH could effectively treat cardiovascular co-morbidities directly and indirectly by increasing CPAP compliance via reductions in TP.
Poster #31
Mechanisms for posterior cerebral artery remodeling during Angiotensin II-induced hypertension
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The brain is highly susceptible to injury caused by hypertension because the increased blood pressure causes artery remodeling that can limit cerebral perfusion and increase the risk for dementia. The posterior cerebral artery (PCA) regulates the perfusion of brain regions associated with memory formation. We tested the hypothesis that AngII-induced hypertension would increase microgial density and the gene expression of microglia secreted factors to mediate PCA inward remodeling and cerebral hyperperfusion. PCAs were collected from 20-week-old male C57Bl/6 mice to assess structure by pressure myography. Data collected at an intraluminal pressure of 60mmHg are presented as mean ± SEM; Sham vs. AngII (800ng/kg/day for 4 weeks). The expression of the mRNAs for TNFa, IL-6, MCP1, MMP-2, and MMP-9 was assessed in isolated PCAs. AngII caused PCA inward remodeling as evidenced by the reduction in lumen diameter (132 ± 5 vs. 109 ± 8μm, p<0.05). The decreased cerebral perfusion (938 ± 32 vs. 597 ± 54 perfusion units, p<0.05) is due primarily to artery remodeling and not to artery rarefaction (21.4 ± 2.8 vs 16.8 ± 0.9 number of vessels/area, p>0.05). AngII did not significantly change the expression of the mRNA for TNFa (1 ± 0 vs 1.5 ± 0.4) or MMP-9 (1 ± 0 vs 1.1 ± 0.4) (p>0.05). However, AngII hypertension increased the expression the mRNAs for MCP1 (1 ± 0 vs 3.6 ± 0.6), IL-6 (1 ± 0 vs 10.3 ± 3.7), and MMP-2 (1 ± 0 vs 1.7 ± 0.1) (p<0.05) suggesting that these factors may be involved in hypertensive artery remodeling. AngII hypertension may also increase the density of the microglia in the cerebral cortex. Our results suggest that AngII-induced hypertension increases the gene expression of microglia secreted factors to mediate PCA remodeling associated with decreased cerebral perfusion; this could have detrimental effects on neuronal function increasing the risk of vascular dementia.

Poster #32
Imaging renin granule exocytosis in juxtaglomerular cells by Total internal reflection (TIRF) microscopy

Renin is essential for angiotensin I generation and blood pressure regulation. Renin is stored in dense core granules in juxtaglomerular (JG) cells. Renin release is highly regulated with only a small percentage (2 to 5%) of the total renin content being released after maximal stimulation with agonists that increase cAMP. In vivo, stimulation of renin release resulted in rare events of renin granule disappearance as shown by electron microscopy. We found that stimulated-renin release is in part mediated by exocytic proteins present in the renin granules. However, real-time visualization of renin granule exocytosis has not been done. We hypothesize that renin exocytosis from JG cells is due to controlled exocytic events, termed kiss-and-run exocytosis, where fusion of the granule with the plasma membrane is transient and granule integrity is maintained. To study this we generated a new construct in which full length mouse renin was tagged with yellow fluorescent protein in its Carboxyl-terminus (Renin-YFP). First, we packed the Renin-YFP construct in adenoviruses (Ad-Ren-YFP) and characterized its proper expression and activity in Att20 cells, an endocrine pituitary cell line that does not express endogenous renin. By Western Blot, we observed a band at the expected molecular mass of renin-YFP (70 kDa), detected with antibodies against YFP or renin (n=4). Then, we tested whether Ad-Renin-YFP retains its enzymatic activity (rate of angiotensin I production from angiotensinogen) in Att20 cells. Renin activity was only detected in Att20 cells transduced with renin-YFP (n=3; p<0.01). To monitor exocytosis of renin-YFP, we transduced primary cultures of mouse JG cells. After 24 hs, we transferred JG cells to a 37C chamber and imaged YFP-containing granules by TIRF microscopy. TIRF allows imaging of granule movements and changes in fluorescence intensity within 250 nm of the plasma membrane. Under baseline conditions we observed an average of 16±4 granules per cell docked with the plasma membrane (n=12). Translational movement (X-Y planes) of docked granules was negligible and most granules remained docked during 20 minutes of imaging. TIRF events were quantified over 10 minutes by measuring: A) recruitment of new granules, B) full fusion followed by granule disappearance and C) rapid axial movement (Z-axis) of docked granules (observed as rapid bursts of fluorescence followed by return to baseline intensity). In the absence of cAMP, the total number of events per cell was low and no full fusion events were detected (1.5 ±0.5 total events per cell, n=4). After stimulation with cAMP, the number and frequency of events increased 4 fold to 5.3±1.0 events per cell (n=8, p<0.05). Only one full fusion event was detected (n=8 cells, 152 granules imaged). Most of the events caused by cAMP (73.5%) were due to fluorescence bursts of docked granules and 36.5% of events were caused by recruitment of newcomer granules to the TIRF field. We conclude that in JG cells, full fusion of granules is not the main mechanism of renin exocytosis. The rapid bursts in fluorescence intensity of docked granules suggest that kiss-and-run is the main mechanism of stimulated-renin exocytosis. This is the first time that a renin-YFP construct was characterized and renin exocytosis is imaged in JG cells.
Poster #33
The Development of a Co-Culturing System to Study the Phenomenon of Cancer-Associated Thrombosis

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The formation of cancer is due to the body’s own cells undergoing mutations that alter the body’s own physiological pathways at the site of the cancer and throughout the body. One of the side effects of cancer is the increased likelihood of diagnosis for venous thromboembolism (VTE). Cancer associated thrombosis (CAT) is one of the leading causes of death of cancer victims, second only to the malignant effects of the disease itself. Nearly all research on the topic of CAT has been clinical. The lack of research in vitro has lead to the inability for defining the key proteins and mechanisms that form the basis of the correlation between cancer and VTE. The aim of this research looks to establish an in vitro model for studying CAT that will allow for the identification of important molecular pathways that play a role in the pathogenesis of CAT. This research will test the effectiveness of a co-culturing model at replicating this phenomenon. This model will be established by culturing a murine glioblastoma multiform (GBM) cancer cell line (GL261), with a murine primary venous endothelial (B6MPVE) cells, separately, while they are immersed in the same media using a trans well system. The RNA from the B6MPVE cells will be collected at various time points to study the acute and chronic changes. The samples will then be analyzed using qRT-PCR to observe the expression of thrombotic and inflammatory factors. Never before has an in vitro model of CAT been developed, limiting the ability to further study the complex pathway interactions between these two conditions, making this research even more valuable. The proposed model will give a starting platform for future studies researching the connection between VTE and cancer.

Poster #34
Psychophysiological Effects of Acute Mindfulness Meditation

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Anxiety is one of the most common mental health disorders in the United States. This disorder is strongly associated with hypertension, arterial stiffness, and cardiovascular disease. Eight-week mindfulness meditation programs are often used to treat anxiety, however there is little known about the psychophysiological effects of a single meditation session. The purpose of this study was to assess the effect of an introductory one-hour mindfulness meditation session on anxiety, blood pressure, heart rate variability, and arterial stiffness. Understanding the effects of acute mindfulness could help to improve the design of anti-anxiety therapies. Five young adults with Beck Anxiety Inventory (BAI) scores of 8 or higher (mild to severe anxiety) participated in three experimental sessions across three weeks. During the first orientation session, the BAI was used to measure the anxiety of potential participants. Baseline cardiovascular variables including blood pressure, heart rate, heart rate variability, and arterial stiffness were measured. The following week, participants returned for the mindfulness session where their baseline cardiovascular measurements were repeated and then they were guided through one-hour of mindfulness meditation. Cardiovascular measurements were recorded again at 0 and 60 minutes post-meditation. The BAI was also taken at the post-60 time. Participants were encouraged to continue to practice mindfulness and they returned a week later for a third BAI survey. Baseline cardiovascular variables were analyzed using paired t-tests to determine the similarity of the orientation and pre-mindfulness measurements. We then utilized a repeated measures ANOVA to analyze BAI scores and cardiovascular variables over the three sessions. There was a significant (p=0.01) time effect for a reduction of BAI scores from the orientation session through week 3. We did not detect any changes in cardiovascular variables with the limited sample size. Preliminary results suggest that one-hour of introductory mindfulness meditation has psychological benefits for anxious individuals.
The mechanistic role of mitochondrial failure in the development of Type 2 Diabetes (T2D) has garnered widespread support. As a consequence mitochondria have been the target of drug intervention with limited success. In the present study mitochondrial properties and bioenergetics metabolites are quantified in peripheral musculature in early stage T2D in Zucker Diabetic Fatty (ZDF) rats. Citrate Synthase (red: 58+/−16 vs 75+/−10, white: 18+/−2 vs 21+/−3 (μmol/g*min) and Cytochrome C Oxidase(red: 8.2+/−3.9 vs 8.7+/−.96, white: 2.6+/−.6 vs 2.6+/−.3(μmol/g*min)), indicators of mitochondrial content, demonstrated no difference between ZDF control and diabetic respectively. Assuming no difference in muscle fiber type, these results suggest mitochondrial content is not a factor in developing T2D in ZDF. Functional capacity was tested in isolated mitochondria from excised hindlimb muscles using in ZDF rats and challenged with ADP and either pyruvate and malate or palmitoyl-carnitine and malate as substrates. Maximum ADP-stimulated respiration rate was not different when using pyruvate and malate as a substrate (743+/−41 vs. 685+/−58 (O2/mg/min)), but was significantly higher in the diabetics using palmitoyl-carnitine and malate (251+/−14 vs. 338+/−14(O2/mg/min)). These results demonstrate mitochondrial capacity for ATP production is not diminished in ZDF diabetics relative to lean controls and suggests mitochondrial function does not play a role at this stage of diabetes. Finally, bioenergetically important metabolites were assayed to determine the metabolic profile of the muscle (in mM/L*Cell H2O). ATP content was not different between ZDF rats in neither red (8.11+/−.56 vs 8.12+/−.27) nor white (9.2+/−.45 vs 9.02+/−.45) muscle fibers. Creatine content was not different in red (34.1+/−1.48 vs 35.6+/−0.77) but increased in white (41.7+/−35 vs 48.7+/−1.21) diabetic muscle. Taken together these results do not support mitochondrial function being mechanistically linked to development of T2D but do not preclude the possibility that mitochondrial function could be altered later in the disease progression.

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Mathematical Modeling of Tissue Bioenergetics in Streptozotocin-Induced Type 2 Diabetic Rat Muscle

Exercise capacity is often reduced in both type 1 and 2 diabetes as well as other diseases including heart failure and COPD. Because aerobic metabolism is the predominant pathway fueling exercise, alterations in mitochondrial oxidative phosphorylation capacity through lost function or mitochondrial content are both logical explanations. We employed a mathematical model of mitochondrial metabolism developed specifically to quantify the effect of mitochondrial changes on muscle energetics. The model was generated using rat skeletal muscle metabolite content and dynamics to simulate changes in phosphocreatine (PCr) during exercise. Such data is routinely acquired in both humans and animal models of disease using magnetic resonance. To test whether alterations in mitochondrial function occur in T1D, data from streptozotocin treated rats were used (Challiss et al. (AJP, 256(1), 1989)). Initial model parameters were adjusted based on reported metabolite values to simulate the diabetic and control animal skeletal muscle conditions (resting PCr, Creatine, ATP, and ADP content). Exercise transients were simulated at 1.0Hz as performed in the in vivo study by invoking an ATPase rate (.14mM/s) which was comparable to measured ATPase rates in vivo. Model simulations accurately predicted the in vivo data collected in controls by simulating resting PCr steady state and the PCr kinetics associated with exercise. However, to simulate the diabetic condition the mitochondrial density had to be adjusted down by 57% that of control. This change in mitochondrial density is comparable to that seen in the literature for STZ-induced diabetes (48%, Larsen et al.(PR 3/7/2015). Taken together these results suggest changes in mitochondrial content are sufficient to explain the decline in tissue bioenergetics observed in vivo in the STZ model of T1D. Supported by NIH DK 095210.
**Poster #37**
The effect of Hibiscus sabdariffa tea on cardiovascular risk factors in prehypertensive adults

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Nearly one-third of the U.S. adult population has hypertension, which is a leading risk factor for cardiovascular disease (CVD). _Hibiscus sabdariffa_ (HS) tea has been suggested to have cardioprotective properties that may be used to prevent or treat hypertension. However, there is currently a lack of clinical trials to support this claim. The purpose of this pilot study was to examine the effects of HS tea on CVD risk factors. Seven prehypertensive males (27 ± 12 years old; BMI: 28.8 ± 3.8 kg/m2) were randomly placed into either a decaffeinated green tea control or HS tea intervention group. Participants drank two cups of tea per day for six weeks. Blood pressure (brachial and aortic) and arterial stiffness were measured before and after the intervention. We utilized a repeated measures ANOVA analysis to compare pre and post intervention values, with the type of intervention as the between-subjects factor. There was a significant time effect (p<0.03) for a reduction of systolic blood pressure from pre to post HS (120 ± 4 vs. 115 ± 6 mmHg) and control (120 ± 4 vs. 112 ± 7 mmHg). There was a trend for a reduction in diastolic blood pressure (time effect p=0.06). There was a tendency for an interaction between groups (p<0.06) regarding aortic pulse pressure, a predictive risk factor for CVD. Aortic pulse pressure tended to be reduced by HS from pre to post (33 ± 4 vs. 28 ± 5 mmHg), but not by control (33 ± 5 vs. 34 ± 6 mmHg). Arterial stiffness assessed via carotid-femoral pulse wave velocity did not change from pre to post intervention. These initial results suggest that HS and decaffeinated green tea may effectively lower brachial blood pressure, and that HS may be able to lower aortic pulse pressure. Future research will aim to determine the potential blood pressure lowering mechanisms of the flavonoids or anthocyanins.

**Poster #38**
Interferon gamma increases lipolysis and fatty acid oxidation in pancreatic β cells.

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Proinflammatory cytokines, including interferons, are secreted by immune cells and have been implicated in pathogenesis of type 1 diabetes. While interferon gamma (IFNγ) has been shown to affect insulin secretion and β cell survival, its role on β cell metabolism and anti-viral response remains to be investigated. In this study, we found that acute treatment of INS-1 β cells and Wistar rat islets with IFNγ (12 hours) increased mRNA and protein levels of adipose triglyceride lipase (ATGL), the major enzyme that contributes to triacylglyceride (TAG) lipolysis. Increased expression of ATGL by IFNγ was associated with increased fatty acid oxidation (FAO) and upregulation of genes involving in this process. Pharmacological inhibition of carnitine palmitoyltransferase 1A (CPT1A), the rate-limiting enzyme of mitochondrial FAO, resulted in a substantial reduction in oxygen consumption rate in IFNγ-treated INS-1 cells. These changes in lipid metabolism occurred concomitantly with a robust increase in expression of genes that involve in inflammation (STATs and NOS2), anti-viral responses (PKR, MxA), and recruitment of immune cells (CXCL10). To test whether IFNγ treatment amplifies antiviral responses, INS-1 cells were treated with IFNγ for 12hrs followed by poly IC for 12hrs. Pretreatment with IFNγ modestly enhanced expression of IFNβ (2-fold), while subsequent treatment with poly IC increased IFNβ (~200-fold) and this occurred with a commensurate increased in ATGL and CPT1A expression. These data suggest that β cells respond to IFNγ by increasing lipolysis and FAO, which ultimately increase ATP production. This change in metabolism may benefit the anti-viral response of β cells in the early stage of β cell autoimmunity. Keywords: Type 1 diabetes, pancreatic beta cells, interferon gamma, lipolysis, fatty acid oxidation, gene expression.
**Poster #39**
Enhancing the anti-leukemia functions of invariant natural killer T cells using a CD1d-anti-CD19 scFv fusion protein

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In the context of leukemogenesis and immune surveillance, invariant natural killer T (iNKT) cells comprise a unique lineage of CD1d-restricted lipid-reactive T lymphocytes that kill tumor cells and exhibit robust immunomodulatory functions. Most often, optimal tumor-directed iNKT cell responses require expression of the antigen-presenting molecule CD1d on tumors; however, many tumors down-regulate CD1d and thus evade iNKT cell recognition. We generated a soluble bi-specific fusion protein to direct iNKT cells to the site of B-cell tumors in a tumor antigen-specific yet CD1d-independent manner. This fusion protein is comprised of a human CD1d molecule joined to a single chain antibody Fv (scFv) fragment specific for CD19, an antigen expressed on B-cell cancers. The CD1d-CD19 fusion protein binds specifically to CD19-expressing, but not CD19-negative human or murine cells. Once loaded with the iNKT cell lipid agonist galactosyl ceramide (GC), the CD1d-CD19 fusion induces robust in vitro activation and cytokine production by human iNKT cells. iNKT cells stimulated by the GC-loaded CD1d-CD19 fusion also strongly trans-activate T, B and NK cell responses and promote DC maturation. Importantly, the GC-loaded fusion induces robust lysis of CD19+CD1d− Epstein-Barr virus immortalized human B-lymphoblastoid cell lines that are otherwise resistant to iNKT cell killing. Consist with these findings; administration of the GC-loaded fusion protein controlled the growth of CD19+CD1d− tumors in vivo, suggesting that it can “link” iNKT cells and CD19+CD1d− targets in a therapeutically beneficial manner. Taken together, these pre-clinical studies demonstrate that this B cell-directed fusion protein can be used to effectively induce iNKT cell anti-tumor responses in vitro and in vivo.

**Poster #40**
Cardiac myosin binding protein C mutants interact with Hsp70-family chaperones and disrupt protein quality control

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The significance of protein homeostasis (proteostasis) in cardiac pathophysiology is poorly understood. The most frequently implicated gene in hypertrophic cardiomyopathy (HCM) is cardiac myosin binding protein C (MYBPC3). >90% of MYBPC3 mutations are nonsense, but whether these mutations manifest in loss- or gain-of-function is unresolved. Evidence suggests mutant MYBPC3 impacts protein quality control. The objective of this study was to evaluate interactions of MYBPC3 with proteostatic systems and test the hypothesis that these interactions affect cardiomyocyte proteostasis.

WT and mutant MYBPC3 constructs were expressed in neonatal rat ventricular cardiomyocytes (NRVMs) via adenovirus. Mutant MYBPC3 induced ubiquitin proteasome reporter GFPu accumulation (fold increase in GFPu-positive cells vs control: WT 138±14.0%, mutant 198±27.2%, mean±SEM, p<0.05 vs control and WT), indicating proteasome dysfunction. Affinity-purification/mass spectrometry identified molecular chaperones Hsp70 and Hsc70 as prominent MYBPC3 interactors. We observed MYBPC3 degradation by cycloheximide chase in response to Hsc70 siRNA knockdown or treatment with Hsp70 activator YM-1. Hsc70 knockdown slowed MYBPC3 degradation (WT control t½ =5.47±0.70hr, WT Hsc70 knockdown t½=13.5±1.62hr; mutant control t½=3.42±0.61hr, mutant Hsc70 knockdown t½=9.87±0.95hr), while YM-1 accelerated degradation (WT DMSO t½=10.2±3.28hr, WT YM-1 t½=3.16±0.61hr; mutant DMSO t½=11.7±2.67hr, mutant YM-1 t½=1.37±1.6hr). We then evaluated whether transferrin uptake via clathrin mediated endocytosis, a critical Hsc70-dependent activity, was affected by mutant MYBPC3. Transferrin uptake was significantly decreased in NRVMs expressing mutant MYBPC3 compared to WT and untreated control (transferrin-positive cells: control 22.93±3.34%, WT 17.47±0.70%, mutant 9.30±1.63%, mean±SEM, p<0.05 vs control and WT).

In conclusion, we present evidence that Hsp70 chaperones interact with MYBPC3 in cardiomyocytes and affect MYBPC3 degradation, suggesting MYBPC3 is an Hsp70-family client. Additionally, expression of mutant MYBPC3 causes ubiquitin proteasome impairment and interferes with normal Hsc70 function. These results support our hypothesis that mutant MYBPC3 affects cardiomyocyte proteostasis.
Poster #41
Hypoxia-regulated expression of GLUT1 in GBM Cell Lines
Kane, Marissa
Northern Michigan University

Unlike normal cells, most cancers can grow in low oxygen growth (hypoxic) environments. Changes in relative oxygen concentration can alter gene expression in tumors to allow for their selective growth. The result of such changes allows the tumor to adapt its cellular metabolism and promote tumor progression. Most notably, hypoxic conditions induce expression of the transcription factor hypoxia inducible factor 1 (HIF-1). HIF-1 is thought to directly affect glucose transporter 1 (GLUT1) expression levels in hypoxic conditions. This proposed study sought to determine the relationship between HIF-1 and GLUT1 expression levels within normoxic and hypoxic environments utilizing an in-vitro GBM cell model. The data accumulated regarding HIF-1 and GLUT1 expression levels will help determine whether GLUT1 is a suitable target for future GBM treatments.

Poster #42
Is Donated Kidney A Source of Hypertension in Kidney Transplant Recipients?
Tantisattamo, Ekamol and Mopuru, Haritha
Oakland University William Beaumont School of Medicine

**Background:** The etiology of hypertension is multi-factorial and genetic kidney disease is one of the common causes. The majority of end-stage renal disease (ESRD) patients have hypertension. However, it is frequently seen that ESRD patients become normotensive at some point successful after successful kidney transplantation; however, the onset of post-transplant hypertension in kidney transplant recipients receiving different types of donated kidneys is unclear.

**Methods:** Seventy kidney transplant recipients during 1 year period were retrieved and divided into deceased donor renal transplant (DDRT), living-related renal transplant (LRRT), and living-unrelated renal transplant (LURT) recipients. Onset of post-transplant hypertension was defined as systolic blood pressure (SBP) ≥ 140 mmHg after kidney transplantation.

**Results:** Of all 70 patients, mean age±SEM was 52.66±1.43 years and 55.4% was male. The patients undergoing DDRT, LRRT, and LURT were 50 (71.4%), 12 (17.14%), and 8 (11.43%), respectively. During a 96-week follow-up, an estimated median onset of post-transplant hypertension was equal in LRRT and LURT recipients (11.14±19.8 weeks; 95% CI 0 – 19.48 for LRRT and 0 - 23.42 for LURT), but slightly shorter in DDRT recipients (10.57±6.17 weeks; 95% CI 0 – 22.67) (p-value =0.935 (log rank test, Figure1). However, 26% of DDRT and 25% of LURT recipients did not develop post-transplant hypertension; whereas, only 16.7% of LRRT recipients were normotensive (relative risk of 0.89 (DDRT vs. LRRT) and 0.62 (LURT vs. LRRT).

**Conclusion:** Although similar in the onset of post-transplant hypertension, LRRT recipients appears to develop hypertension in the highest proportion among all other types of kidney transplantation. Since both living-related donors and recipients are genetically related, factors conveyed by kidney may affect and result in hypertension in at risk patients such LRRT recipients.

![Figure1](image.png)

**Figure1:** Kaplan-Meyer curve showing time to onset of post-transplant hypertension in 3 different types of kidney transplantation. DDRT, Deceased donor renal transplantation; KTx, Kidney transplantation; LRRT, Living-related renal transplantation; LURT, Living-unrelated renal transplantation.
**Poster #43**  
**JAK inhibitors in allergy: a promising alternative for the treatment of severe asthma.**  
**Bansal, P., Kammala, A.K., Subramanian, H., Das, R.**  
**Physiology Department, Michigan State University, Michigan**

Asthma prevalence has increased considerably in recent decades and it is now considered as one of the most common chronic disorders in the world. While the current anti-asthmatic therapies are effective for most asthma patients, there are 10-30% subjects whose disease is not controlled by such agents and they account for about 50% of the healthcare costs of asthma. Such asthmatics develop severe asthma (SA), a condition characterized by a dominant Th1 cytokine response that is accompanied by a Th2 cytokine response. JAK-STAT (Janus Kinase-signal transducing activator of transcription) signaling is very important for the activation of Th1 and Th2 cytokine response. As Ruxolitinib is a JAK-STAT inhibitor, our current study aims at examining the effect of this drug treatment on SA. To induce SA in mice, animals are sensitized with house dust mite extract (HDME) and the adjuvant c-di-GMP (cyclic di-GMP). And then challenged with HDME and a lower dose of c-di-GMP. We have selected HDME to induce allergic response, as it is an important allergen that has been identified as a risk factor for persistent asthma in humans. To assess the effect of Ruxolitinib on SA, airway hyper responsiveness and lung inflammation will be assessed by flexiVent™ studies and histology, respectively. Additionally, bronchoalveolar lavage (BAL) fluid and lungs from these mice will be examined for cellular influx (by differential cell counts and flow cytometry) as well as cytokines and chemokines (by Luminex® & real-time PCR). Serum will be analyzed for total and HDME-specific IgG1 and IgE antibody titers (by ELISA). We hope that the proposed studies will highlight the JAK-STATs as novel, druggable targets for mitigating the cytokine-driven hyper-inflammation that occurs in SA and provide the framework for the incorporation of JAK inhibitors into future clinical trials for patients that have severe or difficult-to manage asthma.

**Poster #44**  
**The effects of social shuffling during puberty on pubertal cytogenesis and behaviors in adulthood.**  
**Wilks, Kristian**  
**Michigan State University**

Mice are known social creatures who thrive from social connection and experience a significant negative impact when that connection is removed. Social isolation during adolescence has been greatly studied and has been shown to negatively affect neurogenesis and promote increased anxiety and depression in mice. Social instability regarding changing social partners, however, has not been given much attention despite social interaction being a key component in development of mice. As a social environment does not naturally consist of only familiar individuals, it is important to look at how a shift in social partners affects the developing brain. This study aimed to examine how an unstable social environment during puberty regarding a constantly changing social partner may have affected pubertal cytogenesis as well as behaviors similar to anxiety and depression in adulthood. For this experiment, mice of the same age were pair-housed during puberty, with one group having a constant cage-mate while one group of mice rotated cage-mates twice per week, every three and four days, until the end of puberty. Both groups received water containing bromodeoxyuridine (BrdU) during early puberty to tag proliferating cells at the time. In adulthood, the mice were tested for anxiety-like behaviors with the light/dark box and the open field test, and potential depression-like behavior was evaluated with the sucrose preference test. The brains were then collected and the density of surviving pubertally born cells was measured in the dentate gyrus of the hippocampus and the posterodorsal medial amygdala.
Poster #45
Store operated Ca2+ mechanisms contribute to mast cell activation via MrgprX2

Occhiuto, Christopher; Subramanian, Hariharan
Michigan State University

Mast cells are tissue resident inflammatory immune cells located throughout a variety of locations in the human body. While mast cells play a prominent role in allergic inflammation, their ability to mediate non-allergic processes cannot be overlooked. Of the chemical mediators mast cells release, histamine, serotonin, eicosanoids, and proteases contribute in an imperative fashion to the allergic and non-allergic inflammation of the brain, gut, and lungs. Mast cell activation is critically regulated by the concentration of intracellular Ca2+, which is generally controlled by several Ca2+ ion channels present in the cell. One versatile receptor that mediates both allergic and non-allergic mast cell responses is the G-protein coupled receptor, MrgprX2. Though it is known that human mast cell activation via MrgprX2 leads to an increase in intracellular Ca2+ levels, the Ca2+ ion channels utilized by this receptor are largely unknown. The objective of this study was to identify the channel(s) through which MrgprX2 mediates mast cell activation. To determine channel participation, we incubated human mast cells with specific Ca2+ channel inhibitors followed by subsequent MrgprX2 stimulation. Intracellular Ca2+ flux was measured using fluorometric Ca2+ release assays. Our data demonstrates that the STIM-1 endoplasmic reticulum Ca2+ sensor and CRAC channels participate in mast cell activation via MrgprX2, while TRPV4 and TRPV1 played a limited role, if any. Thus, blocking excessive mast cell activation by employing STIM-1 and CRAC channel inhibitors could be a potential strategy in alleviating the pathogenesis of allergic and non-allergic inflammation.

Poster #46
Aging enhances atrial fibrillation inducibility in atherosclerotic hosts

Daniel J. Tyrrell, Roberto Ramos Mondragón, Guadalupe Guerrero-Serna, Héctor H. Valdivia, José Jalife, and Daniel R. Goldstein
University of Michigan

Introduction and Rationale Advanced age is the most critical factor for the development of atrial fibrillation (AF); 10% of patients in their 80s have AF and 50% of patients with AF are 80 years of age or older (Kannel WB et al). Atherosclerosis, a chronic inflammatory condition, is also associated with AF but whether this is due to a direct inflammatory alteration or indirectly via atrial remodeling and fibrosis from associated conditions such as hypertension is not known. However, the interplay between aging, atherosclerosis, and AF has not been explored. Here, we examined the effects of a high fat diet (HFD), intended to promote atherosclerosis, and aging on AF inducibility in Ldlr-/- mice.

Methods and Results After programmed electrical stimulation in the right atrium via intravenous catheterization, only 4/10 young (2-4 months of age) chow-fed Ldlr-/- mice exhibited AF, whereas 9/10 young HFD-fed Ldlr-/- mice exhibited AF (Figure 1). Interestingly, 2 of 10 young Ldlr-/- mice fed a HFD exhibited sustained AF (2 hrs), suggesting that a minority of mice fed a HFD for 2 months not only exhibit AF inducibility but sustainability. Under the same protocol, only 1/6 young chow-fed Ldlr-/- mice exhibited AF, in striking contrast to 4/6 aged (15-16 months of age) chow-fed Ldlr-/- mice that exhibited AF. Atrial cardiomyocytes from old, but not young Ldlr-/- mice, exhibited delayed after depolarizations (DADs) and increased propensity for spontaneous calcium release. We next generated bone marrow chimeras of young and aged Ldlr-/- mice by lethally irradiating mice and infusing them with aged-matched or mismatched bone marrow from Ldlr-/- donors, as reported in our prior work (Du W et al). Intriguingly, 3 of 4 young chow-fed Ldlr-/- mice that received aged bone marrow exhibited AF inducibility, whereas transplantation of young bone marrow into aged chow-fed Ldlr-/- mice reduced AF inducibility to 1/6 mice (Figure 2).

Conclusions Atherosclerosis and aging enhance AF inducibility, likely due to hematopoietic factors, in atherosclerotic-prone hosts.

References
**Poster #47**  
**Exercise-trained and long-lived *Drosophila* activate similar genetic programs**  
*Maryam Safdar, Alyson Sujkowski, Robert Arking, Robert Wessells*  
*Wayne State University*

We have developed an endurance training protocol for *Drosophila*, using an automated negative geotaxis machine called the Power Tower. Trained flies show dramatic improvements to negative geotaxis speed and endurance, as well as flight ability and cardiac performance. Similar performance improvements were also observed in a fly line selected for extended longevity, known as *La*. In order to better understand genetic regulation of longevity and health span, we used microarrays to measure transcriptional changes in both exercise-trained and longevity-selected cohorts. We hypothesized that the intersection of transcripts that changed in the same direction following both interventions would be enriched for transcripts that were required for health span and longevity benefits. We observed a large number of overlapping changes following both intervention, and focused on two groups of genes that were highly represented in the overlap, 1) Methuselah-like GPCRs and 2) Alkaline phosphatases. Single-gene representatives of these groups were tested to determine their role in exercise-induced performance improvements and in longevity. I have worked with mutants for the alkaline phosphatase that under express three genes for alkaline phosphatases as these genes were found to be overexpressed in the microarray. The hypothesis is that these flies will be unable to respond to exercise and will give results similar to unexercised wild type flies.  If accepted this hypothesis would provide evidence that alkaline phosphatase overexpression is required for exercise to mediate health benefits.

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**Poster #48**  
**Initiating an Exercise Physiology-Themed PhUn Week Presentation for 4th graders**  
*Conner J. Steffke, & Naveen Sharma*  
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Inspired by the development of PhUn Week activities at other universities across the state, and motivated to develop community outreach programs directed by exercise science students, we initiated an exercise physiology-themed PhUn week involving 2 different elementary schools in the Mount Pleasant, Michigan area. A faculty-student team contacted teachers at a large K-6 public school, and a small K-6 private school in May 2016 to confirm involvement for the November 2016 event. After consultation with the teachers regarding their 4th grade curriculum, activities were designed to incorporate the effects of exercise on physiological systems including the cardiovascular, skeletomuscular, nervous, and integumentary systems. These 4th graders were exposed to general physiology concepts, while utilizing instruments that college students routinely have access to in an exercise physiology lab. Other than content, particular consideration was placed on effectively presenting information to different-sized audiences (smaller school ~20 students, larger school ~80 students). Feedback from teachers and students at both schools was overwhelmingly positive, and we were requested to return next year. Tailoring PhUn week activities to include exercise can provide context to students on how physical activity is physiologically beneficial.
Poster #49
Assessment of cardiac function during the progression of heart failure induced by intracoronary embolizations in the canine model: monitoring in conscious and anesthetized states.

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Wayne State University

Various animal models of acute and chronic heart failure exist including coronary artery occlusion via ligation or microembolization, rapid-ventricular pacing, aortic banding, and fluid challenge. However, to our knowledge, no study has investigated whether anesthesia alters conclusions regarding hemodynamic effects associated with heart failure development. In this study, global ischemic cardiomyopathy, via repeated coronary artery microembolizations, (range: 5-15 embolization procedures per animal) was induced to lower ejection fraction to < 40% in ten chronically instrumented canines. We measured the progression of myocardial dysfunction over time, with each animal serving as its own control in conscious and anesthetized states to elucidate the effect of anesthesia on cardiovascular parameters. Hemodynamic measurements were taken in three conditions: anesthetized, immediately prior to each embolization, anesthetized 1.25 minutes after each embolization, and in a conscious, non-anesthetized state at rest, 1 week after each embolization. We observed that anesthesia lowered arterial pressure (D-31.8 ± 3.7%) and cardiac output (D-22.2 ± 3.4%) when compared with the conscious state and that the effect of the embolization on ventricular function was better revealed in conscious animals. Thus, anesthesia affects the conclusions drawn regarding the extent of cardiovascular impairment induced via the embolization model. Supported by Novartis Pharmaceuticals CRLX030AUSNC01T and NIH HL55473 and HL126706.

Poster #50
Titin-dependent Auxotonic Cardiac Relaxation

Schick, Brianna; Chung, Charles
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Isovolumic relaxation is an important phase of the cardiac cycle where the left ventricular pressure drops after systolic ejection to accommodate diastolic filling. While the volume does not change during relaxation, there is strong evidence that normal isovolumic relaxation is auxotonic, i.e. the pressure declines while there is myocardial relengthening. The goal of this study is to determine how auxotonic relaxation is dependent on the giant elastic protein titin both in vivo and in vitro. A rat model that expresses long, compliant titin isoforms due to a mutation in the RNA binding motif 20 (RBM20) protein will be used. Wild type, heterozygous and homozygous groups will be used. In vivo studies will use simultaneous invasive left ventricular pressure measurement and ultrasound speckle tracking strain measurement. Data will be aligned and plotted to create pressure-strain relationships and determine the magnitude of auxotonic relaxation and the dependence of the pressure relaxation rate on myocardial strain or strain rate. In vitro studies will use free-standing trabeculae isolated from the ventricular walls, mounted between a force transducer and length motor. A feed-back control protocol will allow the muscle to mimic ejection. Subsequently, the velocity of the muscle will be controlled during relaxation. The relaxation rate of the force will be compared to the velocity of the muscle. Preliminary results suggest that the auxotonic relaxation rate is less sensitive to the velocity of the muscle in trabeculae with more compliant titin. Titin based stiffness may be an important modifier of myocardial relaxation.
Poster # 51
Beta Cell ER Homeostasis
Cesar Barrabi, Xuequn Chen
Wayne State University

Rough endoplasmic Reticulum (ER) homeostasis is a key component to proinsulin folding, insulin production and beta cell survival. ER stress can be induced by many stimuli that can interfere with proper insulin folding, and utilization of insulin. Misfolded proinsulin caused by ER stress contributes to Type 2 Diabetes (T2D), which underlines the significance of studying ER stress in the pancreatic beta cells. To date most studies focus on changes in mRNA transcripts during ER stress, however focusing on actual protein changes via Western blotting is a more accurate way to capture changes in protein expression in the beta cell ER. A combination of ER stress inducers was used on either MIN6 or INS-1 cells to analyze their effects on protein changes. Proteins that change expression level during ER stress may indicate that they play an important role in causing the stress, or potentially play a role in reversing stress. Identifying proteins that change expression levels during stress can be indicative of further routes of study, as well as potential targets for pharmacological intervention for attenuating the effects of ER stress during T2D. Once key protein changes have been distinguished, knockout methods can be used to identify specific functions of a given ER protein. Protein interaction studies can also be used to identify altered binding partners under normal and ER stress conditions. Models of inducing ER stress for these studies have been analyzed on both MIN6 and INS-1 cells for these studies using changes in insulin expression providing evidence confirming ER stress.

Poster #52
Gender Differences in Auditory Response Time
Sosnowski, Benjamin, Speirs, Sue
Grosse Pointe North High School

A previous study states that males have faster auditory response time (ART) than females (Jain A, et. al., 2015). In the discussion section of this study, the authors suggest that this gap in gender ART might be closing due to women's increased participation in fast-action sports (Jain A, et. al., 2015). We hypothesized that this gap is now closed and that the difference in gender ART will not be significant. Seven females and four male participants were studied. Reaction time was measured using two sensors, an accelerometer attached to a patellar hammer and an electromyograph (EMG) sensor. The EMG sensor includes three electrodes that pick up the electrical signals when muscles contract. Specifically, our study examined the right leg quadriceps femoris contraction for each subject in response to the sound of the patellar hammer striking the lab table. Statistical analysis was completed using a t-test (reported mean±SEM). Average ART for females was 0.473±0.0553, n=35. Average ART for males was 0.543±0.0486 n=20. There was no statistical difference between the male and female mean ARTs (0.473±0.0553 vs 0.543±0.0486, p=0.40). Height differences were accounted for with no significant difference females 1.70±0.035 vs males 1.73±0.048, p=0.76. These data supports the hypothesis that females are closing the ART gender gap. This result may be due to increased activity in female lifestyle impacting these neural pathways. Athletes of both genders can improve reaction time through continuous exercise with the muscles involved in the action. For example, those in track can improve starting times through continuous use of the quadriceps muscles. With fast action exercise and training, reaction times improve.
Poster #53
Elmer, Steven; Bye, Thomas; Hudak, Kirsen; Gabe, Alex; Carter, Kathynn
Michigan Tech University

Foundational knowledge of physics is important when learning physiology and medicine. National Biomechanics Day (NBD) is a new international outreach initiative aimed at building relationships with local high schools to promote interest in biomechanics (i.e., physics of the human body). The long term goal of NBD is to foster the development of biomechanics as a standard component in high school science curricula. The Department of Kinesiology and Integrative Physiology at Michigan Technological University organized its first NBD event. We designed a hands-on activity inspired from Dr. Carrier’s research in the Journal of Experimental Biology to emphasize how the laws of physics are used to understand human and animal turning. Turning is important because it accounts for up to 50% of walking steps during the day and is needed for survival when animals elude predators and capture prey. For this activity, undergraduate exercise science students directed high school students from physics and anatomy/physiology classes to work in small teams. Each team used 2 x 4 lumber, wood screws, and a drill to build a wooden apparatus, that when connected to the body, artificially increased rotational inertia (an object’s ability to resist angular motion). Students navigated through a slalom course with and without the wooden apparatus. Times to complete the course were compared between trials to determine the influence of rotational inertia on turning performance. Students compiled their data, graphed their results, and found that increased rotational inertia decreased turning performance by ~20%. Results were connected to sports, rehabilitation, and dinosaur evolution. Our first NBD event was well received and impacted over 70 students from two local high schools. Inclusion of undergraduate students in NBD improved their understanding of biomechanics, provided practice communicating and teaching science to a public audience, and helped establish relationships with local high schools in our rural community.

Poster #54
Reflexes for A and B personality types
Hedman, Casey; Haggerty, Erin
Grosse Pointe North High School

In previous studies, different personality types have been associated with different behaviors. Specifically, in cardiologists Meyer Friedman and RH Rosenman’s study it was found that type-A individuals “tended to sit on the edge of the seat and leaped up frequently.” This difference in behavior in accordance with personality type inspired us to dig deeper into personality differences and their reflexes. Personalities are split into mainly two different categories known as type-A and type-B. Type-A personalities are more aggressive and anal. Type-B personalities are lackadaisical and rag tag. The purpose of this experiment is to explore the impact of Type-A and Type-B personalities on reflex time and reflex muscle amplitude response. We hypothesize that subjects with a type-A personality will have a faster reflex and a larger amplitude during the quadriceps femoris reflex contraction than type B subjects. We tested three type-A subjects and three type-B subjects and recorded five trials for each (n=15). Statistical analysis was completed using a t-test (reported mean±SEM). Reflex times for Type-A 0.029±0.008 vs Type-B 0.10±0.016, p<0.003. The reflex times for type A individuals are faster on average, than the reflexes of type B. The amplitude for Type-A’s quadriceps femoris reflex contraction 2.58±0.14 v Type-B 0.72±0.16, p<0.001. Type A subjects had a higher voltage response than Type B subjects.individuals (p<0.003). This difference in reflex time and amplitude for quadriceps femoris reflex contraction supports the hypothesis that there is a difference in type A and type B personalities.
Poster #55
Blueberries Protect Pancreatic Beta Cell Function
Liu, Weixang; Schoenborn, Jacob; Tang, Xiaoqing
Michigan Technological University

The statement “the most powerful drugs for your body are the foods and beverages you eat and drink everyday” underlines the importance and influence nutrition has on our health. Evidence has demonstrated that an imbalanced diet is a major contributor for obesity and consequently type 2 diabetes mellitus. This makes it important to determine which diet can protect the pancreas against the onset of diabetes. One such food is the blueberry.

Blueberries contain antioxidants which have been shown to protect against oxidant-induced cell damage. In addition, blueberries are full of bioactive substances that help improve insulin sensitivity in muscle and adipose tissue. However, whether blueberries protects pancreatic β-cell function and growth has not been fully evaluated. Pancreatic β-cells are required to maintain normal blood glucose levels by storage and release of insulin.

To investigate blueberry’s effect on beta cell function, a modified high-fat diet supplemented with 4% freeze-dried blueberry powder (HFD+B) was fed to the mice. This was then compared to mice fed with a standard high-fat diet (HFD). The addition of blueberries had no significant change in the body weight and glucose level, but, after 8 weeks feeding, the plasma insulin level was decreased significantly in mice fed with HFD+B. In addition, mice fed with the HFD+B had an increased glucose tolerance and were more sensitive to insulin. The blueberry-supplemented diet also prevented the HFD-induced β-cell expansion and preserved the islet structure within the pancreas. When all of this is taken together, our results indicated that the blueberry diet could protect β-cells, restore impaired glucose homeostasis triggered by HFD, and therefore slow the development of type 2 diabetes. This data will provide new insights into the effects of blueberries on β-cell function and the importance of a balanced diet in treating and preventing diabetes.

Poster #56
Height Impacts Reflex Reaction Time
Doherty, Melina; Rafail, Christina; Castronero, Carmen
Grosse Pointe North High School

Height is something that one cannot control and is predetermined by many biological factors. In our study, we examined how two significantly different heights, tall n=2 1.89±0.03 vs short n=2 1.65±0.03 p < 0.0001, using a t-test (reported mean±SEM), impact patellar reflex and quadriceps femoris reaction speed. The purpose of our project is to determine the relationship between height and its effect on the responses. We hypothesize that the shorter subject group will be faster in both patellar reflex and quadriceps femoris reaction speed. We reasoned that the distance the action potential travels along the neural pathways is shorter for a short person compared to a tall person. In order to test our hypothesis, we measured the two tallest (one male and one female) and two shortest (one male and one female) people in our class. We attached the electromyograph (EMG) electrodes to measure patellar reflex and the auditory response time of kicking the leg when the hammer struck the table five times for each subject. Statistical analysis was completed using a one-way ANOVA (reported mean±SEM). Our data shows tall reflex 0.089±0.02 vs short reflex 0.018±0.003 p<0.05 and tall reaction 0.71±0.03 vs short reaction 0.31±0.01 p<0.01. We accept our hypothesis that a person’s height impacts both patellar reflex speed and quadriceps femoris reaction speed.
Poster #57
Using Anisotropic Ultrasonic Backscatter to Determine Cardiac Fiber Maps in Small Animals
Alkhazal, Thamer; Milne, Michelle; Chung, Charles
Wayne State University

Myocardial fiber architecture is important to generate large left ventricular ejection fractions and high pressures during systole. Prior research anisotropic behavior of acoustic (ultrasonic) backscatter (intensity) can be used to create 3D fiber maps in human-sized mammalian hearts. The goal of our study was to determine if ultrasound-based acoustic fiber mapping is viable in small animal models such as rats. Excised normal rat hearts were fixed using Langendorff perfusion of fixative (2% glutaraldehyde). The anisotropic behavior of acoustic backscatter was determined from these fixed hearts in two ways: First, 3 mm diameter core segments were isolated from the left ventricular free wall. The core segments were mounted and rotated 360 degrees in 5 degree increments while being imaged using a high-frequency ultrasound probe (21 MHz, VisualSonics Vevo2100) to determine the angle dependence of acoustic backscatter. Second, trapezoidal wedges were isolated from the left ventricular free wall and imaged from epicardium to endocardium, parallel to the epicardium, in 100um steps. The segment was then embedded in optimal cutting temperature compound and sectioned in 50 um steps using a cryotome. The acoustic backscatter and histologically determined myocardial fiber angle direction were compared for matching slices. We found that the anisotropic behavior of acoustic backscatter was consistent in the samples, suggesting that the ultrasound-based fiber mapping is viable in small animal models. 3D mapping of whole rat and mouse hearts is in progress.

Poster #58
Music Impacts Amplitude, Not Reflex Time
Badih, Aiyana; Sonaglia, Victoria; Lemanske, Elizabeth
Grosse Pointe North High School

People listen to music for a variety of reasons: to relax, to gain a favorable mindset for a sport or to fall asleep. In our study we are interested in examining the impact of listening to music on the human reflex. We are curious to learn if the Jendrassik Maneuver (JM), a method used to increase the sensitivity of a tendon when testing the reflex, might play a role in decreases inhibitions from the brain to the reflex arc when listening to music. We hypothesize the patellar reflex time will not be impacted by music regardless of tempo or genre and that the amplitude of the contracted quadriceps femoris during the patellar reflex will increase in response to music. To measure if reflex time was affected by an auditory stimulus, three subjects’ reflex muscle action and the amplitude of the quadriceps femoris contraction were measured five times each. Statistical analysis was completed using a one-way ANOVA (reported mean±SEM). The reflex times for silence 0.031±0.002 vs classical 0.03±0.002 vs rap 0.03±0.002 vs favorite 0.032±0.002 showed no significance. The amplitude of the quadriceps femoris contraction during reflex for silence 1.71±0.35 vs classical 0.9±0.14 vs rap 3.35±0.2 is significant p<0.05. The innateness of a reflex can prove useful in situations that can pose threats to our safety because reflexes are not impacted by outside auditory distractions. In contrast, when taking in account the amplitude of the quadriceps muscle contraction during reflex, auditory distractions impacted these data. What is interesting is that each subject the classical music amplitude was less than the amplitude of the muscle contraction when reflex was measured while sitting in silence.
The Two Hour Marathon: What do Students Think?
Steven Elmer, Ian Greenlund, Michael Joyner, Jason Carter
Michigan Technological University, Department of Kinesiology and Integrative Physiology

For over 100 years, athletes, coaches, and scientists have endeavored to improve running performance. The marathon world record is 2:02:57, requiring a 2.4% improvement to achieve a sub-two hour marathon. Joyner (1991) used the main determinants of running performance (VO2max, lactate threshold, running economy) to model that a runner could theoretically run a 1:57:58 marathon. Twenty years later, Joyner and colleagues (2011) revisited this concept and asked the question - The two hour marathon: who and when? This question sparked enthusiasm as physiologists from around the world provided commentary, and industry lead initiatives set a new best marathon time (2:00:25). Given the widespread interest and uncertainty surrounding the two hour marathon, we used this as an opportunity to engage our students in the discussion, and challenged them to connect physiology to current events. Using problem- and project-based learning methods, exercise physiology students (n=48) explored what it would take to achieve the first sub-two hour marathon. Foundational content was off-loaded to video lectures to allow more class time for active learning. Undergraduate students completed a multi-day unit in which they: discussed the Joyner et al. (2011) and follow-up commentary, collaborated in groups to propose additional factors that would impact marathon performance, and presented their ideas to the class. Graduate students expanded upon this through a multi-week unit in which they discussed additional review papers, determined their own running economy, and presented their ideas at a department seminar. Students identified factors ranging from genetics, running economy, drafting, surface material, training volume/intensity, and psychology. Finally, students presented their ideas to Dr. Joyner through Skype and learned about the physiology and history of running. This two hour marathon activity required students to ask and refine questions, debate, make predictions, conduct experiments, analyze data, and communicate their findings to peers and experts in the field.

Dog Age and Auditory Response Time
Dean, Alex; Yerramalli, Gowri; Fazekas, Nicole
Grosse Pointe North High School

A dog’s age can significantly affect its physical performance. As with humans, dogs also present symptoms of aging--loss of hearing and vision, and decreased mobility. According to Fleming, J.M., Creevy, K.E. and Promislow, D.E.L. (2011), the frequency of degenerative deaths is most common in dogs between 8 and 14 years of age. This led us to examine for a correlation between age and auditory response time in dogs of varying ages, (7 years to 11 years, n=3). We hypothesize that older dogs will have slower auditory response time than younger dogs. As a part of our procedure, we measured the auditory response time of the dogs by recording the time between an auditory command and the dog's first physical response. Statistical analysis was completed using a one-way ANOVA (reported mean±SEM). Average ART for Lab/Pointer (7 yrs) 0.27±0.03 vs Havanese (11 yrs) 1.08 +0.15 P<.05, Lab/Pointer (7 yrs) 0.27+0.03 vs Pug (10 yrs) 1.53 +0.2 P<.01, and Havanese (11 yrs) 1.08 +0.15 vs Pug (10 yrs) 1.53 +0.2 nonsignificant. Our study revealed that age had a positive correlation with auditory response time. There was a significant difference between the means of the ten and eleven year old dogs and the 7 year old dog; and no significant difference between the means of the two older dogs. From this data, we concluded that as the dog’s age increased, the auditory response time increased. Many factors may be at work like slowed neurological response, muscle weakness and atrophy causing the older dogs to respond as evidenced in the data.
**Poster #62**

**Distracted Driving Lengthens Reaction Time**

Levick, Steven; Leone, Francesca; Mourad, Rachel

Grosse Pointe North High School

According to the National Highway Traffic Safety Administration, “during daylight hours, approximately 660,000 drivers are using cellphones while driving. That creates enormous potential for deaths and injuries on U.S. roads. Teens were the largest age group reported as distracted at the time of fatal crashes.” Anything pulling the attention of the driver away from the task of driving is a distraction and can ultimately result in car crashes due to a delayed response of the driver. Our study will explore the impact of distractions on three subjects’ reflex and voluntary reaction rate. We hypothesize that distractions will increase voluntary reaction rate and have no observable impact on reflex rate. Subjects’ reflexes and voluntary responses are measured in two trials, one while sitting in a silent room performing the driving app test (DAP) and the other sitting in the distraction room. DAP scores are analyzed using a t-test (reported mean±SEM), DAP-silence 447.5±73.7 vs DAP-distracted 163.3±46.7 p<0.01 Reaction-reflex times are analyzed using one-way ANOVA (reported mean±SEM), Silence-Reflex 0.05±0.01 vs Distracted-Reflex 0.06±0.01 vs Silence-Reaction 0.3±0.03 vs Distracted-Reaction 0.5±0.04 p<0.01. With distractions, the DAP scores are more than 2x reduced due to the number of car crashes recorded during the test. Reaction times are nearly doubled in length when drivers were distracted. We conclude that being distracted while driving lengthens reaction and increases the number of accidents as evidenced by the DAP scores. Reflex times were not impacted in this study. These are significant findings given the daily impact that distractions have on drivers of all ages, especially teenagers. The results of this study can be used to inform drivers that by removing distraction, we can reduce car accidents as a whole.

**Poster #63**

**Use of a Course-based Service Learning Assignment to Increase Understanding of Physiology in Local Schools**

Bye, Thomas; Carter, Kathryn; Elmer, Steven; Carter, Jason

Michigan Tech University

Physiology Understanding Week (“PhUn”) is a national outreach program aimed at building connections between scientists and their local schools. Inclusion of undergraduate students in outreach activities improves their understanding of physiology and also helps to better impact K-12 students. The Department of Kinesiology and Integrative Physiology at Michigan Tech University organized its first PhUn Week event. Our objective was to implement an undergraduate course-based service learning assignment to help foster partnerships with local schools in our rural community and increase K-12 student interest in physiology. To accomplish this, an instructor embedded a PhUn Week service-learning assignment into his introductory Exercise Science course. The instructor brought in a PhUn week consultant, Dr. Erica Wehrwein, to speak to his class to promote enthusiasm for service learning and community outreach. Undergraduate students (n = 19) formed teams of 3-4, selected a physiology topic, and met with the department outreach coordinator for assistance in developing age-appropriate, hands-on activities. Undergraduate students then reached out to six local K-12 teachers to plan activities. Before going to schools teams met to practice delivery and finalize plans. Our PhUn Week event was well received and impacted 151 total students in grades 2-12. Activities introduced to K-12 students were related to the heart, stress, physical activity, and injury prevention. Undergraduate students gained service learning skills and found value in presenting physiology to a public audience. With a team approach (instructor, consultant, outreach coordinator, undergraduate students) the department established relationships and increased understanding of physiology in local schools.
**Poster #64**  
**Characterization of the in vitro glycation of insulin, insulin receptor, and insulin-like growth factor-1**  
*Turkette, Thomas; Rhinesmith, Tyler; Root-Bernstein, Robert*  
*Michigan State University*

Glycation (non-enzymatic glycosylation) is the stochastic addition of a molecule of sugar to an amino group, usually of a protein. This reaction has previously been shown to alter the functionality of proteins such as insulin. Insulin is highly homologous to several proteins including portions of its own receptor (IR) and insulin-like growth factor-1 (IGF-1). In this study, we investigated the sites of glycation on IGF-1, as well as the rate of glycation. IGF-1, insulin, and fragments of IR were glycated for 1, 3, and 6 days in 20mM, 60mM, or 200mM glucose. The extent of glycation was then quantified using MALDI-ToF mass spectrophotometry. Glycation was measurable for all time points and concentrations, with approximately 16% of IGF-1, insulin, and IR105-118 being glycated by day 6. Interestingly, IR897-916 displayed a much higher rate of glycation (33% at day 6). The site of glycation for IGF-1 was then determined using LC/MS/MS. Glycation of IGF-1 was found to occur exclusively on the N-terminal glycine residue for all glucose concentrations and time points. The relatively rapid glycation of these proteins could indicate a role in the development of insulin/IGF-1 resistance and the development of subsequent complications.

**Poster #65**  
**Berberine hydrochloride improves insulin resistance induced by cytokines in C2C12 myoblast cells**  
*Anil Poudel, DVM, PhD and Lixin Li, MD, PhD*  
*Physician Assistant Program, College of Health Professions, Central Michigan University, Mt. Pleasant, Michigan 48859*

Berberine hydrochloride (BBH) is an isoquinoline alkaloid that has been used successfully to treat type 2 diabetes in oriental medicine. However, the mechanism by which BBH’s antidiabetic effect remains largely elusive. To this end, we investigated the mechanism by which BBH prevents development of cytokine mediated insulin resistance in skeletal muscle. We treated C2C12 myoblasts for 24 hours with pro-inflammatory cytokines, interleukin-1β (IL-1β; 10ng/mL) alone or in combination with tumor necrosis factor-alpha (TNF-α; 50 ng/mL) in presence or absence of BBH (10 µM). We observed elevation in iNOS, NF-κB, phospho-SAP/JNK, phospho-c-JUN and SMAD4 proteins induced by chronic exposure to IL-1β alone or in combination with TNFα (p<0.005). These proteins are key regulators of inflammatory pathway and play critical role in initiation of and sustained insulin resistance. Presence of BBH prevented increase in their activity (p<0.005). In addition, BBH treatment upregulated protein level of catalase (p<0.005), an antioxidant enzyme. Our western blot analysis also revealed that IL-1β alone or in combination with TNF-alpha reduces SIRT1, PGC-1α, phosphor-AMPKα, UCP-2, PPARα and NRF2 protein expression in skeletal muscle. SIRT1, PGC-1α and AMPKα are co-activators of mitochondrial biogenesis and play critical roles in mitochondrial metabolism along with PPARα. PPARα is also an important anti-inflammatory mediator and inhibits the release of pro-inflammatory enzymes such as iNOS. Nrf2 is a major regulator of oxidative stress defense. UCP-2, a major uncoupling protein in skeletal muscle has been shown to couple glucose oxidation to mitochondrial metabolism. Reduction in activity of these proteins were recovered by BBH treatment (p<0.005) Finally, BBH also improved activity of IRS1, AKT and GLUT4; proteins involved in regulation of insulin signaling pathways, impaired by cytokines. Together, our data suggest that BBH protects C2C12 myoblasts against cytokine mediated insulin resistance through multiple pathways including downregulation of inflammatory pathway, improvement in insulin signaling and promotion of mitochondrial biogenesis.
The development of respiratory muscle fatigue compromises lower-body exercise at one’s limit of tolerance especially in endurance athletes. It is unclear if respiratory muscle fatigue has a similar effect on upper-body exercise tolerance where respiratory muscles have respiratory, postural, and locomotor roles. We evaluated the effects of inspiratory muscle fatigue on upper-body exercise tolerance. We hypothesized that high-intensity arm-cranking performed by upper-body endurance-trained athletes would induce considerable ventilatory stress causing a significant reduction in upper-body exercise tolerance. Six athletes (23±5yrs, 185±8cm, 78±6kg, upper-body VO2peak and Wattpeak 49±6mL/kg/min, 196±27W, respectively) performed two upper-body exercise trials. For the control trial, athletes performed arm-cranking at ~85% of Wattpeak (180±20W) until their limit of tolerance (Tlim). For the fatigued trial, athletes performed the same task while having a pre-existing level of fatigue in their inspiratory muscles (~20% reduction in maximal inspiratory pressure (MIP)). Tlim and physiological responses were compared between trials. During the control trial, ventilation rates were high (137±18L/min) and after exercise MIP decreased by 14±4% (160±30 vs. 137±29cmH2O) and remained reduced for 8min (P<0.05). Compared to control, fatigued trial Tlim decreased by 27±10% (16.4±7.3 vs. 12.2±6.2min, P<0.01). During the fatigued trial, ventilation and breathing frequency increased whereas tidal volume decreased (all P<0.05). Perceived exertion and dyspnea were elevated in the fatigued trial (P<0.05). For these athletes, high-intensity arm-cranking substantially stressed their respiratory muscles. Possible contributions to the reduced upper-body exercise tolerance include competition for blood between respiratory and locomotor muscles and/or the respiratory metaboreflex. Our findings of increased ventilatory stress and reduced upper-body exercise tolerance in athletes who regularly perform upper-body endurance exercise shed light on previous upper-body research using untrained individuals and support lower-body research using endurance athletes. These findings have implications for athletes performing upper-body exercise (i.e., cross-country skiers, kayakers, sailors) and investigators who use arm-cranking as a research model.
UNDERGRADUATE STUDENTS TO PARTICIPATE IN THE INAUGURAL MICHIGAN PHYSIOLOGY QUIZ (MiPQ)

On June 7, 2017 immediately before the 4th Annual Michigan Physiological Society (MPS) Meeting in Alma College, teams of undergraduate students from six Michigan institutions (listed below) will compete in an oral quiz event to determine which team shows the best proficiency in physiology. Sue Barman (Michigan State) will serve as Quizmaster for the inaugural Michigan Physiology Quiz (MiPQ, “my quiz”). The winning team members will be recognized as the Warren Plimpton Lombard Award recipients. Dr. Lombard was one of the first professors of physiology in the state of Michigan (University of Michigan, 1892 – 1923) and the 8th President of the American Physiological Society (APS) (http://www.the-aps.org/fm/presidents/introwpl.html).

The MiPQ is modeled after an international science Olympiad created by Dr. Hwee-Ming Cheng at the University of Malaysia called the Inter-Medical School Physiology Quiz (IMSPQ). Sue Barman had the honor of attending as an instructor for a refresher course and a quiz judge the 14th annual IMSPQ competition held last summer in Indonesia (Barman SM. Physiology Quiz Show. The Physiologist 59: 347-348, 2016.). This event included approximately 350 students representing 81 medical schools. Sue was overwhelmed by the enthusiasm of the students and proposed to the APS Council that we begin a comparable event in the US with the goal of eventually sending a US team to participate in the IMSPQ. APS Council encouraged the MPS to be the first APS Chapter to include this event as part of their annual meeting. The hope is that other chapters will do the same and then the US may set up a competition among chapter winners to send them to IMSPQ in some future year.

Members of the 1st, 2nd, and 3rd place teams will receive certificates recognizing their accomplishments. Thanks to the generous contributions of some MPS members (see list below), they will also receive a monetary award. All students participating in the MiPQ will receive a copy of the 25th edition of Ganong's Review of Medical Physiology compliments of McGraw-Hill.

Congratulations to all the participants who for the past several weeks have been busy preparing for this event and refreshing their knowledge of all things physiology.

Ferris State
Amanda Gilliam
Hunter Pope
Catherine Mirto
Austin Vanwyk
Faculty Coach: Chris Westerkamp, PhD

Michigan State University
Chris Davis
Savanna Cureton
Katie Grover
Ella Potter
Faculty Coach: Erica Wehrwein, PhD
**Michigan Technological University**
Tom Bye
Hannah Marti
Ross Michaels
Mikayla Revoyr
Faculty Coach: John Durocher, PhD

**Oakland University**
Alexander Bageris
Joseph Bires
Joshua Hohlbein
Bernard LePaige
Zachary Walker
Faculty Coach: Amy Banes-Berceli, PhD

**Wayne State University**
Cristina Al-Jageta
Patrick Etta
Maryam Safdar
Brianna Schick
Sangini Tolia
Faculty Coach: Mohamad El Chami; Pat Mueller, PhD

**DONORS FOR THE MiPQ EVENT**
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Susan M Barman
Jason Carter
Greg Fink
Linda Samuelson
Erica Wehrwein
John Williams
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Erin Haggerty
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