THE 31ST ANNUAL UNIVERSITY OF ARIZONA

UNDERGRADUATE BIOLOGY RESEARCH PROGRAM CONFERENCE

January 25, 2020

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WELCOME

Dear Guests:

Welcome to the 31st Annual Undergraduate Biology Research Program (UBRP) Conference! It is a privilege to see our community come together to celebrate the accomplishments of our program participants at this event.

Whether students aim to enter the fields of research, health care, education, business, or law in the future, through UBRP they gain research experience first-hand, and develop the skills necessary for critical thinking, analysis, and the process of developing new knowledge.

Being a student scientist is no easy task. Our participants have journeyed down the path of scientific inquiry through hours of hard work and all that comes with it: frustration, challenges, and roadblocks, as well as the joy of discovery, "eureka" moments, and the genuine satisfaction that comes with a successful experiment. Many people have played a critical role in this process and in shaping our students' experiences, and for that, we extend our gratitude to:

- Our faculty, post-docs, graduate students, and other colleagues who have mentored, challenged, and trained our students.

- UA administrators, deans, and department heads who understand the importance of undergraduate research experiences and have provided support for students to maximize their education through UBRP.

- Fellow students, friends, and family members, who have provided moral support and encouragement.

- Community members, who are eager to learn more about the scientific work students do and how it impacts, and hopefully improves, the world we share.

You all have played an important part in the lives of the young scientists presenting today. I encourage you to let your curiosity reign as you explore the wide variety of topics presented at today's conference. What are the mechanisms behind Alzheimer's Disease and other neurodegenerative diseases? How do language development and disorders in young children arise? How do migraines occur? How can microbes be used to get rid of invasive buffelgrass in the environment? How do bumblebees make decisions? How is the retina involved in establishing circadian rhythm? How do wildlife populations change in response to fire and other events? How can chronic pain be studied? These questions represent just a few of the exciting projects on which our students are working.

At this start of this new decade, I hope you are encouraged as I am by the capabilities of our amazing young scientists. Thank you for coming, and I hope you enjoy today's conference.

Sincerely,
Jennifer Cubeta
UBRP Director

CONFERENCE AGENDA

ENVIRONMENT & NATURAL RESOURCES 2 BUILDING, 1064 E. LOWELL STREET

9:00am – 10:00am CHECK IN

GROUND AND SECOND FLOORS

- Check in at the registration table
- Preview student posters (second floor)
- Meet & network with UBRP students, faculty mentors, alumni, and guests

10:00am – 11:00am KEYNOTE ADDRESS

ROOM N120, GROUND FLOOR

- Welcome by Jennifer Cubeta, UBRP Director
- Introduction of Keynote Speaker by Dr. William Dantzler
- Keynote Address: "Disparities in Healthcare: Why Your Inclusion in this Discussion is Critical for the Wellbeing of Your Community" by Dr. Oscar Serrano, UBRP Alumnus, Abdominal Transplant & Hepatobiliary Surgeon at Hartford Hospital, and Assistant Professor of Surgery at the University of Connecticut School of Medicine
- Acknowledgement of Donors and Special Opportunity to Support Student Conference Travel by Dr. John Szivek, UBRP Advisory Board Chairman
- Poster Session Logistics by Marisa Lester, UBRP Assistant Director

11:00am – 2:00pm POSTER SESSIONS & ACTIVITIES

POSTER SESSION - ROOMS S215, S223, S225, & S230 ON SECOND FLOOR

- Odd numbered posters present from 11:00am – 12:30pm
- Even numbered posters present from 12:30pm – 2:00pm

SCIENCE ACTIVITIES - GROUND FLOOR

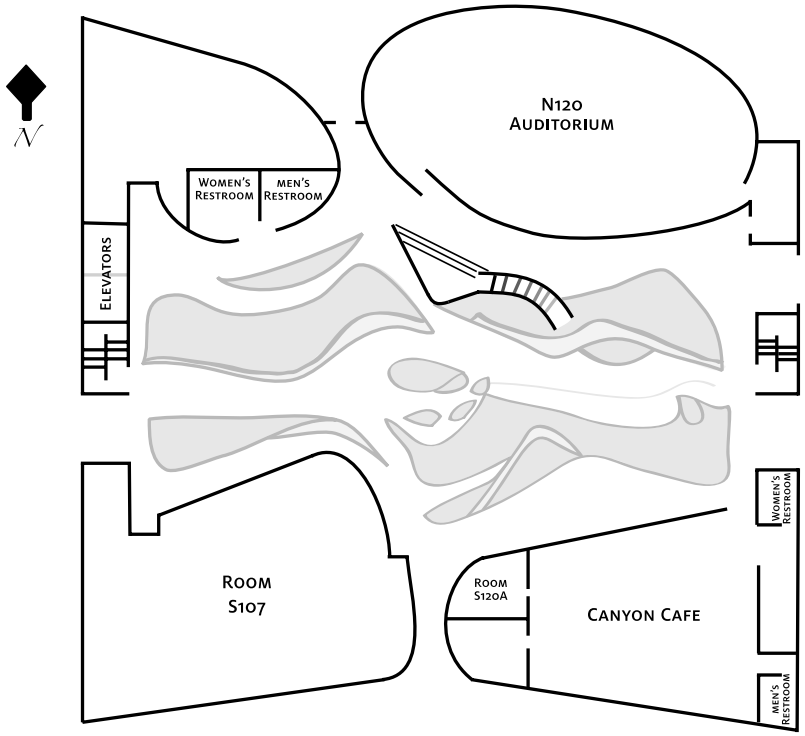
- Symbiosis: An Exhibit of Biological Art – Room S107
- Arthropod Diversity – Courtyard
- Science in Color – Courtyard
- Marine Awareness & Conservation Society – Courtyard

2:00pm – 3:00pm CLOSING REMARKS, AWARDS, & DOOR PRIZES

ROOM N120, GROUND FLOOR

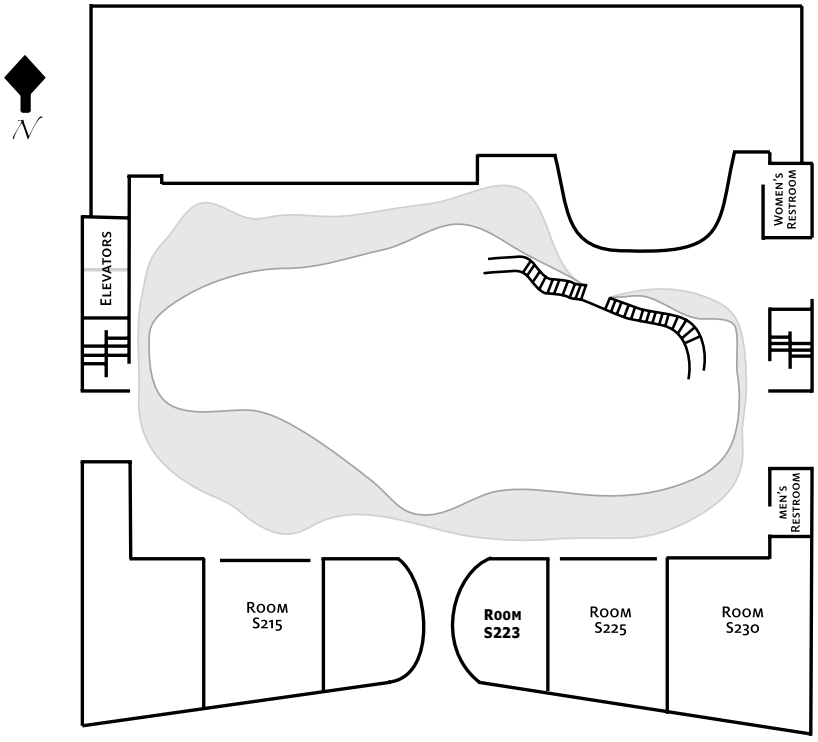
- Outstanding Graduate Student Mentor Award
- Outstanding Faculty Mentor Award
- Recognition of Graduating Seniors
- Recognition of UBRP Conference Poster Presenters
- Door Prizes

MAP OF VENUE:
ENVIRONMENT AND NATURAL RESOURCES 2 BUILDING



GROUND FLOOR

SECOND FLOOR



TODAY'S ACTIVITIES

10:00am – 11:00am ◆ KEYNOTE ADDRESS

"Disparities in Healthcare: Why Your Inclusion in this Discussion is Critical for the Wellbeing of Your Community" by Oscar Serrano, MD

UBRP Alumnus, Abdominal Transplant & Hepatobiliary Surgeon at Hartford Hospital,
and Assistant Professor of Surgery at the University of Connecticut School of Medicine

INTRODUCTION OF KEYNOTE SPEAKER BY
DR. WILLIAM DANTZLER, PROFESSOR EMERITUS, DEPARTMENT OF PHYSIOLOGY

Dr. Oscar K. Serrano is a proud alumnus of the UBRP and BRAVO! programs. An immigrant of Mexico, he spent his teenage years in the rural mining town of Globe, Arizona. In 1996, he enrolled at the University of Arizona, which fueled his passion for scientific inquiry and medicine. He credits Marc Tischler, Bill Dantzler, and Carol Bender for their guidance and mentorship, along with countless other UA faculty who nurtured his passion and ambitions. During his last year at the UA, Dr. Serrano spent a semester abroad with the BRAVO! Program at the University of Wurzburg in Germany working with Drs. Michael Gekle and Stefan Silbernagl on renal ion transplant in the mammalian kidney. Upon his return from Germany, his thirst for international scientific work and collaboration flourished and he decided to defer matriculation to medical school to complete a year abroad with BRAVO! and the Howard Hughes Medical Institute at the University of Florence in Italy working with Giancarlo Pepeu and Renato Corradetti on electrophysiology of the rat hippocampus. This experience would evolve into one of the most transformative and enjoyable experiences of his life, and one which he attributes to the work and vision of Carol Bender.

Upon his return from Italy, Dr. Serrano enrolled at the Stanford University School of Medicine in 2001. During his tenure at Stanford, he focused his research on cancer immunology and immunotherapy. He deferred graduation to spend a year at the National Institutes of Health in Bethesda, Maryland. He began his General Surgery residency in 2006 at the Johns Hopkins Hospital in Baltimore, Maryland, where he met his wife, Kate. After 2 years, he returned to NIH to complete a two-year Intramural Research Training Grant studying cancer pharmacology and metabolism. In 2010, he earned a Master's in Business Administration from the Johns Hopkins University Carey Business School. He completed his General Surgery residency at the Albert Einstein College of Medicine in NYC where his first child, Sophie Claire, was born in 2015. After residency, he completed a two-year multiorgan abdominal transplant fellowship at the University of Minnesota in Minneapolis, where his second child, Connor Javier, was born in 2017.

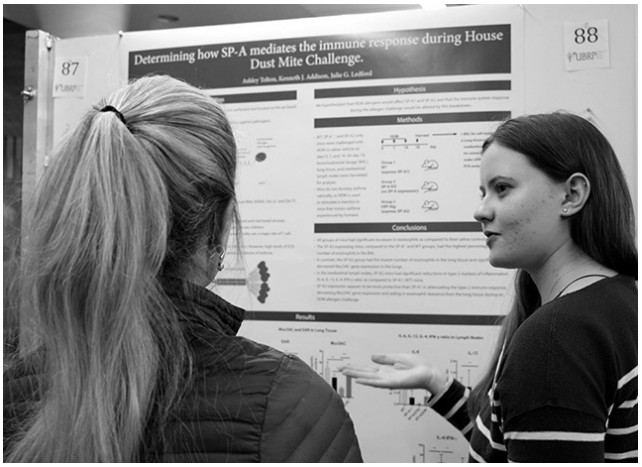
Today, Dr. Serrano is an abdominal transplant and hepatobiliary surgeon at Hartford Hospital and an Assistant Professor of Surgery at the University of Connecticut School of Medicine in Hartford, Connecticut. His primary clinical interests include kidney and liver transplantation, pediatric transplantation, and gastrointestinal cancer surgery. His research focuses on finding causes for disparities in access to care in transplantation and cancer care, specifically for Hispanic patients. He has

published more than 55 peer-reviewed articles and book chapters and has traveled all over the world to present his research. Despite having traveled extensively and worked at many different institutions, he believes there is no place quite like the University of Arizona. He remains a proud Wildcat!

11:00am – 2:00pm ◆ POSTER SESSIONS AND ACTIVITIES

STUDENT POSTER SESSIONS

SECOND FLOOR



Our students are proud to share their work with you! We invite our visitors to be curious and to ask questions such as "*Can you walk me through your poster? How did you get involved in research? What excites you about doing research? What is the 'take home' message from your project?"* You can use the Topical Guide to UBRP Conference Posters and the List of Abstracts located in this booklet to help you identify posters of interest to visit.

To give our students a chance to see each other's work:

- Odd-numbered posters will be presented from 11:00am - 12:30pm.
- Even-numbered posters will be presented from 12:30pm – 2:00pm.

SCIENCE ACTIVITIES

GROUND FLOOR

- **Symbiosis: An Exhibit of Biological Art**, Ground Floor, Room S107. This event showcases the aesthetic appeal of the life sciences and celebrates the fusion of science and art. Symbiosis is hosted by the Neuroscience and Cognitive Science (NSCS) Ambassadors with support from the School of Mind, Brain and Behavior.
- **Arthropod Diversity**, Ground Floor, Courtyard. See arachnids, giant centipedes, and stinging insects! Hosted by Dr. Justin Schmidt, winner of the IgNoble Award and author of *The Sting of the Wild.*
- **Shark Anatomy**, Ground Floor, Courtyard. Learn the anatomy of sharks and how they play an important role in our ocean. Hosted by Marine Awareness and Conservation Society (MACS).
- **Science in Color**, Ground Floor, Courtyard. Explore scientific concepts with colorful experiments. Hosted by the Biochemistry Club.

REFRESHMENTS

GROUND FLOOR, CANYON CAFÉ

2:00PM – 3:00PM ◆ CLOSING REMARKS, AWARDS, & DOOR PRIZES

MENTOR AWARDS

Each year, we celebrate UA's supportive culture for undergraduate research and the outstanding mentorship UBRP students enjoy by granting the Outstanding UBRP Graduate Student, Postdoctoral Fellow, or Research Specialist Mentor Award and the Outstanding UBRP Faculty Mentor Award. Candidates are nominated by current UBRP students and UBRP alumni. A committee composed of UBRP students and alumni reviews the nominees and selects the finalists.

2019-2020 Outstanding UBRP Graduate Student Mentor Awardee:

Cesar A. Medina
Doctoral Student, Neuroscience
Nominated by Eddie Vargas

Nominees: Sam Sugerman (Doctoral Student, Biochemistry), David Duron (Doctoral Student, Medical Pharmacology), and Brittany Uhlorn (Doctoral Student, Cancer Biology)

2019-2020 Outstanding UBRP Faculty Mentor Awardee:

Dr. Jennifer Bea
Associate Research Professor, Nutritional Sciences
Nominated by LeCario Benashley

Nominee: Dr. John Streicher (Assistant Professor, Pharmacology)

GRADUATING SENIORS

Congratulations to UBRP seniors who will be graduating in 2020! To celebrate all they have accomplished through our program, we are recognizing seniors with a teal UBRP graduation cord. Seniors, we wish you all the best for the future and look forward to receiving updates about your post-graduation positions and accomplishments. Make sure to update your profile on LinkedIn and join our UBRP group at https://www.linkedin.com/groups/3762234/.

DOOR PRIZES

UBRP thanks the following organizations and companies for donating today's door prizes:

ATHLETICS

SPECIAL OPPORTUNITY TO SUPPORT STUDENT CONFERENCE TRAVEL

UBRP participants present their research to the general public each year at UBRP's annual conference, but they can benefit further by presenting their work at national and international scientific conferences where they can learn about the latest research and network with other scientists in their field.

For the next month, we have a goal of raising $5,000 for our UBRP Travel Awards to provide ten students with a $500 travel award this year to take the next step as young scientists and present their work at a conference.

Learn more at **crowdfund.arizona.edu/UBRPtravels** and make your gift of any size before the February 27th deadline.

If you prefer, checks made payable to "University of Arizona Foundation" with "UBRP Travels" in the memo line may be dropped off at the registration table during the conference or sent to:

Undergraduate Biology Research Program
The University of Arizona
PO Box 210106
Tucson, AZ 85721

As always, contributions of any amount are greatly appreciated. We thank you for your support!

If you have any questions about supporting UBRP, please contact Jennifer Cubeta, UBRP Director, at (520) 621-9348 or cubeta@email.arizona.edu.

THANK YOU TO OUR DONORS!

FRIENDS OF UBRP

Anonymous
Ms. Carol Arakaki
Dr. Craig Aspinwall
Dr. Richard & Mrs. Laura Austin
Prof. Carol Bender
Dr. Sajiv Boggavarapu
Dr. Amitava Bose
Drs. Margaret Briehl & Dennis Ray
Drs. Gail Burd & John Hildebrand
Dr. Xuemei Cai
Ms. Roxie Catts
Mrs. Jennifer Cubeta
Mr. Richard Edelman
Dr. & Mrs. John Enemark
Dr. Brenda Gardner
Dr. Marilyn Halonen
Dr. James and Mrs. Norma Hazzard
Dr. A. Teresa Isaias
Dr. Thomas and Mrs. Jean Ito
Dr. David Johnson
Dr. Paul Klekotka
Mr. Philip & Mrs. Sharon Lagas
Mr. Brian Massey & Dr. Megan O'Meara
Mr. Robert & Mrs. Kim Nelson
Dr. Linda Restifo & Mr. Arthur Pacheco
Mr. Robert E. Smith
Dr. Anne Suzuki
Dr. Teri Suzuki & Mr. Oleg Lysyj
Dr. John Szivek
Ms. Samantha Szuter
Mr. Daniel Taylor & Dr. Sarah Nelson-Taylor
Dr. Ronald Teed
Dr. Kenneth Teter
Dr. Allison Titcomb - in memory of John R. Hendrickson
Dr. Sheldon Trubatch & Ms. Katharina Phillips
Mr. Doug & Mrs. Andrea Wellington
Dr. Jessica Yingling & Mr. Christopher Mahoney

Additionally, these individuals and entities have donated since January 1, 2019. We are grateful for your support!

Dr. David Bellows & Dr. Alison Bailin
Dr. Janet Chen
Ms. Tiffany Cho
Dr. Anne Chung
Dr. Zhen Fen
Dr. Jeffrey Frelinger
Ms. Emma Harrell
Mr. Rohith Jayaram
Ms. Marisa Lester
Ms. Kristin Perkumas
Pfizer Foundation
Raytheon Company – Matching Gifts
Dr. Daryn Stover
Dr. & Mrs. Gregory Sword
Dr. Kenneth Wertman & Dr. Barbara Caldwell
Dr. Guang Yao
Mr. Benjamin Zaepfel

ACKNOWLEDGEMENTS

ADVISORY BOARD MEMBERS

John Szivek, Chair
Nathan Ellis
Teri Suzuki
Samantha Szuter
Sheldon Trubatch
Kenneth Wertman

Emeriti Board Members:
Carol Bender
John Enemark

AMBASSADORS

UBRP Ambassadors are charged with the responsibility of helping to create community among undergraduate researchers by organizing social activities, providing feedback to program staff, and representing UBRP in speaking to on- and off-campus groups. We thank our 2019-2020 UBRP Ambassador officers for their service!

Allison Eby
President

Andrew Alamban
Vice President

Amelia Lappenbusch
Secretary

Amanda Warner
Volunteer Chair

Randall Eck
Pen Pals Coordinator

SUMMER 2019 SMALL GROUP CO-LEADERS

UBRP students meet in small groups every other week during the summer to discuss their research with their peers. Faculty, postdocs, graduate students, and advanced undergraduates volunteer their time to facilitate these groups and to mentor undergraduate researchers.

We are incredibly fortunate that these individuals volunteered their time and talents to serving as small group leaders in Summer 2019. We deeply appreciate their contributions to enriching UBRP students' experiences.

Tiffani Begay
Senior Program Coordinator, NACP

Dr. Margaret Briehl
Professor, Pathology

Alura Benally
Graduate Student Assistant, NACP

Lindsey Crown
Doctoral Student, Psychology

Randall Eck
Beckman Scholar

Austin Flohrschutz
Doctoral Student, Neuroscience

Sara Honey
Doctoral Student, Molecular & Cellular Biology

Julie Huynh
MD/PhD Student, Molecular & Cellular Biology

Elizabeth Jose
Doctoral Student, Molecular & Cellular Biology

Kathleen Lasick
Doctoral Student, Molecular & Cellular Biology

Erik Lehmkuhl
Doctoral Student, Molecular & Cellular Biology

Stephanie Matijevic
Doctoral Student, Psychology

Jennifer Roxas
Research Associate,
Animal and Comparative Biomedical Sciences

Yannick Schreiber
Beckman Scholar

Siyu Wang
Doctoral Student, Psychology

Amanda Warner
Beckman Scholar

PEN PALS

During the 2008-2009 academic year, UBRPer Misha Pangasa, in conjunction with sixth grade teacher Patricia Robles-Medina at Mansfeld Middle School, initiated the UBRP Pen Pals Project. UBRPers volunteer to correspond with sixth grade students throughout the course of the year. Every May and December, UBRP students host their sixth-grade Pen Pals in a morning of science activities on campus. We thank everyone who participated in the program this year!

Randall Eck
Pen Pals Coordinator

Ms. Susan Sumner
Mansfeld Middle School Pen Pals Teacher

UBRP Pen Pals:

Andrew Alamban
Haley Arnold
Hannah Ball
Paul Bejarano
Kiera Blawn
Alexander Blythe
Allison Eby
Randall Eck
Amanda Gregolynskyj
Nicole Kummet
Amelia Lappenbusch
David Lasansky
Maria Macias
Cecilia Martinez
Madison Mollico
Chloë Paterson
Emily Peters
Rudolph Rodriguez
Siena Schoelen
Maddie Sieffert
Kristina Sin
Erica Spence
Sneha Srinivasan
Ashley Tolton
Renata Vallecillo
Eddie Vargas
Angela Velázquez
Amanda Warner
Raj Watson
Troy Weinstein

2019-2020 PROGRAMS, PARTICIPANTS, & FACULTY MENTORS

OVERVIEW

The programs housed within the Undergraduate Biology Research Program (UBRP) are designed to teach students science by involving them in biologically related research. Students are paid for their time doing research where they develop an understanding of the scientific method and receive a realistic view of research. They also participate in professional development workshops, scientific seminars, and supplementary activities to acquire the tools necessary to be successful in post-graduate studies should they choose careers related to biology or biomedical research, and join a community of scholars as undergraduate researchers.

UNDERGRADUATE BIOLOGY RESEARCH PROGRAM (UBRP)

Funding for UBRP students is provided by private donors, the UA Office of the Provost, Office of Research, Innovation & Impact, BIO5 Institute, the deans of the Colleges of Medicine, Science, Agriculture and Life Sciences, Pharmacy, and the Mel and Enid Zuckerman College of Public Health, and the Department of Biomedical Engineering. Additional individual student support is provided by the Western Alliance to Expand Student Opportunities (WAESO). The UBRP Office is also supported by the Department of Molecular and Cellular Biology. We gratefully acknowledge this support!

Student	Mentor
Anthony Aguilar	Anita Koshy
Andrew Alamban	Janis Burt
Jeremy Anderson	Jean-Marc Fellous
Haley Arnold	Elena Plante
Hannah Ball	Daniela Zarnescu
Brenden Barness	Jamie Edgin
Amanda Bates	Mary Kay O'Rourke
Paul Bejarano	John Streicher
Anne-Laure Blanche	Renee Duckworth
Kiera Blawn	Tally Largent-Milnes
Maya Bose	Rebecca Mosher
Kylie Calderon	Kristian Doyle
Matt Christofferson	Joyce Schroeder
Allison Cully	Linda Restifo
Ava Dickerson	Carol Gregorio
Allison Eby	Stephen Cowen
Jordan Fink	Steven Goldman
Steven Fried	Michael Brown
Frankie Garcia	Kristian Doyle
Amanda Gregolynskyj	Carol Barnes
Emily Harnois	Ronald Lynch
Carly Harris	Elena Plante
Hannah Hart	Martha Bhattacharya
Luis Helfer	Jacob Schwartz
Victoria Howard	A. Elizabeth Arnold
Osagioduwa Igbinoba	Ralph Fregosi
Dong Kyun Kim	Frans Tax
Amelia Lappenbusch	Guang Yao

Student	Mentor
David Lasansky	Indraneel Ghosh
Sarah Lester	Michael Hammer
Eric Lu	Andrew Capaldi
Daniel Lucas	Monica Kraft
Jacob Mapp	Craig Aspinwall
Cecilia Martinez	Mark Beilstein
Brenna McIntyre	Jana U'Ren
Madison Mollico	David Baltrus
Emily Monroe	Stephen Cowen
Nhat Nguyen	Lalitha Madhavan
Megan Nickerson	Jana U'Ren
Caroline O'Neill	Rebecca Page
Chloe Paterson	Anna Dornhaus
Emily Peters	Paulo Pires
Yuxin Qin	Jean-Marc Fellous
Shelby Rheinschmidt	Jared Churko
Rudolph Rodriguez	Ying-Hui Chou
James Rozelle	Mark Beilstein
Siena Schoelen	Mary Alt
Madison Sieffert	John Streicher
Sara Sillik	Brian McKay
Diego Silva-Mendoza	Janis Burt
Kristina Sin	Jared Churko
Sandy Slovikosky	John Koprowski
Saskia Smidt	Torsten Falk
Julian Somers	Mark Beilstein
Tessa Spangler	Anita Koshy
Erica Spence	Samuel Campos
Sneha Srinivasan	Andrew Paek
Deserae Stanerson	Indraneel Ghosh
Jack Stearns	Mark Beilstein
Bradey Stuart	Jon Njardarson & Ingmar Riedel-Kruse
My Duyen Tran	Daniela Zarnescu
Eddie Vargas	Julie Miller
Angela Velazquez	Roberta Brinton
Andres Vizzerra	Alex Badyaev
Kevin Vo	Haijiang Cai
Daniel Wieland	Jacob Schwartz
Haley Williams	May Khanna
Alison Williams	Gayatri Vedantam
Juliana Young	May Khanna
Sylvia Zarnescu	Robert Wilson

THE BECKMAN SCHOLARS PROGRAM

The Beckman Scholars Program is designed to help stimulate, encourage and support research activities by exceptionally talented undergraduate students at our nation's universities and colleges. The Beckman Scholarship at the University of Arizona provides funding for students to conduct in-depth research with one of 15 stellar research mentors in UA's College of Science. Funding for this program is provided by the Arnold and Mabel Beckman Foundation.

Student	Mentor
Randall Eck	Daniela Zarnescu
Yannick Schreiber	John Jewett
Jamie Takashima	Pascale Charest
Amanda Warner	Ross Buchan
Catherine Weibel	Joanna Masel

MARGARET BILSON FELLOWS

Undergraduates from the Department of Molecular and Cellular Biology participated in UBRP with funding from the generous bequest of Margaret Bilson, an Arizona native and UA alumna who had a passion for biology. Additional individual student support is provided by WAESO.

Student	Mentor
Alexander Blythe	Daniela Zarnescu
Nicole Kummet	Emmanuel Katsanis
Maria Macias	Daniela Zarnescu
Amanda Ruelas	Curtis Thorne
Rafael Cancino	Michael Hammer
Ashwin Siby	Timothy Bolger

AMERICAN SOCIETY FOR PHARMACOLOGY AND EXPERIMENTAL THERAPEUTICS SUMMER UNDERGRADUATE RESEARCH FELLOWSHIP (ASPET SURF)

The ASPET SURF Program, funded by a grant from the American Society for Pharmacology and Experimental Therapeutics, supports five undergraduate students per year who work under the mentorship of ASPET members. The program's goal is to introduce undergraduate students to pharmacology research using authentic, mentored research experiences to heighten student interest in careers in research and related health care disciplines.

Student	Mentor
Ryan Hecksel	John Streicher
Haley Kenner	Ron Lukas & Rajesh Khanna
Angela Smith	Todd Vanderah
Raj Watson	Xinxin Ding
Troy Weinstein	Frank Porreca

ENVIRONMENTAL HEALTH SCIENCES TRANSFORMATIVE RESEARCH UNDERGRADUATE EXPERIENCE (EHS-TRUE)

EHS-TRUE, funded by the National Institute of Environmental Health Sciences grant #1-R25-ES025494 under Dr. Walt Klimecki, provides classroom training, two years of paid training and research experience in an environmental health sciences research laboratory, and funds for tutoring and travel to scientific meetings. The program targets students from STEM-underrepresented backgrounds. The goal of EHS-TRUE is to enhance the competitiveness of our undergraduates for post-baccalaureate education in the environmental health sciences. Additional individual student support is provided by WAESO.

Student	Mentor
Adam Carl	Mary Kay O'Rourke
Jasmine Lock	Zelieann Craig
Estevan Sandoval	Scott Boitano
Ashley Tolton	Julie Ledford
Renata Vallecillo	Julie Ledford

PARTNERSHIP FOR NATIVE AMERICAN CANCER PREVENTION (NACP)

The Partnership for Native American Cancer Prevention (NACP) is a collaboration between Northern Arizona University and the University of Arizona's Cancer Center. The mission of the NACP is to alleviate the unequal burden of cancer among Native Americans of the Southwest through research, training and community outreach programs in collaboration with the communities they serve. The Training Core of NACP at the University of Arizona is led by Dr. Margaret Briehl, and funding for NACP is provided by the National Cancer Institute grant #2U54CA143924. Additional individual student support is provided by WAESO.

Student	Mentor
Myka Becenti	Craig Aspinwall
Brenn Belone	Daniela Zarnescu
LeCario Benashley	Jennifer Bea
Shanoa Nez	Ronald Heimark
Mykell Sam	A. Elizabeth Arnold
Roxanne Vann	Heidi Hamann

TOPICAL GUIDE TO CONFERENCE POSTERS

BIOMEDICAL ENGINEERING

Presenter	Title of Poster	Poster Number	Room
Jeremy Anderson	SPHERAT: A COMPLEX SPATIAL NAVIGATION GAME	3	S230
Alana Gonzales	MODELING T CELL AND VASCULAR INTERACTIONS IN VITRO	30	S225
Jacob Mapp	DEVELOPMENT OF MOBILE AND COST-EFFECTIVE CAPILLARY ELECTROPHORESIS INSTRUMENTATION	55	S225
Jocelyne Rivera	RED BLOOD CELL-MIMETIC ARTIFICIAL PROTEIN HYDROGELS: TRANSLATING PROTEIN NANOMECHANICS INTO FUNCTIONAL HYDROGELS	72	S223
Frank Servin		80	S215
Sara Sillik	CIRCADIAN FUNCTION IN RETINAL PIGMENT EPITHELIUM	83	S215
Deserae Stanerson	SELECTIVE DOMAIN INSERTION CONTROL OF SRC KINASE	93	S215

CANCER

Presenter	Title of Poster	Poster Number	Room
Andrew Alamban	ALTERNATELY TRANSLATED PRODUCT OF CX37 FAILS TO ARREST GROWTH IN RIN CELLS BUT ALTERS CX37 HEMICHANNEL FUNCTION WHEN CO-EXPRESSED	2	S230
Myka Becenti	SYNTHESIS OF FLUORESCENT INDICATORS TO USE IN RAPID CONJUGATION REACTIONS OF AMINES AND THIOLS	8	S230
LeCario Benashley	DEVELOPING A CLINICAL TRIAL FROM THE GROUND UP	11	S230
Isabella Brown	MECHANISTIC TARGET OF RAPAMYCIN COMPLEX 2 (MTORC2) REGULATION IN CANCER CELL MIGRATION	16	S230
Matt Christofferson	TARGETING SORTING NEXINS AS A NOVEL TRIPLE NEGATIVE BREAST CANCER THERAPUETIC	20	S230
Alana Gonzales	MODELING T CELL AND VASCULAR INTERACTIONS IN VITRO	30	S225
Luis Helfer		36	S225
Nicole Kummet	PRE-TRANSPLANT BENDAMUSTINE CONDITIONING INDUCES GRAFT-VERSUS-LEUKEMIA EFFECT WITH LOWER GRAFT-VERSUS-HOST DISEASE THAN CYCLOPHOSPHAMIDE IN MURINE MODELS	45	S225
Amelia Lappenbusch	POSITIVE FEEDBACK LOOPS IN RB-E2F PATHWAY UNDERLIE ULTRA-SENSITIVITY IN DNA DAMAGE INDUCED CELL CYCLE ARREST	46	S225
Daniela Ortiz	NUCLEAR EGFR DRIVES EPIGENETIC DYSREGULATION	65	S223
Rudolph Rodriguez	THE EMOTIONAL STROOP TASK FOR CANCER PATIENTS	73	S223
Amanda Ruelas	CHARACTERIZING NOVEL KINASES IN THE COLONIC EPITHELIUM	75	S215
Andres Sanchez	INVESTIGATING FGFR3 AS A THERAPEUTIC TARGET FOR HEAD AND NECK SQUAMOUS CELL CARCINOMAS	76	S215
Erica Spence	HUMAN PAPILLOMAVIRUS EARLY GENES AS A MECHANISM FOR INNATE IMMUNE SYSTEM EVASION THROUGH THE CGAS-STING PATHWAY	91	S215
Jamie Takashima	APEX2-MEDIATED PROXIMITY LABELING IN *DICTYOSTELIUM DISCOIDEUM*	95	S215

GENETICS

Presenter	Title of Poster	Poster Number	Room
Alexander Blythe	DALLIANCE WITH WNT SIGNALING: A POTENTIAL TARGET OF DYSREGULATED TRANSLATION DURING TDP-43-MEDIATED NEURODEGENERATION	14	S230
Maya Bose	ABUNDANCES OF RDDM-PRODUCED 23NT AND 24NT SRNAS ARE THE SAME ACROSS TISSUE TYPES IN *BRASSICA RAPA* AND *ARABIDOPSIS THALIANA*	15	S230
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EFFECT OF TYPE II *TOXOPLASMA GONDII* PARASITE BURDEN ON AMYLOID BETA PLAQUE DEPOSITION IN THE J20 ALZHEIMER'S DISEASE MURINE MODEL

***ANTHONY AGUILAR*, EMILY MERRITT, ANITA KOSHY**

Alzheimer's Disease (AD) is a neurodegenerative disorder that disrupts neuronal signaling and leads to memory loss, cognitive decline, ultimately resulting in the inability to carry out autonomic body functions. The amyloid hypothesis states that misfolded proteins, known as amyloid beta (Aβ) play a key role in initiating AD pathogenesis. A hallmark characteristic of AD is the accumulation of these misfolded proteins in the form of Aβ plaques in the brain. A specific strain type of Toxoplasma gondii, a brain-persisting parasite, has been shown to provide a neuroprotective effect against Aβ plaques in AD mouse models. This finding is of particular research interest because insights into a molecular or immunological mechanism of plaque protection could assist in developing a novel treatment or therapy for AD. Our lab previously identified that this protection against plaque deposition occurred in mice infected with type II parasites, but not type III parasites. In addition to this finding, we noticed that the type II parasites had a higher parasite burden in the brain than type III parasites after the 6-month infection. These data led us to ask: is this decrease in plaque deposition strain specific or is it a consequence of the number of parasites in the brain? To further define the latter question and investigate this phenomenon, hAPP J20 AD model mice were inoculated with 500 type II parasites, 5,000 type II parasites, or saline as a control. Mice were aged for 6 months, and brains were then harvested for plaque quantification through immunohistochemical staining. Brain parasite burden was analyzed by quantitative PCR of B1, a Toxoplasma-specific gene. Surprisingly, parasite burden was found to be significantly higher in mice infected with 500 parasites compared to 5,000, and yet plaque deposition was found to be not significantly different between the two infections when compared to saline. These results preliminarily indicate the magnitude of type II parasite burden does not play a particularly significant role in protection from Aβ plaques. This research is funded by the National Institutes of Health (NIH), the Western Alliance to Expand Student Opportunities (WAESO) Louis Stokes Alliance for Minority Participation (LSAMP) National Science Foundation (NSF) Cooperative Agreement No. HRD-1101728, the UA Honors College, and the Undergraduate Biology Research Program with funds from the Office of the Provost and the College of Agriculture and Life Sciences.

ALTERNATELY TRANSLATED PRODUCT OF CX37 FAILS TO ARREST GROWTH IN RIN CELLS BUT ALTERS CX37 HEMICHANNEL FUNCTION WHEN CO-EXPRESSED

***ANDREW ALAMBAN*, MARGRET A. FYE, TASHA K. PONTIFEX, JANIS M. BURT**

Connexin proteins facilitate intercellular communication via channel formation (hemichannel) and intercellular docking of apposed hemichannels (gap junction channel). Additionally, they play a role in determining growth phenotype (e.g., arrest, proliferation, death). Proper regulation of cell growth is necessary for the daily function of organisms. Connexin 37 (Cx37) expressed in connexin-deficient rat insulinoma (Rin) cells, induces growth arrest or cell death in a manner dependent on the phosphorylation of specific carboxyl terminus (CT) residues. Notably, mimicking phosphorylation with aspartate substitution at

either S275 or S321 is sufficient to induce cell death. Thirteen of the 21 members of the connexin gene family have a conserved methionine at position 213; in Cx43 mRNA, this methionine codon serves as an alternate translation start site that produces a CT-spanning translation product. This 20kDa (20k) product interacts with Cx43, chaperoning its trafficking to the membrane. Here we test the hypotheses that 1) this conserved codon in Cx37 mRNA also serves as an alternate translation start site to produce a 13kDa product (13k) that, when expressed alone, regulates growth phenotype of Rin cells and 2) when co-expressed with Cx37, regulates its channel function. To test these hypotheses, we expressed V5-tagged 13k or one of two phosphomimetic mutants, 13k-S321D and 13k-S275D, in Rin cells or in Rin cells expressing Cx37. We assessed growth phenotype of these cells. Despite a similar phosphorylation profile to Cx37, as determined by mass spectrometry, 13k did not arrest Rin cell growth and neither phosphomimetic mutant induced cell death nor growth arrest. In co-expressing cells, immunoprecipitation (IP) of 13k failed to co-IP full-length Cx37, suggesting no direct interaction between these isoforms; however, 13k and the phosphomimetic mutants altered Cx37 hemichannel conductance and open probability properties, suggesting an indirect interaction of 13k isoforms with Cx37. In conclusion, these data suggest that while 13k and phosphomimetic mutants alone are insufficient in altering the growth phenotype of Rin cells toward growth arrest or death, they alter channel properties of full-length Cx37. The findings herein will inform further research in understanding how connexins are regulated and, in turn, the role they play in regulating cell growth phenotypes. This work is supported by the National Heart, Lung, and Blood Institute (NHLBI) of the National Institutes of Health (NIH) under award number R01HL131712, university funds, and the Undergraduate Biology Research Program with funds from the Office of the Provost.

SPHERAT: A COMPLEX SPATIAL NAVIGATION GAME

***JEREMY ANDERSON*, KRISTINE GRADISHER, JEAN-MARC FELLOUS**

I present a 3D computer game, named SPHERAT, which is designed to imitate the spatial navigation experiments performed by the rats in The Computational and Experimental Neuroscience Laboratory (Dr. Jean-Marc Fellous). These tasks require the rat/player to navigate around a room with a variable number of obstacles and find all available rewards in a given time limit. The main purpose for SPHERAT is to introduce people outside of STEM fields to the research conducted in the CENL lab through an interactive medium, which is meant to address the disconnect between the layperson and complex scientific research. An additional application is in behavioral research using virtual environments; SPHERAT can be modified through a series of text files encoding game parameters and outputs relevant game data, which is used in experimental analysis. This allows for behavioral human navigational studies to be performed in an efficient manner, as the game has no required setup and few physical limitations, unlike traditional behavioral research. The game creation software Unity was used to create SPHERAT, with all programming done in C#. The game requires a user to pilot a sphere (the SPHERAT) around an arena and collect all rewards present while navigating around any obstacles. Position and event data are collected in a format compatible with the CENL lab's analysis software. Game parameters such as obstacle coordinates, reward coordinates, time limits, and trial limits can all be modified through a set of input text files, giving the game a large degree of customizability. SPHERAT mainly has implications for the CENL lab, as a fun method for familiarizing non-STEM people with the CENL lab's research or as a tool for human navigational research imitating our rat experiments but could potentially be used in other research settings for easy, efficient navigational research requiring little setup. Funding was provided by the Undergraduate Biology Research Program with funds from the Office of the Provost and the Department of Biomedical Engineering, and the National Science Foundation grants 1429929 and 1703340.

THE EFFECT OF WORD GENERATION ON THE RETENTION OF NOVEL WORDS IN CHILDREN WITH DEVELOPMENTAL LANGUAGE DISORDER

***HALEY ARNOLD*, LUCIA SWEENEY, ELENA PLANTE, REBECCA GOMEZ**

Developmental Language Disorder (DLD) affects nearly 10% of the population and is characterized by persistent speech, language, and communication problems that are not accompanied by outside cognitive or intelligence impairments. Considering that children with DLD often struggle with learning and retaining vocabulary, we examined whether having a child

with DLD generate a novel word out loud increases their ability to accurately recall and identify that same word. We taught twenty monolingual 4- and 5-year-old children six novel words over three sessions, half of which they generated twice per session, followed up with two tests to assess their retention of the words 48 hours and three weeks after the last session administered. Overall, no differences were found between retention of generated versus non-generated words at either the 48 hour or three-week testing period. These results reflect what was previously found in a high-variability condition where three different physical representations of the novel word were present. This shows that even in the low-variability item condition, where three identical physical representations of the novel word are present, no significant differences were found. This research indicates that although previous studies with typical language children showed better retention with word generation, that this may not be the case for children with DLD. This project is supported by the National Institutes of Health (NIH) under award number R01DC015642, the Undergraduate Biology Research Program with funds from the College of Science and the Office of the Provost, as well as through the generous donations from Cecile Moore.

CHARACTERIZATION OF METABOLIC DEFECTS ACROSS MULTIPLE MODELS OF ALS

***HANNAH BALL*, SUVITHANANDHINI LOGANATHAN, ERNESTO MANZO, ABIGAIL O'CONNOR, GABE BIRCHAK, DANIELA C. ZARNESCU**

Amyotrophic lateral sclerosis (ALS) is a fatal neurodegenerative disease that disrupts muscle function and has no cure. TAR DNA Binding Protein (TDP-43) is an RNA binding protein that has been found in cytoplasmic aggregates in 97% of ALS cases regardless of etiology. *Drosophila* is a well-established genetic model for human disease that we used to develop a model for ALS based on TDP-43. Flies expressing either wild-type or mutant human TDP-43 (wild-type and mutant) recapitulate several symptoms of ALS, including motor dysfunction and a reduced survival. Recently, we found that glycolysis is upregulated in this model as a compensatory mechanism that improves locomotor function and increases lifespan. To determine whether glycolysis is similarly altered in other types of ALS we are using different fly models based on C9, SOD1 and a recently generated CRISPR model of TDP-43 proteinopathy. Preliminary results suggest that similar to the TDP-43 proteinopathy model based on overexpression, the CRISPR model as well as SOD1 and C9 models can also benefit from increased glucose availability. We will report on the neuroprotective potential of increased glycolysis on key ALS phenotypes including locomotor function, neuromuscular junction morphology and lifespan across several *Drosophila* models of ALS. This work was funded by National Institutes of Health (NIH) under award number RO1NS091299, the Muscular Dystrophy Association award 418515 (to DCZ), the Howard Hughes Medical Institute Gilliam Fellowship (to EM), and the Undergraduate Biology Research Program with funds from the Office of the Provost and the College of Science (to HB).

STANDARDIZED AND NOVEL EXECUTIVE FUNCTION TASK PERFORMANCE IN CHILDREN WITH DOWN SYNDROME IN RELATION TO PARENT PERCEPTION OF EXECUTIVE FUNCTION

***BRENDEN BARNESS*, MICHELLE LOPEZ, KATHARINE HUGHES, LAUREN PISANI, TYRA PROTHO, MIRANDA SAMPSEL, NANCY LEE, LEN ABBEDUTO, ANGELA THURMAN, PAYAL KHOSLA, JAMIE EDGIN**

Executive function (EF) is broadly defined as the set of processes that deal with managing oneself and the environment in order to achieve a goal (Cooper-Kahn & Dietzel, 2008). There are several ways of measuring EF, including standardized assessments such as the Flanker task and the BRIEF parent-report questionnaire. Previous research using these types of assessments suggests a broad impairment of EF in children with Down syndrome (DS) (Gioia et al., 2000; Traverso et al., 2018). The current analyses evaluate the validity of the Arizona Memory Assessment for Preschoolers and Special Populations (AMAP), which tests cognitive function among children with intellectual disabilities. As part of a multisite measurement validation study, we administered the AMAP, Flanker, and BRIEF in school-age children with DS (N = 46) and mental-age matched TD children (N = 46). For the current project, we looked at an EF-specific phase in the AMAP where an image was displayed at the center of an iPad with 'distractor' images around it. Each image was paired with a corresponding object that was located in either bottom corner of the screen. The participant was required to match the central image with the correct object in the appropriate corner. Eight sequences were tested within this phase, allowing each image to be both the middle image (desired), as well as the

'distractor' image, depending on the sequence. Results indicate a significant difference in Phase 12 average scores with the DS group displaying lower scores than their TD counterparts (5.53 vs. 6.63). There was a significantly positive correlation between the AMAP total score and Flanker incongruent score (p = 0.009) as well as the AMAP total score and Flanker total score (p = 0.007) in the DS group. In the TD group, AMAP total score was positively associated with the Flanker congruent score (p = 0.002) and total score (p = 0.018) but negatively correlated with the Flanker congruent reaction time (p = 0017). The Flanker congruent and incongruent total scores were also positively associated in the TD group (p < 0.001) and DS group (p = 0.017). We found no significant correlations between the AMAP variables and the Global Executive Composite (GEC) score on the BRIEF and the Flanker task and GEC in either diagnosis group. These results confirm the presence of EF deficits in DS and highlight new methods of assessing these domains in individuals with DS. This work was supported in part by the Undergraduate Biology Research Program with funds from the Office of the Provost.

A QUANTIFICATION OF POLYCYCLIC AROMATIC HYDROCARBONS AND VOLATILE ORGANIC COMPOUNDS FROM RESIDENTIAL TRASH BURNING

***AMANDA BATES*, EMMANUEL GONZALEZ FIGUEROA, LORETTA STONE, MARTI LINDSEY, MARY KAY O'ROURKE**

Polycyclic Aromatic Hydrocarbons (PAHs) and Volatile Organic Compounds (VOCs) are air pollutants produced when burning trash and fossil fuels. Inhalation exposures to PAHs and VOCs are associated with adverse health effects such as respiratory illnesses, immunological effects, and various cancers. PAHs are created by incomplete combustion reactions. This study quantified various air contaminants from burning residential trash and examined how exposure could precipitate adverse health effects. Real-time monitors for PAHs and VOCs were placed ~5-10 ft from a burn barrel at each of nine homes across the four residential districts near Globe, AZ. PAHs were sampled using an ECO-Chem pas-2000 monitor operated at a flow rate of 2 L/min using a Casella Epex 3 and logging data each second. VOCs were sampled at one-minute intervals using an Ion Tiger VOC monitor. Additional area samples were collected using an Absorbent Tenax TA Tube at 800 mL/min. At each house, trash was sorted into bins labeled organic material, plastic, metal, paper, and other (determined by the EPA trash Classification System). The sorted trash was mixed before being placed into the burn barrel to replicate a burn by the residents. The mean PAH (SD) concentration was 160.41ng/min (223.9) and mean VOC (SD) concentration was 287.87 ng/min (240.7). Plastic was the dominant trash type (27.5%) followed by organics (27.9%). Higher VOCs were associated with households that burned a greater percentage of paper, plastics, and metals for some households. PAH concentrations were completely independent of other measures having no relationship to VOC concentrations, trash burning, or specific trash types being burned. Further sampling and analysis would be required to understand the inverse relationship between PAHs and VOCs by sorting more specific sub-types of the sorted categories and for greater representation of the population with a larger sampling size. This work was supported by the Undergraduate Biology Research Program with funds from the Office of the Provost and the College of Public Health (MEZCOPH). Field collection, equipment and analysis support was provided by National Institutes of Health under award number P50ES026089 and the Environmental Protection Agency under assistance agreements no. 83615101. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health or the U.S. Environmental Protection Agency. It has not been formally reviewed by EPA. The EPA does not endorse any products or commercial services mentioned in this publication.

SYNTHESIS OF FLUORESCENT INDICATORS TO USE IN RAPID CONJUGATION REACTIONS OF AMINES AND THIOLS

***MYKA BECENTI*, MINHU HAN, CRAIG A. ASPINWALL**

Redox signaling pathways are crucial in mediating cellular health through biological signaling processes. Reactive nitrogen species (ROS), including nitric oxide (NO) and nitroxyl (HNO) play a key role in a number of metabolic pathways and can serve as important pharmaceutical agents. The evaluation of HNO signaling pathways, in particular, can advance diagnosis and treatment of many diseases, including cardiovascular disorders, cancer, and alcoholism. However, this metabolic signaling molecule is difficult to quantify because of its high reactivity and short biological half-life. As a result, current methods for

quantitatively measuring HNO in vivo are limited. The measurement of HNO must be detected indirectly e.g. using the reaction product between an indicator and HNO. This ongoing study is geared at enhancing the understanding of cell signaling in pancreatic beta cells in metabolic pathways through detection of intracellular HNO concentration in response to HNO releasing pharmaceutical compounds. As a result, the synthesis of the CFS indicator dye can enable detection of HNO upon exposure to HNO producing drugs that can help to alleviate symptoms of diabetes that include cardiomyopathy, as well as limit cancer progression and better treat alcoholism. This research was funded through the Partnership for Native America Cancer Prevention (NACP) through a grant from the National Cancer Institute, grant #2U54CA143924.

IDENTIFICATION OF THE HSP90 ISOFORMS AND CO-CHAPERONES THAT REPRESS OPIOID ANTI-NOCICEPTION IN THE SPINAL CORD

***PAUL BEJARANO*, DAVID I. DURON, SANKET MISHRA, BRIAN S.J. BLAGG, JOHN M. STREICHER**

The various isoforms of the chaperone protein heat shock protein 90 (Hsp90) in conjunction with specific co-chaperones are key regulators of signal transduction. We have recently identified a novel role for Hsp90 within downstream mu opioid receptor (MOR) signaling both within the brain and spinal cord. Within the brain, we found that Hsp90α and the co-chaperones Cdc37 and p23 promote MOR-induced ERK phosphorylation and subsequent anti-nociception. However, in the spinal cord Hsp90 represses MOR-induced ERK phosphorylation and subsequent anti-nociception, so that Hsp90 inhibitor treatment in the spinal cord results in enhanced opioid pain relief. This study aimed to identify the Hsp90 isoforms and co-chaperones necessary for this mechanism within the spinal cord, and whether they differ from the brain. We used novel isoform and co-chaperone selective small molecule inhibitors and in vivo gene knockdown using CRISPR/Cas9 to selectively block each of the Hsp90 isoforms and co-chaperones in the spinal cords of male and female CD-1 mice. We then used opioid-induced anti-nociception in the tail flick assay as a readout, combined with immunohistochemistry to validate CRISPR/Cas9 knockdown in the spinal cord. In contrast to our earlier brain studies, we found that the isoforms Hsp90α, Hsp90β, and Grp94 all acted to repress opioid anti-nociception in the spinal cord, in that inhibiting these isoforms resulted in increased opioid anti-nociception. We similarly found that the co-chaperones Cdc37, p23, and Aha1 all similarly repressed opioid anti-nociception in the spinal cord. These findings suggest that Hsp90α, Grp94, and Aha1 all act within the spinal cord but not the brain to regulate opioid anti-nociception. These findings also suggest, in line with our earlier studies, that blocking these isoforms with systemic selective inhibitors could represent a novel therapeutic approach to enhance opioid therapy, increasing analgesic efficacy while reducing side effects like tolerance and reward/addiction. These studies were supported by the Arizona Biomedical Research Commission New Investigator Award (#ADHS18-198875), institutional funds from the University of Arizona to JMS, the National Institutes of Health (NIH) under award number R01CA213566 to BB, and the Undergraduate Biology Research Program with funds from the Office of the Provost. BB is a founder with an equity stake in Grannus Therapeutics, a virtual startup for developing novel Hsp90 inhibitors. No other author has any relevant conflicts to declare.

DEVELOPING A CLINICAL TRIAL FROM THE GROUND UP

***LECARIO BENASHLEY*, ANA BUCY, JENNIFER BEA**

In research, much time is dedicated to the discussion of a study's intervention and its subsequent results, but hardly any time is given to detail the equally important steps which had to happen before a study and its results could even manifest. This project seeks to highlight those many necessary steps. That journey will be demonstrated using the study titled Prevention of Falls among Older Multiethnic Cancer Patients receiving chemotherapy (PERFORM ANEW). The trial is a complex multicomponent project which aims to reduce the number of falls in the most at-risk population. It offers to be the perfect case-study to illustrate the people needed, the approvals required, the protocols to be incepted, the materials to be created, and the overall work necessary for a research idea to be ethically implemented and successful in its aims. Study conducted with funding from Disarm Therapeutics and the National Cancer Institute, including the University of Arizona Cancer Support Grant (CA023074) and the Partnership for Native America Cancer Prevention (NACP) through a grant from the National Cancer Institute, grant

#2U54CA143924. Expertise and clinical advice were given by The University of Arizona Cancer Center Breast Team, Arizona Oncology Associates (Flagstaff), and NRG Oncology members.

SOCIAL CONTEXT INFLUENCES EXPRESSION OF AGGRESSION IN THE ZEBRA FINCH

***ANNE-LAURE BLANCHE*, KATHRYN CHENARD, RENEE DUCKWORTH**

By definition, social behaviors are always expressed with the context of other individuals. However, this poses challenges to measuring them in a standardized fashion because measurements outside the normal social context may not act as a true measure of natural behavior. Here, we assess the importance of social context in the expression of aggression in zebra finches, a social species in which aggression is important in resource acquisition and the establishment and perpetuation of dominance hierarchies. Aggression was measured repeatedly in a familiar flock and was measured at least once for each individual outside their normal flock using a mirror test. Birds in a flock were recorded accessing a feeder, and the number and intensity of aggressive interactions, as well as their dominance position, were assessed. During the mirror test, aggressive response to the individual's own reflection in a mirror was scored to assess an individual's reaction to an unfamiliar but equally-matched bird. Aggression and dominance are highly repeatable in the flock context and are positively correlated with one another. Preliminary results indicate that mirror tests do not provide reliable estimates of aggression under typical scenarios. This may be caused by the level of familiarity rather than the loss of a typical flock, as previous studies have shown that displacements decrease with increasing familiarity. In the future, I will create small flocks, imitating naturally-occurring foraging groups, with birds that have been in contact for differing amounts of time to research the effect of group size and familiarity on aggression. Given that many studies measure aggression by using the mirror test, it is important to determine how it relates to the variation in aggression expressed in normal flock contexts. This research was support by the Undergraduate Biology Research Program with funds from the BIO5 Institute.

EXAMINING OATP1A2'S ROLE IN SUMATRIPTAN UPTAKE DURING CORTICAL SPREADING DEPRESSION

***KIERA BLAWN*, TALLY LARGENT-MILNES, SEPH PALOMINO, KYLE BHATT, ANYA BURTMAN**

Migraines affect around a billion people worldwide and can include symptoms such as nausea, dizziness, irritability, and sensory disturbances. In a stage of migraine called aura, a phenomenon known as cortical spreading depression (CSD) has been observed to occur. CSD is characterized in the brain as a wave of hyperactivity followed by inhibition of the neurons. During CSD, changes in the permeability of the blood brain barrier (BBB) have been found. A possible explanation for the increase in permeability is that transporters, which selectively allow molecules into the brain, are also changing during CSD. A transporter called OATP1A2, found in the capillary endothelial cells of the BBB, uptakes anti-migraine agents into the central nervous system, and its expression levels have been observed to increase during migraines. The upregulation of OATP1A2 could result in a change in the uptake of migraine drugs and needs further study. This project examines OATP1A2s involvement in the uptake of an antimigraine drug, Sumatriptan, by measuring periorbital allodynia in rats induced with CSD and looks at Sumatriptan transport in bEnd.3 cells. In addition, OATP1A2 changes in expression during CSD were quantified using western blots. This data can suggest how OATP1A2 is affected during CSD and if it is directly involved in the transport of Sumatriptan. This research was support by the Undergraduate Biology Research Program with funds from the BIO5 Institute and the Office of the Provost.

DALLIANCE WITH WNT SIGNALING: A POTENTIAL TARGET OF DYSREGULATED TRANSLATION DURING TDP-43-MEDIATED NEURODEGENERATION

***ALEXANDER BLYTHE*, ERIK LEHMKUHL, ERIC ALSOP, DIANNE BARRAMEDA, BHAVANI B. SIDDEGOWDA, ARCHI JOARDAR, KENDALL JENSEN, DANIELA C. ZARNESCU**

Amyotrophic lateral sclerosis (ALS) is a debilitating neurodegenerative disease defined by the progressive loss of motor neurons and for which there is no single established genetic or environmental cause. Cytoplasmic insoluble complexes containing the TAR DNA-binding protein TDP-43 have been found in 97% of ALS cases. To study the role of TDP-43 proteinopathy in motor neurons during ALS progression, a TDP-43-based genetic model of ALS has been developed in *Drosophila melanogaster* by overexpressing wild-type or mutant TDP-43, which results in the recapitulation of ALS phenotypes such as attenuated locomotor function and significantly reduced lifespan. One hypothesized contribution to motor neuron degeneration is the sequestration of specific mRNAs by TDP-43 into cytoplasmic insoluble complexes, thereby preventing translation of a wide range of mRNAs. This hypothesis is supported by previous literature showing futsch as a target that is translationally inhibited in ALS-afflicted fruit flies. A potentially dysregulated secondary target has been identified in dally-like protein (DLP), a glypican present in the extracellular matrix of motor neurons in the neuromuscular junction. We observe that overexpression of DLP in diseased flies is neuroprotective and knockdown of DLP exacerbates ALS phenotypes, while DLP expression is concentrated in the ventral nerve cord and reduced at the neuromuscular junction in flies expressing mutant TDP-43. Funding for this research was provided by Margaret Bilson Endowment.

ABUNDANCES OF RDDM-PRODUCED 23NT AND 24NT SRNAS ARE THE SAME ACROSS TISSUE TYPES IN *BRASSICA RAPA* AND *ARABIDOPSIS THALIANA*

***MAYA BOSE*, JEFFREY W. GROVER, REBECCA A. MOSHER**

The RNA-directed DNA Methylation (RdDM) pathway plays a major part in epigenetic silencing in plants by facilitating de novo methylation, which helps protect the genome from transposable elements. In this pathway, a small RNA (sRNA) duplex is transcribed by the plant-specific RNA Polymerase IV (Pol IV) and RNA-dependent RNA Polymerase 2 (RDR2). This duplex is processed by a Dicer-Like 3 (DCL3) and loaded onto Argonaute 4 (AGO4). The sRNA-bound AGO4 is then recruited to a scaffold transcript of Pol V and linked to Domains Rearranged Methyltransferase 2 (DRM2), which performs de novo DNA methylation. While research surrounding the RdDM pathway has increased, many details of the process remain unknown. We investigated the biogenesis and degradation of the DCL3-processed sRNA duplex. Recent research suggests that RDR2 leaves an additional, untemplated nucleotide at the 3' end of its transcript. As a result, the DCL3 processed duplex is thought to consist of a 24-nt Pol IV product guide strand paired with a 23-nt RDR2 product passenger strand. We compared mapping rates and abundances of 24-nt sRNAs to those of 23-nt sRNAs in *Brassica rapa* and *Arabidopsis thaliana* samples. From this, we determined that there is essentially no variation in abundance of 23-nt sRNAs compared to 24-nt sRNAs between tissue types in *B. rapa* and *A. thaliana*. This supports a model in which the DCL3 processed duplex remains stably paired up to the point when the 24-nt strand is loaded to AGO4, at which point the 23-nt strand is ejected and quickly degraded. We have also created a Snakemake workflow to automate the data analysis pipeline used for the process of trimming, filtering, and mapping sRNA reads to a reference genome. This makes the data analysis process more accessible to those less comfortable with using bioinformatic tools, in addition to increasing the reproducibility of the data analysis process. This research is supported by the Undergraduate Biology Research Program with funds from the Office of the Provost and the College of Agriculture and Life Science.

MECHANISTIC TARGET OF RAPAMYCIN COMPLEX 2 (MTORC2) REGULATION IN CANCER CELL MIGRATION

***ISABELLA BROWN*, SHANNON COLLINS, PASCALE CHAREST**

Breast cancer affects one in seven women and is the third most deadly cancer in the United States. Breast cancer is deadly when the cancer cells acquire migratory ability and invade the local tissue. Clinically, breast cancer is treated based on the presence or absence of growth receptors estrogen (ER), progestogen (PR), and human epidermal growth factor receptor 2 (HER2). Ras is the most commonly mutated oncogene in human cancer and is associated with aggressive cancer tumors. Ras is a small GTPase that when localized to the membrane activates effectors such as PI3K and MAPK. We previously reported a new potential effector of Ras, the Mechanistic Target of Rapamycin Complex 2 (mTORC2), which is a protein complex, consisting of mTOR, Rictor, Sin1, and Lst8. Once the protein complex is formed, mTORC2 creates a stable serine threonine kinase that activates pathways through direct phosphorylation, including AKT, which is commonly used as a marker for mTORC2 activation. The regulation of mTORC2 is not yet known, however. Based on preliminary experiments, I hypothesize that Ras regulates mTORC2. Literature has shown, when Ras is upregulated, there is an increase in cancer cell proliferation, invasion, and metastasis and the enhanced migration may be mediated through mTORC2. To test this hypothesis, a normal breast tissue cell model and breast cancer cell model were used in migration experiments. Using Ras functional inhibitors, which works by mis-localizing Ras from the membrane, we observed a decrease in cell migration in both models when Ras function was inhibited. When mTORC2 was knocked down, a decrease in cell migration was also observed. This suggests that both Ras and mTORC2 have a role in cell migration.

BACE1 INHIBITION MITIGATES NEUROPATHOLOGICAL HALLMARKS OF ALZHEIMER'S DISEASE BUT IMPAIRS RECOVERY FOLLOWING STROKE

***KYLIE CALDERON*, FRANKIE GARCIA, JENNIFER FRYE, RACHAEL HEARNE, JACOB ZBESKO, DANIELLE BECKTELL, KRISTIAN P. DOYLE, THUY-VI V. NGUYEN**

Post-mortem analyses indicate that nearly 54% of patients clinically diagnosed with Alzheimer's disease (AD) show not only pathology characteristic of AD, but also pathological signs of having at least one stroke during their lifetime. Given the frequent coexistence of AD and stroke pathology, the goal of this study is to investigate how stroke could be causing the development or exacerbation of AD. We recently demonstrated that the chronic sequelae of stroke place a stress on myelin homeostasis causing the upregulation of the neuregulin-1 (NRG1) type III myelin repair pathway for at least seven weeks following stroke. An enzyme involved in this pathway is β-secretase 1 (BACE1), which cleaves NRG1 type III, releasing a peptide that stimulates myelination. However, BACE1 also cleaves amyloid-beta precursor protein (AβPP), leading to the release of amyloid beta (Aβ). Aβ can fibrillate and form plaques, a hallmark of AD. We hypothesize that by activating BACE1 for myelin repair, stroke also amplifies Aβ production, promoting the development of AD. We are testing this hypothesis in aged mice genetically engineered to develop AD. We previously found that compared to mice that underwent sham surgery, mice that underwent stroke showed more BACE+ and NRG1 type III+ immunostaining, suggesting increased activation of the BACE1/NRG1 myelin repair pathway in the months following stroke. We also saw more advanced AD pathology in mice that underwent stroke, including more ThioS+, Aβ42+, and p-τ+ deposits. In the current study, we assessed the behavioral and neuropathological effects of BACE1 inhibition following stroke. In behavioral tests, mice treated with a BACE1 inhibitor showed better spatial working memory and intermediate memory than saline-treated mice, but reduced motor recovery following stroke. Through immunostaining, we found fewer BACE1+, Aβ42+, and ThioS+ plaques in mice treated with BACE1 inhibitor, suggesting that BACE1 is involved in Aβ production following stroke. Although BACE1 inhibition mitigated AD pathology and some cognitive decline following stroke, BACE1 inhibitors have repeatedly failed in clinical trials. We suspect this is because BACE1 inhibition suppresses myelin repair, which is important for healthy neuronal function during aging and following stroke. We are currently testing the role of BACE1 in myelin repair following stroke through immunostaining for NRG1 type III, myelin, and cholinergic neurons in mice treated with a BACE1 inhibitor. This research was supported in part by the Western Alliance to Expand Student Opportunities (WAESO) Louis Stokes Alliance for Minority Participation (LSAMP) National Science Foundation (NSF) Cooperative Agreement No. HRD-1101728 and by the Undergraduate Biology Research Program with funds from the Offices of the Provost and Research, Innovation & Impact.

OBSERVATIONAL STUDY: COOKING BREAD OVER AN OPEN FIRE

***ADAM CARL*, MODHI ALSHAMMARI, BEATRICE NORTON, ROBIN HARRIS, MARY KAY O'ROURKE**

Exposure to high levels of particulate matter 2.5 ($PM_{2.5}$) from household air pollution is associated with respiratory infections such as, chronic obstructive pulmonary disease and lower respiratory diseases. Burning of solid mass like wood as a source of energy is common throughout rural and underserved communities. This contributes to elevated household air pollution when cooking over an open fire. We performed an observational study on the making of bread over an open fire. The cooking practice is often done inside a special home. Cooking is done on a stone over an open fire. We measured particulate matter 2.5 ($PM_{2.5}$) in three special homes using a personal DataRAM (pDR-1500) and MicroPEM monitor. Placement occurred inside the homes in the summer, each assessment ran for approximately five hours. The pDR-1500 contained filters that were analyzed for metal concentrations using inductively coupled plasma mass spectrometry (ICP-MS). This observational study aimed to identify which activities caused peak $PM_{2.5}$ exposure when making the bread. Each home had a different type of smoke ventilation (chimney or no chimney). Peak $PM_{2.5}$ exposures were correlated with cooking activities. Results: Data were collected in a total of three special homes. A maximum peak of 44491.42 $\mu g/m^3$ was recorded by the pDR-1500 and 12041.122 $\mu g/m^3$ by the MicroPEM monitor. Peak exposures were seen in each home during the ignition of the fire and placement of batter on the stone. During the ignition of the fire, a peak of 935.646 $\mu g/m^3$ was recorded in the house with a chimney and 1900.834 $\mu g/m^3$ in the house without a chimney. Lower $PM_{2.5}$ levels were seen in homes with a chimney compared to those without a chimney. Preliminary data indicates different ventilation methods effect the particulate matter 2.5 levels within the special homes when cooking over an open fire. This research is supported with funds from the Environmental Health Sciences – Transformative Research Undergraduate Experience (EHS-TRUE) through the National Institute of Environmental Health Sciences Grant #1-R25-ES025494.

TARGETING SORTING NEXINS AS A NOVEL TRIPLE NEGATIVE BREAST CANCER THERAPUETIC

***MATTHEW CHRISTOFFERSON*, BEN ATWELL, JOYCE SCHROEDER**

Triple negative breast cancer (TNBC) has the worst prognosis of all the subtypes of breast cancers and is defined by a lack of estrogen, progesterone, and Her2 receptors. In TNBC, along with the lack of these receptors, around 60 percent have an amplification and overexpression of the Epidermal Growth factor Rector (EGFR) (American Cancer Society). Activated EGFR has been shown to lead to downstream signaling cascades that end with migration, proliferation, and survival. These phenotypes are necessary for normal cellular functions, however when they are uncontrolled, EGFR is also associated with cancerous phenotypes such as metastasis. This occurs in TNBC when EGFR undergoes retrograde trafficking which can occur under conditions of polarity loss and/or colocalization with Mucin1 (MUC1) (Maisel et.al 2018). Retrograde trafficking of EGFR results in EGFR bypassing the lysosome, allowing EGFR to continue signaling and to traffic to the nucleus (Bitler et. al., 2010). The constitutively active EGFR downstream cascade leads to a dramatic increase in the cancerous phenotypes of migration, tumor aggressiveness, proliferation, and invasion (reviewed in Maisel et. al., 2019). Sorting nexin (SNX) proteins are known to be involved in the orchestration of intracellular lysosomal trafficking of EGFR (Chin et. al., 2001). One of the main components of SNX proteins is the SNX-Bar domain, which is the binding domain for dimerization of the SNX proteins and influence the trafficking of vesicles. These SNX proteins are also able to interact with EGFR family receptors via the same Bar domain. It has been shown that overexpression in the SNX1 binding domain to EGFR leads to an increase in the degradation of EGFR (Chin et. al., 2001). This led to the understanding that targeting the SNX proteins could lead to a novel therapeutic for TNBC. Our lab thus created a novel peptide SNX1.3, made from the SNX-Bar domain as a way to redirect EGFR trafficking to the lysosome. We hypothesize that SNX1.3 changes the retrograde trafficking of EGFR and directs it to the lysosome for degradation, thereby decreasing EGFRs signaling cascade and reversing the cancerous phenotypes such as migration associated with EGFR signaling. This research is supported by the Undergraduate Biology Research Program with funds from the BIO5 Institute and the College of Agriculture and Life Sciences.

DEVELOPMENT OF A LUCIFERASE REPORTER ASSAY TO STUDY NONSENSE MEDIATED DECAY IN CARDIOMYOPATHY

***AVA DICKERSON*, CAROL GREGORIO, CHRISTOPHER PAPPAS**

Leiomodin-2 (LMOD2) is a protein in cardiac muscle that is essential for actin-thin filament assembly. Currently, we are investigating the case of a baby that was born with a nonsense mutation in LMOD2. mRNAs with premature stop codons are degraded by a pathway called nonsense mediated decay (NMD), which significantly reduced the amount of truncated LMOD2 that was produced in the baby's heart. The loss of LMOD2 caused a detrimental phenotype, dilated cardiomyopathy, which led to cardiac failure just after birth. With many possible drug treatments to inhibit NMD, a quick assay was needed to screen their efficacy. Fusing LMOD2 to firefly luciferase allows us to measure levels of luminescence, and thus detect the levels of LMOD2 in a cell within an hour after collection. The fused LMOD2-luciferase protein with a premature stop codon is also susceptible to NMD, so the efficacy of NMD-inhibiting drugs can be determined by measuring luminescence levels. NMD may be inhibited by antisense oligonucleotides, which bind to mRNA and allow the transcript to proceed to translation instead of being degraded. We conducted experiments with antisense oligonucleotides, which partially restored levels of mutated LMOD2 mRNA and increased the amount of truncated LMOD2 within the cell. We have evidence that the truncated protein is partially functional, so its expression is better than no LMOD2 at all. The luciferase reporter assay and antisense oligonucleotide experiments may be extended to similar mutations, proving to be valuable for future studies. This project was supported by the Undergraduate Biology Research Program with funds from the Office of Research, Innovation & Impact and the College of Medicine.

UNDERSTANDING THE EFFECTS OF LOW-DOSE KETAMINE ON RAT MOTOR CORTICAL NEURONS

***ALLISON EBY*, CAMERON A. WILHITE, MITCHELL J. BARTLETT, TONY YE, TORSTEN FALK, STEPHEN L. COWEN**

Ketamine has gained traction as an effective treatment for depression and pain; however, the mechanisms underlying ketamine's effectiveness are not known. By implanting mobile tetrodes into the motor cortex of rat brains, we can take a closer look at how a neuron's amplitude, firing rate, and frequency changes after low-dose ketamine is administered intraperitoneally (20 mg/kg). We predict the firing rate of neurons due to ketamine will increase so much so that the interactions between principal neurons and interneurons will outcompete other interactions responsible for dyskinesia. Recording acute neuron activity before, during, and after injection will provide the information needed to compare these characteristics and piece together the mechanism. This could be particularly useful as recent findings have indicated that low-dose ketamine injections significantly reduce the symptoms of levodopa-induced dyskinesia (LID). LID is a side-effect of the drug levodopa, an efficient treatment for Parkinson's disease (PD); however, long-term administration of levodopa induces motor dyskinesias in patients. Understanding how ketamine acts within the brain will allow for better drug production in the future and more effective outcomes for PD patients. This research was supported by the Undergraduate Biology Research Program with funds from the Offices of the Provost and Research, Innovation & Impact.

USING GROWTH FACTORS TO STIMULATE CARDIAC REGENERATION AND PROGENITOR CELL DIFFERENTIATION

***JORDAN FINK*, JORDAN LANCASTER, STEVEN GOLDMAN**

Over 500,000 new cases of Chronic Heart Failure (CHF) occur annually in the United States alone, with mechanical assist devices and heart transplantation as the only options for end -stage CHF. Regenerative medicine has shown potential to restore cardiac function and improve the quality/length of life in patients with CHF. The primary cause of CHF is the loss of billions of cardiomyocytes which are unable to spontaneously regenerate. Endogenous repair mechanisms such as cardiomyocyte proliferation, mobilization and homing of progenitor cells, or resident cardiac stem cells do not induce significant regeneration post-injury. These endogenous methods are hindered by the inability of cardiomyocytes to proliferate in the adult mammalian

heart within a microenvironment distinguished by ischemia, inflammation, fibrosis, and poor angiogenesis. Recently, growth factors have shown to mitigate the microenvironmental effects and increase the regenerative potential of the adult heart. Many growth factors targeting angiogenesis, chemotaxis, anti-apoptosis, and cardiomyocyte proliferation have individually improved cardiac function post-myocardial infarction. We suggest that these growth factors can be applied more consistently, directly and cooperatively to produce an additive and synergistic effect for increased cardiac regeneration. By screening for growth factor combinations that increase cardiomyocyte cell cycle re-entry, and differentiation of cardiac progenitors into cardiomyocytes or pacemaker cells in the heart, we can increase the efficacy of current regenerative treatments. This work was supported partly by the Undergraduate Biology Research Program with funds from the Office of the Provost.

GENETIC ANALYSIS OF CRISPR/CAS9 KNOCKOUT MUTANTS OF TRANSCRIPTION FACTOR GENES INVOLVED IN THE DIFFERENTIATION OF THE BASAL TRANSFER CELL LAYER OF MAIZE ENDOSPERM

***RYAN FINNEL*, CHOONG-HWAN RYU, KAITLYNN GOFF, RAMIN YADEGARI**

The endosperm is a nutritive structure in the maize kernel and supports embryogenesis or seedling development upon germination. The basal transfer cell layer (BETL) of the endosperm mediates sugar and nutrient transport from the maternal tissue into the developing endosperm. Previous studies indicated that a clade of MYBR type transcription factor proteins, including MYBR33 and MYBR81, are likely involved in BETL differentiation within the first 4-6 days after fertilization. To understand the function of MYBR genes, we used a CRISPR/Cas9 system to induce loss of function mutations in MYBR33 and MYBR81 genes. Here we report on the nature and transmission of the engineered mutant alleles through two generations. Further work is focused on generation of additional alleles and analysis of any associated endosperm phenotypes.

THE TYPE SIX SECRETION SYSTEM IN THE ENTOMOPATHOGENIC BACTERIUM *XENORHABDUS BOVIENII* (ENTEROBACTERIACEAE) PLAYS A KEY ROLE IN BACTERIAL COMPETITION

***ISABEL FORLASTRO*, REBECCA MCQUADE-KOCHANOWSKI, CHRISTINE M. BRADSHAW, S. PATRICIA STOCK**

The Type Six Secretion System (T6SS) is a molecular mechanism employed by multifarious gram-negative bacteria to inject effector proteins into a target cell. T6SSs have been shown to play key roles in pathogenesis with eukaryotic cells, and in competition with other bacteria. *Xenorhabdus bovienii* strain *jolietti* (XBJ) is an entomopathogenic gram-negative bacteria that encodes two separate T6SSs. XBJ exhibits an unusual dual-lifestyle: living in a mutualistic relationship within the intestine of *Steinernema* nematodes, while also being pathogenic to a wide variety of insects and associated microbes. Since much is unknown about these T6SSs in XBJ, we generated various XBJ mutants lacking the key structural protein Hcp from each T6SS and assessed the role of this secretion system in interbacterial competition. We also conducted intra haemocoel injections using the fifth-instar larvae of the insect model *Galleria mellonella* to assess if our Hcp mutants had an effect on insect virulence. Here we demonstrate that one of the T6SSs is more highly expressed in culture and has antimicrobial activity against other *Xenorhabdus* strains, while neither play a significant role in insect pathogenesis.

FUNCTIONALLY ACTIVE G-PROTEIN-COUPLED RECEPTORS ARE SOLVENT-SWOLLEN

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G-protein-coupled receptors (GPCRs) are transmembrane proteins that play critical roles in a number of physiological signaling processes. In the standard biochemical model, GPCRs behave as primarily agonist-dependent bimodal switches, with little influence of the surrounding medium. However, using the visual receptor rhodopsin as a model GPCR, we show that water drives rhodopsin to a partially disordered, solvent-swollen conformational ensemble upon light absorption, rendering the standard model obsolete. We placed rhodopsin under varying degrees of osmotic stress using polyethylene glycol solutes and investigated the activation equilibrium response using UV-visible spectroscopy. Our results show a flood of ~80 water molecules into the rhodopsin interior during photoactivation, a result supported by atomistic MD simulations [1]. Furthermore, the osmolyte effects on rhodopsin activation are size-dependent: large osmolytes back shift the equilibrium to inactive metarhodopsin-I (MI), while small osmolytes forward-shift the equilibrium to active metarhodopsin-II (MII). We attribute these size effects to varying degrees of osmolyte penetration into the rhodopsin core. Large polymers behave similarly to ideal osmolytes and dehydrate rhodopsin, while smaller polymers wriggle into the rhodopsin interior and stabilize the open MII conformation of rhodopsin. Besides osmotic pressure, the application of hydrostatic pressure also back shifts the metarhodopsin equilibrium but for fundamentally different reasons. Integrating the two force-based methods together with neutron scattering experiments [2] indicates that the active state of rhodopsin is more hydrated yet more locally collapsed. At the same time, the active GPCR undergoes volume fluctuations and solvent coupling, which give rise to greater thermal volume. The combined force-based results necessitate a new understanding of GPCR activation in which the surrounding soft matter is paramount in governing conformational energy landscapes. This work was supported by the Undergraduate Biology Research Program with funds from the Office of the Provost and the College of Science, and by grants EY026041 from The National Institutes of Health (NIH) and CHE 1904125 from the National Science Foundation. [1] N. Leioatts et al. (2014) Biochemistry 53, 376?385. [2] S.M.D.C. Perera et al. (2018) J. Phys. Chem. Lett. 9, 7064-7071.

INVESTIGATING THE ROLE OF SULFATIDES IN THE CHRONIC INFLAMMATORY RESPONSE TO STROKE

***FRANKIE GARCIA*, KYLIE CALDERON, JACOB ZBESKO, DANIELLE BECKTEL, RACHAEL HEARNE, THUY-VI V. NGUYEN, KRISTIAN P. DOYLE**

Following an ischemic stroke, there is a chronic inflammatory response within the damaged part of the brain as the immune system breaks down the lipid-rich myelin sheath of dead neurons. This chronic inflammatory response causes secondary neurodegeneration to the tissue surrounding the area of damage and contributes to post-stroke cognitive decline in animal models of stroke. A major component of myelin is the sulfated galactolipid, sulfatide. In other neurological disorders, sulfatides have been found to be pro-inflammatory and neurotoxic. We hypothesize that following stroke the breakdown of myelin results in the accumulation of sulfatide within the infarct, and that sulfatide is a primary driver of the chronic inflammatory response to stroke. To test this hypothesis, we quantified sulfatide accumulation in the brain following stroke using the histological dye Alcian blue. Alcian blue staining revealed that sulfatide accumulates and persists within stroke infarcts for at least 7 weeks following stroke. We have also tested whether the administration of β-cyclodextrin, a lipid solvate, can prevent sulfatide from accumulating within stroke infarcts in the weeks following stroke. Mice were treated three times a week for six weeks with 4g/kg of cyclodextrin beginning one week following stroke. Cyclodextrin treated mice had significantly reduced neutral triglyceride and lipid levels within their infarcts, however there were no differences in sulfatide levels. We have also tested whether antibodies that are produced within chronic stroke infarcts are part of an endogenous sulfatide clearance mechanism. Alcian blue staining on brain tissue from transgenic muMt mice, which lack mature B-cells, and wildtype mice, revealed no differences in sulfatide levels between the transgenics and controls. We are currently in the process of determining how a drug that alters myelin metabolism impacts sulfatide levels in the brain following stroke. With these experiments we are furthering our knowledge of how the sulfatide component of myelin debris is processed following stroke, and the extent to which it drives the chronic inflammatory response to stroke. This research was supported in part by the Western Alliance to Expand Student Opportunities (WAESO) Louis Stokes Alliance for Minority Participation (LSAMP) National Science Foundation (NSF) Cooperative Agreement No. HRD-1101728 and by the Undergraduate Biology Research Program with funds from the Office of Research, Innovation & Impact.

MODELING T CELL AND VASCULAR INTERACTIONS IN VITRO

***ALANA GONZALES*, SARAH SHELTON, ROGER KAMM**

Immunotherapy drugs have become a widespread cancer treatment. Researchers are developing methods to predict patient-specific responses, but these have not examined the influence of the vascular endothelium in immune cell migration to tumors. We aim to model this interaction using in vitro systems by evaluating T cell migration and adhesion to human endothelial cells (ECs). Initial 2D experiments used conditioned media from four cancer cell lines to provide cytokine stimulus. Porous membranes were used to quantify T cell migration in response to a cytokine gradient, while adhesion was measured by treating EC monolayers with conditioned media prior to incubation with T cells. There was a significant difference in T cell adhesion across the four cell lines (p=1.8105) and in T cell migration in response to cytokine gradients (p=2.5103). Results showed a nearly 3-fold increase in T cell adhesion to ECs (p=3105) and a 3-fold increase in T cell migration (p=0.0017) in response to MDA-MB-231 conditioned media compared to the negative controls. Therefore, this model demonstrates that cytokine stimulus affects T cell migration and adhesion to EC monolayers. Future experiments aim to examine migration and adhesion profiles in more detail using vascularized 3D microfluidic devices to model the tumor microenvironment. Funding for this project was provided by the National Institutes of Health (NIH) Maximizing Access to Research Careers (MARC) Training Grant T34 GM08718, National Institutes of Health (NIH) under award numbers U01CA214381 and K00CA212227, and the Massachusetts Institute of Technology Summer Research Program.

TARGETING THE PANCREATIC β-CELL VIA GLP-1 AND KISSPEPTIN-10

***EMILY HARNOIS*, RONALD LYNCH, CRAIG WEBER**

In all forms of Diabetes Mellitus, the insulin secreting cells of the pancreas gradually lose function and are eventually destroyed. The diseased state arises secondary to autoimmune dysfunction (Type I) or as a result of chronic hyperglycemia/hyperinsulinemia and eventual insulin resistance (Type II). Several drugs have been shown to improve pancreatic β-cell survival and thereby insulin secretion, however off-target side effects often limit use at their effective dosages. For example, we recently showed that Kisspeptin-10 (Kiss-10) has positive effects on β-cell mass, and also supports glucose stimulated insulin secretion (GSIS). However, these effects occur only at μM concentrations which are much higher than seen in the blood. Our lab proposed that linking Kiss-10 to Glucagon-like-peptide-1 (GLP-1) which is active at nM concentrations, would allow Kiss-10 activity at nM concentrations. In order to determine if Kiss-10 is effective in activating β-cells within the Multivalent GLP-1/Kiss-10 construct, we first needed to ascertain Kiss-10's downstream signaling pathways relative to GLP-1. Both GLP-1 and Kiss-10 have been shown to potentiate GSIS from β-cells, and our lab has shown that these effects are additive suggesting these peptides act via different signaling mechanisms. The effects of Kiss-10 on several potential signaling mechanisms including cyclic-AMP, Ca2+ signaling and metabolic responses were analyzed. GLP-1 is known to activate a Gs pathway thereby elevating cAMP in β-cells, however Kiss-10 had no effect on cAMP formation, supporting the proposal that they work through different pathways. Next, I evaluated Ca2+ signaling and neither GLP-1 or Kiss-10 has any effect on β-cell Ca2+ indicating neither peptide modulates the Gq pathway. Metabolic signaling was evaluated by imaging NADH production or monitoring oxygen consumption. Kiss-10 was found to increase mitochondrial NADH and also likely oxygen consumption within pancreatic islets β-cells, however these studies are not currently completed. These results indicate that Kiss-10 may stimulate β-cells via activation of metabolic pathways, as opposed to GLP-1 which activates Gs mediate pathways. Current studies are investigating the effect of GLP-1/Kiss-10 on GSIS, with specific focus on the activation of metabolic activity promoted by binding of the GLP-1 element at ηM concentrations. Funding for this project was provided by the American Diabetes Association and Juvenile Diabetes Research Foundation, and the Undergraduate Biology Research Program with funds from the Office of the Provost and the College of Medicine.

IDENTIFICATION OF TREATMENT PARAMETERS THAT MAXIMIZE LANGUAGE TREATMENT EFFICACY FOR CHILDREN

***CARLY HARRIS*, ELENA PLANTE**

Development language disorder (DLD) is a prevalent problem in children and requires early and thorough intervention to best manage. DLD is characterized as a delay in language learning in either production, reception, or both. It can cause difficulties in the children's academics and future careers, especially if it is not recognized and treated from early childhood. This study focuses on the positive effects of recast therapy for children with DLD, both in long input and short input format. The participants were 16 children with DLD aged four and five. Ten of the participants were male and six were female. The children took part in classroom activities such as crafts and educational linguistic games, as well as participated in 30 to 45 minutes of recast therapy five days a week for six weeks. Half of the participants were randomly assigned to a condition where the clinician recast the child's utterance in three words or less (short). The others were assigned to a condition where the clinician recast in five words or more (long). Probes took place three times a week to test the children's progress in their target utterance. The goal was to determine if short or long recasts were more effective for language development. The findings showed that both recasting conditions had a positive effect on the children, but more evidence is necessary to conclude whether short or long recasts are more effective in children's language development. Funded through The National Institute on Deafness and Other Communication Disorders of the National Institutes of Health under award number R01DC015642-01 and the Undergraduate Biology Research Program with funds from the Office of the Provost and the College of Science.

THE ROLE OF TMEM184B IN THE MAPK AND STAT-3 PATHWAYS IN RELATION TO NEURODEGENERATION

***HANNAH HART*, MARTHA BHATTACHARYA**

This lab is investigating how our protein of interest, a transmembrane protein named TMEM184b, interacts with MAPK and STAT-3 pathways within hippocampal lysates. This is to see TMEM184b's possible role in neurodegeneration, based on previous data suggesting it is pro-degenerative in axons in neuronal cells, and likely necessary for proper degeneration of unhealthy neurons. We performed Western blots in both wild type and mutated mice (TMEM184b GT/GT) to see if there were different levels of expression or activity for MAPK and STAT-3 pathways. The results, which some will be shown, give rise to various interactions between TMEM184b and the MAPK and STAT-3 pathways, but not significantly. There are many factors that could contribute to this. Ultimately, there needs to be more data and understanding of the molecular mechanism behind TMEM184b before there can be any conclusions. We thank the National Institutes of Health (NIH) under award number R01NS105680, the Arizona Technology and Research Initiative Fund (TRIF), and the Undergraduate Biology Research Program with funds from the BIO5 Institute and the Office of the Provost for their funding.

TESTING VALENCE USING REAL TIME PLACE PREFERENCE

***TAHIA HASNEEN*, HAIJIANG CAI, MATTHEW SCHMIT**

Protein Kinase C Delta (PKC-δ) neurons are GABAergic cells that are located in the lateral part of the Central Amygdala (CeA) that are involved in inhibiting feeding. Activating these neurons reduces total food intake in both fasted and fed animals and silencing them increases food intake in fed animals. However, they are activated during feeding in fasted mice when they approach food, which suggests they may be associated with the pleasure of eating. Therefore, we need to understand the valence of PKC-δ+ neuron activation. Valence refers to something that is pleasurable (positive valence) or aversive (negative valence). In order to test this, we used a real time place preference test (RTPP). It consists of a two-chamber system to test the mice's aversiveness to photostimulation. Each chamber has different visual cues on the walls, except in one of the chambers the stimulation is on, while the other it is off. They are able to move freely between the two chambers. By measuring the amount of time the mice spend in each chamber, we can infer whether the stimulation is aversive or rewarding. We placed

each mouse into the chamber without any photostimulation for 10 minutes to test their memory of previous tests. Then, we tested their preference for 15 minutes with the photostimulation. We hypothesized that activating the neurons will cause an aversion for the Channelrhodopsin (ChR) mice compared to the EYFP mice. We tested three different conditions: fed mice and 24 hour fasted mice. ChR mice exhibited significant place preference for the non-photostimulation paired chamber, compared to the control mice. Interestingly, ChR mice who were fasted for 24 hours, showed very little aversion to the previously photostimulation paired chamber during the 10 minutes (without any photostimulation) and they did not show any preference between the two chambers. Further exploration of valence could include only comparing fasted mice with fed mice without any addition of injections and to see how it affects their aversiveness to the 10Hz photostimulation. This work is supported by the National Alliance for Research on Schizophrenia & Depression (NARSAD) Young Investigator Award from the Brain and Behavior Research Foundation, the Foundation for Prader-Willi Research, and the Klarman Family Foundation.

IN DEFENSE OF THE "ENTOURAGE EFFECT": TERPENES FOUND IN *CANNABIS SATIVA* ACTIVATE THE CB1 RECEPTOR IN VITRO AND IN VIVO

***RYAN HECKSEL*, JUSTIN LAVIGNE, JOHN M. STREICHER**

Marijuana has been understudied for decades, primarily due to social stigma and legal restrictions. However, as legal restrictions begin to loosen among states, the potential medical benefits and pharmacological properties of marijuana are beginning to be explored. Terpenes, an expansive group of organic chemicals that impart odor and taste, are found in the *Cannabis sativa* plant and may work synergistically with cannabinoids, such as THC and CBD, in a term deemed the entourage effect. Anecdotally among the recreational and medical use community, terpenes have been reported to enhance the potency and physiological effects of marijuana. However, scientific evidence for the entourage effect is very limited. To evaluate this hypothesis, we obtained the *C. sativa*-relevant terpenes: b-pinene, a-humulene, geraniol, and linalool. Utilizing Chinese hamster ovary cells (CHO) expressing the human cannabinoid receptor type 1 (CB1, CB1-CHO) we screened these terpenes for CB1-dependent phosphorylation of extracellular signal-regulated kinase 1/2 (ERK1/2), a well-known downstream target of CB1 activation, using Western blot. We observed that pERK levels were efficaciously stimulated by all four terpenes when compared to positive control, the selective CB1 agonist WIN 55,212-2. These results appeared to be CB1-dependent, as pre-treatment of these cells with a selective CB1 antagonist, rimonabant (SRI141716), blocked ERK phosphorylation by each of the terpenes. We further verified the CB1-dependent nature of these effects by examining ERK phosphorylation by the terpenes in wild type CHO (WT CHO) cells, which do not express the CB1 receptor. In these cells, b-pinene and a-humulene treatment resulted in ERK phosphorylation while linalool or geraniol treatment did not. In WT CHO cells, the ERK phosphorylation induced by b-pinene and a-humulene was not CB1-dependent, as rimonabant pre-treatment did not block it. These results thus suggest that geraniol and linalool could be CB1-selective agonists, whereas b-pinene and a-humulene are non-selective and may also activate one or more receptors besides CB1. In vivo, we have found that intraperitoneal administration of these terpenes results in behaviors (hypothermia, analgesia, catalepsy, hypo-locomotion) consistent with traditional CB1 agonists. Follow-up studies will examine other measures of CB1 activity (binding, Barr2 recruitment, cAMP signaling, GTPγS coupling) to characterize the binding and functional properties of these terpenes at the CB1 receptor, as well as identifying the other targets of b-pinene and a-humulene. Once we have characterized these terpenes individually, we aim to investigate their role in the entourage effect, by testing their modulation of typical cannabinoid (THC, etc.) pharmacology, both in vitro and in vivo. Translationally, these findings could have implications in marijuana cultivar breeding and could help produce strains optimized for specific terpene profiles, which could be more efficacious for chronic pain management and other therapeutic uses. This study was supported by institutional funds from the University of Arizona. RH would like to thank the American Society for Pharmacology and Experimental Therapeutics (ASPET) Summer Undergraduate Research Fellowship and the College of Pharmacy for summer fellowship funding. JMS has received research funding from Botanical Results, LLC, a company that develops cannabidiol products (not related to current study).

MICROBIAL SYMBIONTS OF AN INVASIVE GRASS DIFFER IN URBAN AND EX-URBAN ENVIRONMENTS

***VICTORIA HOWARD*, A. ELIZABETH ARNOLD**

Cenchrus ciliaris (buffelgrass) is a widespread, invasive plant in the Sonoran Desert. It establishes readily, weathers drought, alters fire regimes, and is costly and labor-intensive to eradicate. Investigating microbial symbionts of buffelgrass may identify factors that promote its establishment and spread. We examined fungal microbiomes associated with buffelgrass in urban areas (alleyways with poor soil and limited plant cover) and ex-urban areas (sites with natural soil and vegetation) in and near Tucson, AZ. We isolated fungal endophytes from healthy roots and shoots and characterized them via DNA barcoding. We found that endophytes of buffelgrass were more abundant in urban areas, but more diverse in ex-urban areas. Endophyte communities differed between urban and ex-urban sites. Our data suggest context-specific symbioses in which buffelgrass recruits distinctive microbiomes under different environmental conditions. In future research, we will identify plant-microbe interactions that influence the fitness, germination, and stress tolerance of buffelgrass as an invasive plant. This research was sponsored by the Undergraduate Biology Research Program through the Office of Research, Innovation & Impact and the College of Agriculture and Life Sciences at the University of Arizona, as well as private donors.

EFFECTS OF ALPHA-SYNUCLEIN OVEREXPRESSION ON TIME SPENT SINGING IN ZEBRA FINCHES

***NAYA IBRAHIM*, EDDIE VARGAS, CESAR A. MEDINA, STEPHANIE MUNGER, JULIE E. MILLER**

Parkinson's disease (PD) is a neurodegenerative disease that impairs motor control. The neuropathology includes abnormal protein expression of the gene, SNCA (alpha-synuclein), at synapses and in neuronal cell bodies. Previous evidence suggests that changes in voice and speech are early symptoms of the disease, occurring prior to some of the later emerging limb motor deficits. In addition, recent research has shown that overexpressing the human SNCA gene in mice models some of the vocal deficits seen in the human disease. However, the basal ganglia brain circuitry for vocal behavior in mice has not been well mapped. Therefore, to understand the molecular and cellular brain mechanisms responsible for the vocal deficits induced by alpha-synuclein overexpression, we use songbirds. Songbirds have a well-described brain circuit for song that is similar to human speech. I hypothesize that overexpressing alpha-synuclein in a region of the bird basal ganglia dedicated to song, Area X, will cause song deficits similar to speech deficits seen in most cases of PD. To test our hypothesis, a viral vector containing the human wild-type alpha-synuclein gene is injected into Area X. As a first step, I quantified the average motif duration for alpha-synuclein virus-injected finches and control (GFP) injected finches, where a motif is considered as a repeated sequence of syllables separated by silent periods (80-120 ms). Motif duration and time spent singing over a two-hour period across multiple mornings for each finch was then quantified and compared at pre and post-injection monthly time points. Motif duration refers to the mean of 93 motifs across three days per bird and time spent singing is calculated by multiplying motif duration by the total number of motifs over the two-hour period of recording. My findings indicate no significant differences between groups for these measures. Our lab group did find that a related measure, the total amount of singing over a two-hour period, did decrease in alpha-synuclein injected birds at two months post-injection meaning the birds sing less (see Vargas et al. UBRP abstract). Future directions will determine if aggregated alpha-synuclein is present in Area X to determine if this correlates with changes in song duration and amount given that aggregation is a major hallmark of human PD. Results will contribute to our understanding of the proposed early-stage vocal symptoms in PD. Acknowledgment: the National Institutes of Health (NIH) Maximizing Access to Research Careers (MARC) Training Grant T34 GM08718.

HOW NICOTINE EXPOSURE DURING THE FETAL AND NEONATAL PERIODS ("DEVELOPMENTAL NICOTINE EXPOSURE" OR DNE) ALTERS THE STRUCTURAL AND FUNCTIONAL DEVELOPMENT OF THE HYPOGLOSSAL MOTOR NEURONS (XIMNS)

***OSAGIODUWA IGBINOBA*, NIK RODRIGUEZ, AMANDA GONG, EMILY FLANIGAN, SERES BENNETT, LILA WOLLMAN, RALPH FREGOSI**

About 500,000 infants are born to tobacco smoking mothers who smoked during and/or after pregnancy in the U.S. each year. The rise of nicotine exposure through e-cigarettes, nicotine patches, and nicotine gum among pregnant women and their offspring is a crisis. Prenatal nicotine exposure, with continued exposure through breast milk disrupts the growth and function of hypoglossal motoneurons (XIIMNs), which play a critical role in key functions such as breathing, swallowing, suckling and mastication. Through immunohistochemistry techniques, neurotransmitter signaling, and receptor expression is examined in neonates. Our goal is to determine how nicotine exposure during the fetal and neonatal periods (developmental nicotine exposure or DNE) alters the structural and functional development of the hypoglossal Motor Neurons (XIMNs). This is done using various techniques that focus on how DNE alters neurotransmitter signaling and receptor expression. These include Gaba and Glycine inhibitory receptors, and excitatory Glutamate receptors. To examine receptor expression, we use immunohistochemistry. The main techniques include Transcardial Perfusions of paraformaldehyde to fix the brain, dissecting the brain, and using a Vibratome to cut 60 micron-thick slices of the brainstem, that are then mounted on microscope slides and exposing them to various stains and/or antibodies. Lastly, XIMNs are observed and analyzed based on the staining intensity using microscopes and software. Analysis of stained slides include dendritic length, surface area, and number of intersections formed and receptor density. This research was supported in part by the Undergraduate Biology Research Program with funds from the Office of Research, Innovation & Impact and the College of Medicine, the National Institutes of Health (NIH) under award number R01HD071302, and by the Western Alliance to Expand Student Opportunities (WAESO) Louis Stokes Alliance for Minority Participation (LSAMP) National Science Foundation (NSF) Cooperative Agreement No. HRD-1101728.

BIOPHYSICAL NATURE OF INTERACTIONS BETWEEN NS1 AND SP1

***CHRISTA IMRICH*, NILOOFAR GHADIRIAN, NANCY HORTON**

Human parvovirus B19 is a member of the family *Parvoviridae* and is pathogenic in humans, resulting in conditions including hydrops fetalis, erythema infectiosum, and rheumatoid arthritis. While most cases of B19V infection are mild and pass without complication, a subset of infections lead to life-threatening conditions. Nonstructural protein 1 (NS1) of B19V plays a variety of roles throughout viral replication and has been shown to upregulate the function of the P6 promoter, the functional promoter in the viral genome. Human specificity protein 1 (Sp1) has been implicated in viral replication and has been shown to bind the P6 promoter, though its role has not been fully characterized in literature to date. The goal of this study is to elucidate information about potential biomolecular interactions between NS1 and Sp1. Recombinant expression of hMBP/NS1 and GST/Sp1 in *E. coli* has been confirmed. Subsequently, a series of affinity-based purification techniques were employed to obtain pure samples of glutathione S-transferase (GST)-tagged Sp1 and histidine and maltose binding protein (hMBP)-tagged NS1. Following the successful purification of NS1 and Sp1, a series of pull-down assays will be performed to detect the potential binding of these proteins. Determining the biophysical nature of interactions between these proteins, if any, could have implications in control of the viral P6 promoter and would further our understanding of viral infection. Acknowledgement of funding: the National Institutes of Health (NIH) Maximizing Access to Research Careers (MARC) Training Grant T34 GM08718.

INNOVATION IN BUMBLEBEES IS AFFECTED BY ENVIRONMENTAL COMPLEXITY, BUT NOT RESPONSIVENESS

***JAK KEARSLEY*, DAVID KIKUCHI, TIMOTHY POLNASZEK, ANNA DORNHAUS**

Innovation enables animals to either exploit new opportunities or find new ways to take advantage of old ones. Foraging innovations give animals novel ways of accessing resources in their environment. Innovation propensity could be influenced by internal factors such as linked behavioral traits, or by external factors such as the conditions and arrangement of their environment. We tested the effects of the visual and spatial complexity of a foraging environment and an individual's tendency to explore novelty (responsiveness) on feeding innovation in bumble bees (*Bombus impatiens*). We administered a series of foraging trials in either a simple or complex environment that required bees to access novel flowers to obtain a sucrose reward. We used survival analysis to test whether they were able to solve the flower and if so, how long it took them, depending on the environmental complexity treatment and their individual responsiveness. In two out of four of the novel flower trials, bees in the simple environment were more likely to solve the flower while in the other half there was no effect from environmental complexity. We found no effect of responsiveness on ability to solve flowers in any of the trials. These findings suggest that in general, less complex foraging environments allow for innovation in accessing novel flowers, but an individual's responsiveness to the environment is not related to their innovation rate. Thank you to the National Science Foundation for funding to make this project possible.

CHARACTERIZATION OF POTASSIUM (K+) CONDUCTANCES THAT MAY CONTRIBUTE TO Aβ-INDUCED BASAL FOREBRAIN CHOLINERGIC NEURONAL (BFCN) HYPEREXCITATION

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Alzheimer's disease (AD), a progressive neurodegenerative disorder, afflicts over five million Americans and is characterized by gradual cognitive decline and mental deterioration. The degree of cognitive impairment correlates with the loss of basal forebrain cholinergic neurons (BFCNs), the first to deteriorate in AD. BFCNs serve as the major source of cholinergic transmission to the neocortex, hippocampus, and amygdala and mediate cognitive processing and memory acquisition. While the specific mechanism by which BFCNs are disrupted is not well established, previous studies indicate that interactions between soluble oligomeric amyloid-beta (Aβo) and nicotinic acetylcholine receptors (nAChRs) containing α7 and β2 subunits (α7β2 nAChRs) in BFCNs, which mediate synaptic transmission and neuronal excitability, lead to neuronal functional instability. Here, we demonstrate that Aβo/nAChR interactions contribute to deficits in spatial reference memory in the APP/PS1 mouse model of AD using Morris water maze testing: APP/PS1 mice genetically null for the β2 nAChR subunit gene (APP/PS1-β2KO) showed an improvement in latency and distance swum to platform, time spent in the platform quadrant, and day-to-day memory acquisition when compared to APP/PS1 mice. These results demonstrate a causative role of Aβo/α7β2 nAChR interactions in spatial reference and overnight memory retention. Using ex vivo slice electrophysiology, we demonstrate that Aβo interacts with α7β2-nAChRs, inducing BFCN hyperexcitation. This is mediated in part by action potential afterhyperpolarization (AHP), which can be normalized through pharmacological or genetic alteration. These data support our hypothesis that, in AD, Aβo/ α7β2 nAChR interactions directly alter BFCN excitability by modulating K+ channel function, resulting in BFCN hyperexcitation. We used well-established K+ channel pharmacology and whole-cell current-clamp recordings to identify medial septal/diagonal band (MSDB) BFCN K+ channel subtypes that contribute to distinct phases of AHP. We show that BFCNs exposed to charybdotoxin (large conductance [BK] Ca2+-activated K+ channel antagonist) or apamin (small conductance [SK] Ca2+-activated K+ channel antagonist) exhibited attenuated medium AHP (mAHP) magnitude and altered spike frequency. Additionally, BFCNs exposed to charybdotoxin showed enhanced spike half-width, indicating a potential role of BK channels in BFCN repolarization. Altogether, this is a first step toward understanding the role of K+ channels that potentially mediate BFCN instability resulting from Aβo/nAChR interactions. Special thanks to the American Society for Pharmacology and Experimental Therapeutics (ASPET) Summer Undergraduate Research Fellowship and the College of Pharmacy for their funding in this project.

FORMING FILAMENTS WITH ENZYMES: A POTENTIAL MECHANISM FOR PLANTS TO ADJUST TO STRESSFUL ENVIRONMENTAL CONDITIONS

***DONG KYUN KIM*, NYSSA MORGAN, FRANS TAX**

The general topic of our research is how plants respond to environmental stress. The focus of this study is on how plant roots respond to different concentrations of nitrogen. Through a collaboration with a proteomics group, we identified around fifty proteins that interact with XIP1/CEPR1, a receptor protein that is involved in sensing nitrogen levels and regulating lateral root growth in *Arabidopsis*. Of these interactors, we selected ten that we hypothesize are important for lateral root growth. A recent study of some of these proteins involved in sugar and amino acid metabolism in *Arabidopsis* and other organisms were found to be in unexpected, highly-concentrated structures called filaments. Filaments are structures that are made from multimers of the enzymes, sometimes forming large helices. These filaments can be visualized as foci when the proteins are made as translational fusions to Green Fluorescent Protein. Although there have been studies on these filaments, little is known about the formation and function of these structures. One interactor protein that has been analyzed for filament formation is Phosphofructokinase (PFK). This enzyme forms filaments in *Drosophila*, human cells, and plants, showing that this formation is evolutionarily conserved and potentially important for metabolic activity and growth. We want to understand how our receptors may function in filament formation. To better understand these interacting proteins, we created transformation constructs using cDNAs from the SALK database. Using Gateway Cloning, we created plasmids with the 35S Promoter region driving the expression of the protein of interest which is translationally fused to Green Fluorescent Protein. We then transformed these constructs into Agrobacterium and into *Arabidopsis* plants. To understand how the receptors might regulate filament formation, we inserted these constructs into the wild type and XIP1/CEPR1 and CEPR2 double mutant *Arabidopsis*. Next, we will test whether these enzymes form filaments in mutants and in wildtype controls, test if filaments affect enzyme function, and whether phosphorylation of these enzymes results in filamentation. Due to climate change, it is becoming more important to understand how plants would respond to different environmental stresses like low nitrogen concentration, temperature stress, and oxidative stress. We hypothesize that enzyme filamentation could be a mechanism for plants to rapidly start and maintain a high metabolic state or for plants to lower their metabolic activity, in the case of stress under low nitrogen concentrations. Acknowledgements: I would like to acknowledge the Tax lab, and the Western Alliance to Expand Student Opportunities (WAESO) Louis Stokes Alliance for Minority Participation (LSAMP) National Science Foundation (NSF) Cooperative Agreement No. HRD-1101728 (NM) for providing funding and support for this project. Also, I would like to acknowledge the Undergraduate Biology Research Program (UBRP) with funds from the BIO5 Institute for this research opportunity.

NEST ARCHITECTURE OF SOCIAL PSEUDOSCORPIONS

***ANNA KITTLE*, ANNA DORNHAUS, KENNETH JAMES CHAPIN**

Social animals may build nests together to minimize the energy required for nest construction. *Paratemnoides elongatus* is a species of social arachnid that live in colonies and create silk nests containing multiple cells in which they molt. As such, we hypothesize that *P. elongatus* build nests collectively to minimize silk use, thereby maximizing nest construction efficiency. We measured the number of cells, perimeter, area, and roundness of 74 nests. We found that nests with more cells had higher overall area but lower roundness. Pseudoscorpions benefit from building social nests to reduce silk use to maximize silk use efficiency. Funding was provided by the American Arachnological Society and the National Science Foundation grants IOS 3014230 and ABI 3019760.

PRE-TRANSPLANT BENDAMUSTINE CONDITIONING INDUCES GRAFT-VERSUS-LEUKEMIA EFFECT WITH LOWER GRAFT-VERSUS-HOST DISEASE THAN CYCLOPHOSPHAMIDE IN MURINE MODELS

***NICOLE KUMMET*, EMELY A. HOFFMAN, JESSICA STOKES, MEGAN S. MOLINA, EMMANUEL KATSANIS**

Our laboratory has previously shown that pre-transplant conditioning with bendamustine (BEN) and total body irradiation (TBI) significantly reduces graft-versus-host disease (GvHD) when compared to cyclophosphamide (CY) and TBI in a major histocompatibility complex (MHC) mismatched murine bone marrow transplant model. Despite low GvHD, we show that conditioning with BEN + TBI induces GvL effects in mice with A20-Luc, an acute lymphoblastic leukemia transfected with luciferase. By measuring the bioluminescence of the tumor cells, we are able to view and quantify tumor progression, which is tracked along with GvHD scores. To determine which cells are responsible for the GvL effect seen with BEN-TBI, we depleted CD4+ and CD8+ T-cells, and NK cells. We see the greatest deficit in anti-tumor effect when CD8+ T-cells are depleted, followed by CD4+ T-cells, with depletion of NK cells showing little change to GvL effects. This suggests that CD8+ T-cells primarily contribute to the GvL effect seen in this model. These results indicate that BEN-TBI pre-transplant conditioning may be a safer and more effective conditioning regimen due to increased GvL effect and reduced GvHD. Funding provided by the Margaret Bilson Endowment, the Leukemia and Lymphoma Society, and People Acting Now Discover Answers (PANDA).

POSITIVE FEEDBACK LOOPS IN RB-E2F PATHWAY UNDERLIE ULTRA-SENSITIVITY IN DNA DAMAGE INDUCED CELL CYCLE ARREST

***AMELIA LAPPENBUSCH*, KOTARO FUJIMAKI, GUANG YAO**

Most cells in the human body are in a reversible, non-dividing state called cellular quiescence. Quiescence is an essential part of maintaining tissue homeostasis through regulating repair. When given access to growth signal, these cells can re-enter the cell cycle to continue growth and replace damaged cells. One factor in whether a cell will re-enter the cell cycle is the level of DNA damage to the cell, which arrests cells allowing them to repair damage before they continue their growth. In some cases, the machinery which regulates DNA damage induced arrest can become dysregulated. There is little quantitative knowledge of how this mechanism functions, or how it could be altered. Here we show that we were able to modulate DNA damage induced arrest by altering specific targets within the Rb/E2F and the DNA damage response pathways. Mathematical modeling of DNA damage response and the Rb/E2F pathway predicted the cell response to DNA damage exhibits a threshold at which cells arrest. We tested this prediction experimentally by re-stimulating quiescent cells exposed to ultraviolet light or radiomimetic reagents at various doses. We found that the cells arrest in an ultra-sensitive manner in response to DNA damage, consistent with the model. The model also predicts that the positive feedback loops within the Rb/E2F pathways are necessary for the ultra-sensitivity of DNA damage induced arrest. By pharmacologically targeting proteins which affect the positive feedback loop, we observed reduction/elimination of the ultra-sensitivity. Our results show that DNA damage induced arrest can be controlled, suggesting that further exploration in modification of Rb-E2F and/or DNA-damage-response pathways could potentially resolve DNA damage arrest dysregulation seen in aged and cancerous cells. This research was supported by the Undergraduate Biology Research Program with funds from the Office of the Provost and the College of Science, as well as the Yao Laboratory.

COMPUTATIONAL DESIGN OF SPLIT-PROTEINS

***DAVID LASANSKY*, BISMARK AMOFAH, INDRANEEL GHOSH**

Split-proteins systems represent a powerful tool for identifying interactions of a protein with DNA, RNA or another protein in living cells. These are known as protein-fragment complementation assays, and they rely on the use of a well characterized split-protein such as the green fluorescent protein or luciferase enzyme of which the N-terminal and C-terminal halves of the protein are attached to two supposedly interacting molecules. These molecules are often called the "bait" and "prey" since the

interaction of the bait with its prey will be recognized by a signal from the split-protein reforming into its native and active state. Our lab has also demonstrated the potential use for split-proteins as a method for controlling the activity of a specific protein in order to interrogate its signaling pathways when other methods are infeasible. These split-proteins were all created by identifying regions which are not crucial for the function and overall fold of the protein to facilitate the reformation of the native structure. However, finding the exact place to split a protein of interest remains a difficult problem to solve, and methods for identifying split-sites often require a trial and error approach in which numerous candidates must be generated. In order to streamline the design of split-proteins, our lab has developed a program which utilizes only the primary sequence data of a protein of interest and its homologs to find regions of low consensus at which a potential split-site may be present. The great wealth of sequence information which is now available allows for many new split-proteins to be designed computationally and rationally. This research is funded in part by the Undergraduate Biology Research Program with funds from the Office of Research, Innovation & Impact.

ASSOCIATIONS BETWEEN SOIL BIOAVAILABLE PHOSPHORUS AND GUAYULE GROWTH AND RUBBER PRODUCTION

***JESSICA LEDESMA*, COLLEEN MCMAHAN, DANTE PLACIDO, DIAA E. ELSHIKHA, CHEN DONG, GRISEL PONCIANO, LIA OSSANNA, RAINA M. MAIER, JULIA W. NEILSON**

Historically, the Brazilian *Hevea brasiliensis* rubber tree has been largely used as a source of natural rubber for commercial use. In recent years, the native desert shrub, Guayule (*Parthenium argentatum G.*), has been studied as a sustainable alternative for commercial production of natural rubber. However, because the shrub requires little nutritional amendments and is particularly drought tolerant, the mechanisms for maximizing rubber production are poorly understood. In particular, soil bioavailable phosphorus is of interest as phosphorus is a key constituent in vital plant mechanisms such as energy transfer and photosynthesis. Additionally, phosphorus is an important component of the rubber monomer for rubber particle biosynthesis. This research identifies associations between soil bioavailable phosphorus and Guayule growth stage, rubber production, overall plant growth, and soil DNA biomass. For this research, a Guayule field trial was conducted at the Maricopa Agricultural Center. Six different plants were harvested on three sampling dates representing different growth stages. These plant samples were quantified for rubber transferase activity, natural rubber content, and photosynthetic rate by the USDA-ARS-WRRC lab in Albany, CA. Additionally, soil samples were collected for chemical and microbial analysis of bioavailable phosphorus and soil DNA biomass to identify associations between microbial potential and bioavailable phosphorus concentrations. Bioavailable phosphorus was extracted using a modified Olsen P bioavailable phosphorus extraction method for arid soils. Correlations were identified between bioavailable phosphorus and plant growth stage, plant biomass, and rubber production. Understanding soil and plant phosphorus dynamics in relation to rubber production has implications for agricultural management strategies to maximize Guayule rubber production providing a viable and sustainable source of natural rubber for industrial use. Acknowledgements: This research was funded by the United States Department of Agriculture (USDA) grant # 2017-68005-2686; Sustainable Bioeconomy for Arid Lands. Opinions, findings, conclusion or recommendation expressed in this publication are those of the author(s) and do not necessarily reflect the views of the USDA.

A TRANSCRIPTOMIC ANALYSIS OF EPILEPTOGENESIS IN A KAINATE MODEL

***SARAH LESTER*, MICHAEL HAMMER, G. HERMEY, C. MAHLKE, J.J. GUTZMANN, J. SCHREIBER, N. BLUTHGEN, D. KUHL**

Epileptogenesis is the underlying pathological process that influences whether a single seizure develops into the recurrent seizures associated with chronic epilepsy. Past studies have been concerned with finding specific genes whose expression is altered after induced seizures. The issue with this approach is that it shows gene expression in a vacuum; it does not address how one gene can influence another and result in a given biological outcome. In reality, genes do not act by themselves, they work together in a pathway structure to accomplish a purpose. In this study, gene expression was quantified by comparing kainate-treated versus untreated control hippocampal RNA and genes that were activated or deactivated were analyzed using GSEA to determine which biological pathways are activated or deactivated. Therefore, this method represents a holistic

approach to determine what is influencing the tissue to become epileptogenic. We found that the most dramatic genetic effects are not necessarily immediately after the seizures, but up to 8 and 24 hours afterwards, with the 24hr time point having the most differentially expressed genes, followed by 1hr, 8hr, and 4hr. The 8hr time point had the largest number of altered pathways (accounting for only pathways that were expressed across multiple timepoints), followed by 24hr, 1hr, and 4hr. This research was supported in part by the Undergraduate Biology Research Program with funds from the Offices of the Provost and Research, Innovation & Impact.

GENERATION OF CELL-SPECIFIC RIBOSOME-BOUND MESSENGER RNA IN MOUSE OVARIES

***JASMINE LOCK*, ESTELA JAUREGUI, ZELIEANN CRAIG**

Phthalates are endocrine-disrupting chemicals (EDCs) heavily used in consumer products because of their ability to impart flexibility and durability. They are found in many products including latex adhesives, dyes, personal care products, and the coating of medications. Due to the fact that phthalates are not chemically bound to their host product, they are able to leak into the content of the material in which they are added; thus, exposing humans to their effects. One area of phthalate exposure that is of increasing concern worldwide is its effect on reproduction and fertility since phthalates have been associated with adverse reproductive outcomes. Most experiments studying the reproductive toxicity of phthalates have been focused on the ovary. However, due to the heterogenous nature of the ovary, there is a need to separate the ovary into its cell types in order to study specific cell types. In this project, experimental mice with isolated ribosome-associated mRNA transcripts were generated in three different types of ovarian cells: theca cells, granulosa cells, and oocytes. Using the Cre-lox system, RiboTag/Cyp19iCre-positive, RiboTag/Cyp17iCre-positive, and RiboTag/Zp3iCre-positive female mice were generated to investigate cell-specific gene expression in the granulosa cells, theca cells, or oocytes respectively. Genotyping using specific primers and electrophoresis to detect the presence of the Cre recombinase and the Human Influenza Hemagglutinin (HA) tag in the granulosa cells and oocytes was performed for each female experimental mouse. As expected, the genotyping confirmed that RiboTag/Cyp19iCre-positive, RiboTag/Cyp17iCre-positive, and RiboTag/ZP3iCre were successfully generated. Further studies involve using immunohistochemistry and immunoprecipitation to confirm appropriate targeting of the cells of interest. Funded by the Environmental Health Sciences – Transformative Research Undergraduate Experience (EHS-TRUE) through the National Institute of Environmental Health Sciences Grant #1-R25-ES025494.

IDENTIFYING NOVEL REGULATORS OF THE TORC1 SIGNALING NETWORK

***ERIC LU*, ARRON SULLIVAN, RYAN WALLACE, JACOB CECIL, XIANGXIA LUO, ANDREW CAPALDI**

The Target of Rapamycin Complex 1 (TORC1) is a highly conserved multimeric protein kinase complex that regulates cellular growth, proliferation and metabolism in eukaryotes. In the presence of growth signals and sufficient nutrients, TORC1 is activated and drives protein, lipid, and nucleotide synthesis. Conversely, when cells are starved for nutrients, TORC1 is rapidly inactivated to halt growth and drive entry into a quiescent state. Here, we identify several novel proteins which modulate the activity of the complex. We have previously shown that starvation conditions also cause TORC1 to move from around the vacuolar membrane to single foci at the edge of the vacuole. We determined a set of physically interacting proteins via immuno-purification and mass spectrometry. A deletion library was subsequently constructed from these identified interactors, and a reverse genetic screen was conducted. Fluorescence microscopy was used to follow TORC1 as it formed bodies under nitrogen starvation in each mutant strain. We identified five genes - YDL180W, MKS1, PBP1, SER33, and RTG3 which strongly inhibit TORC1 aggregation, and 15 additional genes which are necessary for these aggregates to form. These findings provide greater knowledge about the systems-level model of TORC1 regulation which we have previously described, as well as some insight into the purpose of reversible protein aggregation in response to starvation. This study was funded by the National Institutes of Health (NIH) under award number R01GM097329, and the Undergraduate Biology Research Program with funds from the BIO5 Institute.

REDUCING OXIDATIVE STRESS AS A MECHANISM BEHIND THE NEUROPROTECTIVE EFFECT OF PHOSPHOFRUCTOKINASE IN ALS

***MARIA MACIAS*, SUVITHANANDHINI LOGANATHAN, ERNESTO MANZO, GABE BIRCHAK, DANIELA C. ZARNESCU**

Amyotrophic lateral sclerosis (ALS) is a fatal neurodegenerative disease that affects motor neurons in the brain and spinal cord, thus impairing muscle function. Death occurs within 2-5 years of diagnosis and there currently is no cure. TAR DNA Binding Protein (TDP-43), an RNA binding protein, has been found in pathological aggregates of >97% of ALS cases. When expressed in *Drosophila* motor neurons, TDP-43 leads to the formation of cytoplasmic aggregates, causes locomotor deficits, abnormalities in the ventral nerve cord (VNCs), and reduced lifespan. Both ALS patients and animal models of ALS show evidence of free radicals suggesting that oxidative stress is involved in motor neuron death. Our lab has shown that glycolysis upregulation, specifically Phosphofructokinase-1 (PFK1) overexpression, rescues deficits caused by TDP-43. Pyruvate, the end result of glycolysis, has been shown to protect the mitochondria from oxidative stress by scavenging Reactive oxygen species (ROS). Our hypothesis is that overexpression of PFK mitigates motor neuron dysfunction through the antioxidant properties of pyruvate. We stained larval brains using dihydroethidium (DHE), which is a mitochondrial superoxide indicator and found that without PFK overexpression wild type and mutant TDP-43 had increased ROS levels. When PFK was overexpressed in the context of TDP-43 we saw a reduction in ROS levels, which have yet to be quantified. This research was funded by the Undergraduate Biology Research Program with funds from the Margaret Bilson Endowment, the National Institutes of Health (NIH) under award number RO1NS091299, the Muscular Dystrophy Association grant 418515, and the Western Alliance to Expand Student Opportunities (WAESO) Louis Stokes Alliance for Minority Participation (LSAMP) National Science Foundation (NSF) Cooperative Agreement No. HRD-1101728.

DEVELOPMENT OF MOBILE AND COST-EFFECTIVE CAPILLARY ELECTROPHORESIS INSTRUMENTATION

***JACOB MAPP*, CRAIG ASPINWALL**

Capillary Electrophoresis (CE) is an analytical technique used to separate molecular species based on charge and hydrodynamic radius. CE provides limits of detection with conventional instrumentation in the femtomole to attomole range (10-15 mol to 10-18 mol), making CE applicable for a wide range of uses, such as in hospitals, forensics, food safety, and water purity. To broaden the applicability of CE, we have developed a more mobile and cost-effective CE instrument through the use of additive manufacturing, better known as 3D printing. A 3D printed computer mouse-sized instrument was fabricated that houses the optical components necessary for CE, including an excitation filter, emission filter, dichroic mirror, and microscope objective, as well as a capillary holder. To test the viability of this instrument design for Capillary Zone Electrophoresis (CZE), we analyzed varying concentrations of fluorescein in solution. The overall goal is the optimization of the instrumentation to achieve limits of detection comparable to or better than commercial instrumentation. A small and mobile 3D printed instrument with such limits of detection and cost-effectiveness would greatly increase the applicability and accessibility for CE. Additionally, the 3D printed design is readily transferable, enabling greater potential for replication and relatively quick, in-house modifications to address institution-specific issues and limitations. This research was supported by the Undergraduate Biology Research Program with funds from the Office of the Provost and the Department of Biomedical Engineering.

RNA-DIRECTED DNA METHYLATION MEDIATES PARENTAL CONFLICT DURING SEED DEVELOPMENT IN *CAPSELLA GRANDIFLORA*

***CECILIA MARTINEZ*, JACK STEARNS, BRANDON DAVID, KELLY DEW-BUDD, MARK BEILSTEIN**

Seed development in plants is affected by the methylation status of DNA. The methylation pathway most active during seed development is RNA-directed DNA methylation (RdDM). RdDM is responsible for silencing genes in the developing embryo through the production of small RNAs (sRNAs). These sRNAs are products of RNA polymerase IV and interact with the

transcripts produced by RNA polymerase V. RdDM is required for proper seed development in *Brassica rapa*; RdDM mutants in this species have reduced seed set and seed weight. In contrast, mutations in RdDM show little to no impact on seed development in *Arabidopsis thaliana*. The primary differences between these two species are their breeding systems and ploidy level: *B. rapa* is as an outcrossing mesopolyploid species and *A. thaliana* is a primarily inbreeding diploid species. In light of these genomic differences, we hypothesized that *B. rapa* exhibits a more significant phenotype in RdDM mutants because the RdDM pathway mediates either maternal vs. paternal genome conflicts, and/or conflict among subgenomes in polyploids. Our subsequent experiments focused on targeting species with differences in breeding system and ploidy in the following species: *Capsella rubella*, an inbreeding diploid species, *Capsella grandiflora*, an obligate out-crosser with a diploid genome, and *Camelina sativa*, an inbreeder with a polyploid genome. We observed that *C. grandiflora* has a large reduction in its seed set and weight in RdDM mutants. Our preliminary conclusion is that the RdDM pathway plays a more important role in mediating conflict between maternal and paternal genomes, while its impact in mediating conflict between subgenomes is less profound. By elucidating a role for RdDM, we can produce a greater understanding of how gene silencing and gene modifications impact seed development. This research was supported by the Undergraduate Biology Research Program with funds from the Office of the Provost.

EXAMINATION OF ENDOPHYTIC FUNGI IN THE BIOSPHERE 2 TROPICAL RAINFOREST

***BRENNA MCINTYRE*, MARISSA CLOVER, LILLIAN P. MOORE, JÜRGEN KREUZWIESER, LAURA MEREDITH, JANA M. U'REN**

Volatile Organic Compounds (VOC) are low molecular-weight organic compounds that easily evaporate at room temperature. VOCs are produced as secondary metabolites in plants, bacteria, and fungi, often playing important roles in environmental signaling and stress response. To date, ca. 250 VOCs have been described from fungi; however, little is known about VOC diversity and function in fungi. The goal of this study was to examine the relationship between VOCs and foliar endophyte fungi--a functional group of symbionts that live within plant tissues without causing disease--in *Clitoria fairchildiana* trees in the Biosphere 2 Tropical Rain Forest (B2-TRF). The B2-TRF is an artificial mesocosm that contains ca. 100 plant species from different tropical regions of South and Central America. Due to the glass enclosure, temperature in the B2-TRF increases with height, with temperatures at the top of the rainforest canopy exceeding 40°C in summer months. The goal of this study was to examine whether tree individual or canopy height is a better predictor of foliar fungal communities in *Clitoria*, and whether there is a relationship between leaf VOCs and endophytic fungal communities. We used passive air samplers coupled with high resolution gas chromatography to measure VOC production directly from *C. fairchildiana* leaves. These same leaves were then used for DNA extraction to examine endophyte communities using next-generation sequencing (NGS) of the fungal 'barcode' locus (ITSnrDNA). In addition, we isolated endophytes in culture from a subset of leaves of one tree to examine VOCs directly from fungal cultures. Overall, culturing yielded over 70 fungal isolates representing >20 species. Taxonomic identity of fungal cultures was very similar to endophytic fungi from trees in Panama and South America, reflecting the tropical environment of B2-TRF. Leaf-level VOC composition differed by individual tree rather than primarily as a function of canopy height. Fungal cultures also produced diverse VOCs and future analyses will examine the relationship of these VOCs to total plant VOCs. On-going sampling will examine the impact of drought on foliar fungal communities in B2-TRF. Funding for this study was provided by the Office of the Provost through the Undergraduate Biology Research Program.

MAJOR CHILDHOOD TRAUMA PREDICTS INCREASED DELAY DISCOUNTING IN YOUNG ADULTS

***CORINNE MEINHAUSEN*, ANNELISE MURILLO, BENJAMIN M. ROSENBERG, CHRISTINA F. SANDMAN, ROBIN NUSSLOCK, RICHARD ZINBARG, SUSAN BOOKHEIMER, MICHELLE G. CRASKE**

Objective Delayed reward discounting, or the tendency to discount future rewards in favor of smaller immediate gains, is linked to addiction and other risk-taking behaviors. Stressful life experiences, such as childhood trauma and lower socioeconomic status predict greater delayed discounting. However, it is unknown whether major trauma in early childhood will influence performance on a delay discounting task independent of current financial stress. We hypothesized that major childhood trauma would predict increased delayed reward discounting, and the relationship would be present above and beyond current financial stress. Participants and Methods: 265 college students between the ages 18-20 years (M = 18.66, SD = 0.56) completed the

study. Participants received the Childhood Trauma Interview to determine the number of major and minor traumatic events that occurred between the ages of 0-9 years and were assigned a current financial stress rating as determined by a Life Stress Interview. Participants preference for immediate versus delayed rewards was assessed using a hypothetical monetary delay discounting task. Results: A regression analysis of delay discounting and current financial stress did not result is a significant effect ($F(1, 261) = 0.972$, $p = 0.365$). A regression analysis of delay discounting and major childhood trauma resulted in a significant effect ($F(1, 261) = 12.299$, $p = 0.001$). A multivariate regression analysis indicated major childhood trauma was a significant predictor of delay discounting ($t = -3.696$, $p < 0.001$) above and beyond the effect of current financial stress ($t = -0.152$, $p = 0.880$) and minor childhood trauma ($t = 1.535$, $p = 0.126$). Conclusions: Major early childhood trauma is a significant predictor of delay discounting and the relationship is present when accounting for current financial stress. The findings suggest a long-lasting impact of major childhood trauma on future decision making. This project was supported by a research grant from the National Institutes of Health (NIH) Maximizing Access to Research Careers (MARC) Training Grant T34 GM08718 and the University of California, Los Angeles.

EVALUATION OF SUSPECTED TARGETS FOR A CRISPR SYSTEM ENCODED ON A MEGAPLASMID IN PSEUDOMONAS

***MADISON MOLLICO*, DAVID BALTRUS, MEARA CLARK**

Horizontal gene transfer (HGT) is the exchange of genetic material between individuals that are not necessarily genetically related. HGT often confers fitness benefits, such as antibiotic resistance, to the receiving organism. Bacteria frequently exchange plasmids in this manner. Plasmids are circular DNA which are separate from, and therefore replicate independently of chromosomal DNA. Plasmids may compete with other plasmids to express genes within an organism. One method of competition is mobilization of clustered regularly interspaced short palindromic repeat regions (CRISPR)-associated proteins. These proteins cut at specific targets in chromosomal or plasmid DNA, preventing replication. Here, we examine one such CRISPR system located on the megaplasmid pBASL58, which is approximately 1 MB in size. The pBASL58 CRISPR system is the only known type 1F CRISPR system completely contained on a plasmid in Gram negative bacteria. This property could potentially allow it to be passed between species by HGT and expressed without requiring the presence of other genes in the chromosomal DNA, or on other plasmids. This potentially enables the system to carry out precise, targeted killing of other bacterial cells or to block the uptake of other plasmids within engineered stains, such as those that contain antibiotic resistance genes. Experiments were conducted to determine if the CRISPR system on pBASL58 is active. Gateway cloning was used to generate two target plasmids, each corresponding to a CRISPR target encoded by spacer regions on the megaplasmid and containing a gene for kanamycin resistance. Triparental conjugations between the megaplasmid-containing strain (closely related to *Pseudomonas putida*) and the cloned target-containing strains (Escherichia coli) were performed. Colonies were selected based on kanamycin resistance. A lack of growth would suggest CRISPR activity, since CAS proteins would prevent replication of the plasmid that encodes kanamycin resistance. Clones containing CRISPR target sequences grew at the same rate as clones in which CRISPR target sequences were altered. Based on these findings, this CRISPR system does not target sequences identical to its spacer regions under standard conditions. This work is made possible by funding from the National Science Foundation and the Undergraduate Biology Research Program with funds from the Office of Research, Innovation & Impact.

THE PEG FOREST AS A NOVEL MAZE TO EXAMINE SPATIAL MEMORY

***EMILY MONROE*, LINDSEY CROWN, FRANCHESCA FERNANDEZ, L. MATTHEW LAW, JONATHAN LIFSHITZ, STEPHEN COWEN**

One very important function of the brain is spatial learning and memory, or one's knowledge of where they are in space and what actions must be taken to get to another location. This study aims to validate a new maze for studying spatial learning and memory in rodents, called the Peg Forest. This maze has advantages over those commonly used in research, as it allows for the rodent to freely explore an environment, similar to a grassy field, without causing severe stress. The maze also has the potential to represent countless novel environments, a flexibility that many other commonly used spatial memory tests do not have. By

placing a mouse on one configuration of pegs for five days, then switching the configuration, we can determine if the rodent becomes familiar with the environment, and then realizes a novel environment, by analyzing measures of exploration such as distance traveled, total entropy, and time spent active, on each day. We found that although the mice decrease in these measures of exploration over time within each configuration, there is no significant increase upon a switch in configuration. This suggests that there is a novelty effect of the maze, but the mice may not realize the differences in peg configuration. This research was supported by the Undergraduate Biology Research Program with funds from the Office of Research, Innovation & Impact, and the Department of Biomedical Engineering.

GALLBLADDER DISEASE AND HELICOBACTER PYLORI INFECTION IN NORTHERN ARIZONA – A POPULATION STUDY

***SHANOA NEZ*, ROBIN HARRIS, HEIDI BROWN, DARIEN FULLER, RACHELLE BEGAY**

Purpose: The primary objectives of this research are to determine the prevalence of gallstone disease and *Helicobacter pylori* (*H. pylori*) in American Indians and explore the potential association between the two. Introduction: *H. pylori* is one of the most common chronic bacterial infections in the world. It is a predisposing factor for the formation of gallstones due to inflammatory changes in the gallbladder caused by the bacterium. While American Indians have both high rates of gallstone disease and *H. pylori*, there is no study for a potential association in this population. Methods: In the summer of 2018, a research team from the Native American Cancer Prevention (NACP) Program administered a cross-sectional survey to residents of 72 households from three Southwest American Indian communities, that asked about self-reported gallstone disease and cholecystectomy. Data were collected on *H. pylori* infection based on the urea breath test (UBT). Frequency of self-reported gallbladder disease and *H. pylori* infection were calculated as well as chi-square tests; all analyses done using R version 3.5.1. Results: Of 101 participants with UBT data, 23.8% reported gallstone disease, and 9.9% reported undergoing cholecystectomy, and 65.3% were positive for *H pylori*. Among those positive for *H. pylori*, 21.2% had a history of gallstones compared to 28.6% for those who tested negative for *H. pylori*, p =0.41. Conclusion: While there is no strong association between gallstone disease and *H. pylori* in this small sample, there is a high prevalence of both gallstone disease and *H. pylori* in American Indians in Arizona. Further research is needed to understand the impact of diseases for American Indians. This research was funded through the Partnership for Native America Cancer Prevention (NACP) through a grant from the National Cancer Institute, grant #2U54CA143924.

A NOVEL MOUSE MODEL TO STUDY THE ROLE OF ALPHA-SYNUCLEIN IN PARKINSON'S DISEASE

***NHAT NGUYEN*, ANANDHAN ANNADURAI, LUKE DREHER, MANDI CORENBLUM, DONNA ZHANG, LALITHA MADHAVAN**

Parkinson's disease (PD) is a chronic and progressive neurodegenerative disorder that affects more than 8 million people worldwide - a number that is expected to continue to grow over time as the population ages. However, the mechanisms underlying the pathogenesis of PD are not well understood. Currently, much research is focused on the protein Alpha-Synuclein (α-Syn), which is found within dying dopaminergic (DA) neurons in PD patients' brains. The function of this protein is not fully understood, but several studies have shown that α-Syn might play an essential role in the development of PD. Specifically, it has been proposed that the aggregation of this protein in intracytoplasmic inclusions called Lewy bodies impedes neuronal function, which consequently causes damage to and even death of the neurons. Nrf2, a transcription factor and master regulator of cellular homeostasis, is a potential therapeutic target for PD. Studies have shown that Nrf2 regulates the expression of an array of protective and pro-survival genes, including antioxidant and anti-inflammatory genes. It also controls the autophagic processing of many proteins, including α-Syn. Given this, we have generated a novel mouse model to comprehensively study the potential role and therapeutic value of Nrf2 in the context of synucleinopathy in PD. We crossed Nrf2 knock-out (Nrf2 -/-) and Nrf2 wild type (Nrf2 +/+) mice with transgenic mice overexpressing wild type human α-Syn, which generated four different genotypes: hα-Syn - / Nrf2 +/+, hα-Syn - / Nrf2 -/-, hα-Syn + / Nrf2+/+, hα-Syn + / Nrf2 -/-. We then analyzed the animals at three months of age using specific behavioral tests and molecular assays. We specifically subjected the

animals to the tapered beam, spontaneous movements in cylinder, nest building (Nestlet), and open field tasks to evaluate their motor function. Correspondingly, we assessed the protein and mRNA level changes in different brain regions (striatum, midbrain, cortex, hippocampus) through Western blotting and qPCR. Our data showed that the α-Syn + / Nrf2 -/- made more errors while taking a longer to cross the tapered beam compared to the α-Syn + / Nrf2 +/+ mice. In the cylinder task, it was found that the α-Syn + / Nrf2 -/- made fewer number of rears, as well as hindlimb steps, compared to the α-Syn + / Nrf2 +/+ mice. In the nestlet task, the α-Syn + / Nrf2 -/- exhibited significantly less usage and pulldown of the cotton nest materials compared to the α-Syn + / Nrf2 +/+ mice. Specific motor deficits were also found in open field task, in α-Syn + / Nrf2 -/- mice, compared to the α-Syn + / Nrf2 +/+ mice. With regards to the molecular analysis, so far, we find higher phosphorylated α-Syn, and increased iNOS2 (oxidative stress marker) and LC3II (autophagy marker) especially in the striatum and midbrain of α-Syn + / Nrf2 -/- mice. Overall, we expect that this project will reveal α-Syn and Nrf2 interactions, important in understanding the mechanistic basis as well as therapeutic development in PD. This research was supported in part by the Undergraduate Biology Research Program with funds from the Office of Research, Innovation & Impact.

IMPACT OF POLYPHENOLIC COMPOUNDS ON FOLIAR ENDOPHYTIC FUNGI OF *QUERCUS*

***MEGAN NICKERSON*, LILLIAN P. MOORE, LORENA ENDARA, J. GORDON BURLEIGH, MALAK TFAILY, JANA M. U'REN**

Plants produce a wide array of chemical products or 'secondary metabolites' that provide protection against biotic and abiotic stress. Polyphenols (compounds composed of an aromatic ring with one or more hydroxyl group) are one of the most abundant metabolite groups. Polyphenols act as defensive and antimicrobial compounds that protect plants from UV radiation, extreme temperatures, herbivores, and pathogens. Although known to inhibit the growth of some fungi and bacteria, few studies have addressed the impact of polyphenols on the growth and colonization of non-pathogenic symbiotic fungi. Thus, the goal of this study was to examine the relationship between leaf polyphenols and the diversity and composition of foliar fungal symbionts in *Quercus* (oak). We collected living, asymptomatic leaves and leaf litter from red and white oaks in four distinct biogeographic locations throughout the US (Arizona, Florida, Minnesota, and California). We examined the diversity and taxonomic composition of symbiont communities using high-throughput next-generation amplicon sequencing (NGS). From subsets of the same leaves, we measured total phenolic content and used liquid chromatography tandem mass spectrometry (LC-MS/MS) to determine polyphenol composition. Lastly, we performed in vitro growth assays for phylogenetically diverse fungi on six different phenolic compounds. We tested the following hypotheses: (i) decaying leaf litter will contain more diverse fungal communities than living leaves; (ii) leaves with higher total phenolic concentration will harbor less diverse fungal communities due to the antimicrobial activity of phenolics; and (iii) fungal symbionts with higher host specificity to *Quercus* will display higher growth on phenolic concentrations in vitro. Overall, we found that leaf life span impacts symbiont communities: fungi were more abundant and diverse in oak species with evergreen leaves compared to deciduous leaves. We observed a correlation between phenolic content and fungal diversity; however, the pattern appears driven by differences between red and white oak species. Lastly, in vitro growth assays illustrate that many phenolic compounds can promote symbiont growth. On-going analyses will assess the role of specific phenolic compounds on fungal diversity and the correlation to in vitro fungal growth. Funding for this study was provided by the Honors College, and the Undergraduate Biology Research Program with funds from the Office of the Provost and the College of Agriculture and Life Sciences.

QUANTIFICATION OF PULMONARY VASCULAR STRUCTURE IN THE SUGEN/HYPOXIA MOUSE MODEL OF PULMONARY HYPERTENSION

***RYAN OCHOA*, PRITESH JAIN, SUSUMU HOSOKAWA, JASON X.J. YUAN, REBECCA R. VANDERPOOL**

Right ventricular (RV) function is the main determinant of mortality in patients with Pulmonary arterial hypertension (PAH). PAH is characterized by an increase in mean pulmonary artery pressure, a low pulmonary capillary wedge pressure, and increased pulmonary vascular resistance due to pulmonary vascular remodeling. Available treatments slow the progression but not the reversal of pulmonary vascular remodeling. The aim of the study is to quantify the structural changes in the pulmonary vasculature in relation to right ventricular function in the sugen/hypoxia mouse model of pulmonary hypertension. Mice (8 to

12-week-old) were exposed to four weeks of sugen/hypoxia (n = 6-8 male C57BL/6J mice) or normoxia (n = 6-8 male C57BL/6J mice). Pulmonary and RV hemodynamics were measured using closed chest cardiac catheterization (Millar, Houston, TX). The lungs were then perfused with MicroFil (Flowtech, Massachusetts) and imaged in the MicroCT (Siemens Inveon). Measurements of vascular volume and surface area were made from segmentations of the pulmonary vasculature (Slicer 3D). Data collected from catheterization suggests a significant difference between normoxia and hypoxia mice lungs when their mean pulmonary arterial pressure, systolic arterial pressure, and systolic right ventricular pressure are compared to each other. Preliminary analysis of microCT imaging of sugen/hypoxia mice suggest they have a vascular volume of 77.25 +/- 39.08 mm^3 and a surface area of 1597.39 +/- 669.39 mm^2. Further investigation of normoxia and hypoxia mice lungs is required to determine if there is a significant difference between the vascular volume and surface area of the two groups. Funding: Biomedical Research Centre – National Institute on Aging (NIA), PVDOMICS study National Heart, Lung, and Blood Institute (NHLBI): U01 HL125208, the National Institutes of Health (NIH) Maximizing Access to Research Careers (MARC) Training Grant T34 GM08718.

NUCLEAR EGFR DRIVES EPIGENETIC DYSREGULATION

***DANIELA ORTIZ*, CARLY WIERSMA, BENJAMIN G. BITLER, JOYCE A. SCHROEDER**

The Epidermal Growth Factor Receptor (EGFR) is a receptor tyrosine kinase located at the basolateral membrane and is responsible for cell-signaling events. In cancer, however, it is often amplified throughout the whole membrane of a transformed cell. Intracellularly, EGFR takes a retrograde trafficking route that localizes it into the nucleus rather than being recycled or degraded. The glycoprotein Mucin 1 (MUC1) protects EGFR from lysosomal degradation and promotes its internalization to the nucleus. Nuclear EGFR is correlated with high levels of acetylated histones in the DNA. As previously established, MUC1 alters the interaction between EGFR and promoter regions that are transcriptionally active. The information leads us to hypothesize that nuclear EGFR deregulates the transcription of Cyclin D1 (CCND1) epigenetically through direct contact with the CCND1 promoter. Here, we tested the interaction between EGFR and the Cyclin D1 promoter under the influence of MUC1 and also tested by blocking retrograde trafficking. We tested the interaction through Chromatin Immunoprecipitation (ChIP) with the purpose of finding a sequence in the DNA where a specific protein is binding to, in our case the CCND1 promoter and EGFR. Our results suggest that serum promotes interaction between EGFR and chromatin at the CCND1 promoter. This experiment offers more comprehension on retrograde trafficking and further directions to studying nuclear EGFRs effects as a co-transcriptional factor.

MEASURING POPULATION FITNESS USING DEATHS CORRELATED TO FITNESS

***MICHAEL OSIPOV*, JOSEPH DANIEL MATHESON, JOANNA MASEL**

An important factor in any species existence is its fitness over time. To find this a number of models can be used. In this case a computer model was created that would simulate a population of creatures in order to find if a human's beneficial mutation rate was sustainable and if so, what rate was infeasible. While I plan to expand my current model, the only factor looked at in this stage of the experiment was death due to low fitness in a population and how this would affect the populations fitness over thousands of generations. I experimented with a population starting at similar fitness and how deleterious mutations would affect the average fitness over time. The population was modeled using relative fitness meaning no individuals would be added to the population nor would any death coincided with the loss of population. The program also looked to create a population by focusing on only births and deaths of each individual within it and no other factors. The individual with the lowest fitness would be killed off by the program and replaced with a child from two parents picked at random. At each birth a calculation was to see the new child's fitness which was based on a combination of the parent's fitness and mutations that would occur during birth. The result that was obtained was the average fitness of the population after thousands of runs of this process. I then ran the model at several mutation rates to find what mutation rates were feasible with this model. What I found was that under conditions of a regular human genome the average fitness of the population increased while under the condition of deaths due to low fitness. Funding for this was obtained from the University of Arizona.

FACTORS CONTRIBUTING TO EXPLORATION IN FORAGING BUMBLE BEES

***CHLOË PATERSON*, JACK-MORGAN MIZELL, ANNA DORNHAUS**

The exploit-explore tradeoff is a problem that all adaptive organisms must face. Striking an appropriate balance between exploratory and exploitative behaviors is necessary for survival; for optimum performance, organisms must both draw from resources they know to be reliable and explore options they know less about but could turn out to be more advantageous. We investigated what is driving exploration in our model, the common eastern bumble bee (*Bombus impatiens*). We tested the bees using a foraging arena, wherein a bee could choose freely between two colors of flower. One flower type provided a highly rewarding nectar concentration, while the other provided a poorer reward. By varying the proportion of each flower type, the bee gained different levels of information about the reward provided by each color of flower. The bee was considered to be exploiting when it sampled from the option it was more experienced with and exploring when it sampled from the more novel option. We examined the hypothesis that bees are faster when exploiting compared to exploring by using video analysis software to determine the reaction time of the bee when presented with a novel versus experienced option. We defined reaction time as the time in seconds that a bee took to select a choice. We also analyzed sampling time, which is the amount of time a bee remained on a given flower, regardless of whether or not it sampled. We documented whether the bee sampled, which order it sampled in, and how many flowers were in the condition. Each bee experienced two sample conditions containing six possible choices that preceded a test condition. Current data suggests that bees do choose experienced options more quickly than they choose novel options, so they may exploit more quickly than they explore while foraging. Moreover, their initial reaction time was longer compared to subsequent reaction times, as the bees tended to speed up as they progressed through each trial. A linear regression model was used to interpret the data. This work expands our understanding of the factors that influence bumble bee decision-making. We thank the National Science Foundation grant no. DBI 1564521 to Anna Dornhaus, the University of Arizona Graduate and Professional Student Council Reap Grant and National Science Foundation Graduate Research Fellowship Program to Jack-Morgan Mizell, and the Undergraduate Biology Research Program and the BIO5 Institute to Chloë Paterson for funding this project.

ABERRANT CAP-DEPENDENT TRANSLATION IN A TDP-43 MODEL OF ALS

***NICOLAI PENA*, SAMANTHA MACKLIN, CLARE HANSS, RACHEL BEAR, BEN ZAEPFEL, SHIZUKA YAMADA, DANIELA C. ZARNESCU**

Amyotrophic lateral sclerosis (ALS), a progressive and fatal neurodegenerative disorder, primarily targets motor neurons. An overwhelming majority of ALS patients exhibit cytoplasmic aggregates containing TAR DNA-binding protein-43 (TDP-43), a nucleic acid binding protein involved in RNA processing. While several cellular processes are altered in ALS, RNA transport and translation appear to be a key mechanism altered in disease. Here we focus on TDP-43's role in mRNA translation and investigate protein synthesis alterations related to ALS. First, using a *Drosophila* model of ALS based on TDP-43 overexpression in motor neurons, we investigated possible functional interactions between TDP-43 and eukaryotic initiation factors (eIFs). More specifically, we tested whether modulating eIF levels affects TDP-43 dependent locomotor deficits as measured by larval turning assays. Our data show that enhanced activity of eukaryotic initiation factor 4E binding protein (4EBP) worsens locomotor phenotypes caused by TDP-43 expression in motor neurons. Next, we directly tested whether TDP-43 causes translational dysregulation by evaluating puromycin incorporation into nascent peptide chains. Preliminary data from patient derived lymphoblastoid cells show reduced puromycin incorporation, suggesting reduced global translation caused by TDP-43 proteinopathy. To further probe the role of TDP-43 in translation we are currently using non-canonical amino acid tagging (BONCAT/FUNCAT) which measures newly synthesized proteins in-vivo and affords the identification of specific proteins altered at the level of translation in motor neurons.

AMYLOID-$\beta_{(1-42)}$ INHIBITS CALCIUM^{2+} TRANSIENTS INDUCED BY N-METHYL-D-ASPARTATE RECEPTOR IN THE CEREBRAL VASCULATURE

***EMILY PETERS*, RAMON JOSE AYON, PAULO WAGNER PIRES**

Endothelium-dependent dilation of cerebral arteries, a process dependent on transient increases in intracellular calcium (Ca^{2+}), contributes to neurovascular coupling (NVC), which is altered in cerebral amyloid angiopathy (CAA).The N-methyl-D-aspartate receptor (NMDAR), a nonselective cation channel with high Ca^{2+} permeability, has been shown to mediate endothelium-dependent dilation in cerebral arteries. NMDAR activity is reduced by amyloid-β, which accumulates around the cerebral vasculature during CAA. We hypothesized that amyloid-β impairs NMDAR-induced Ca^{2+} transients in endothelial cells of cerebral arteries, which impairs endothelium-mediated dilation in mice. All animal experiments were approved by the University of Arizona IACUC. Data are means ± SEM, both in male and female mice (no sex differences were observed). Cerebral arteries isolated from mice expressing the genetically-encoded calcium indicator GCaMP8 in endothelial cells (*cdh5:Gcamp8*) were prepared *en face* for time-lapse imaging of endothelial Ca^{2+} transients induced by NMDAR activation. In fields of view that displayed Ca^{2+} transients, we found that the NMDAR agonist NMDA (1 μM) increased the frequency of endothelial Ca^{2+} transients compared to baseline (0.22 ± 0.06 vs 0.58 ± 0.15 Hz, baseline vs NMDA, n = 10 and 13 fields of view from at least 3 mice, $p < 0.05$, one-way ANOVA). Pre-incubation of preparations with the NMDAR antagonist D-AP5 (10 μM) prevented NMDA induction of endothelial cell Ca^{2+} transients (frequency: 0.14 ± 0.05, n = 10 fields of view, $p < 0.05$ vs NMDA, one-way ANOVA). These data suggest that endothelial NMDAR Ca^{2+} transients can be stimulated in cerebral arteries via NMDA. We then tested whether the peptide amyloid-$\beta_{(1-42)}$, commonly found in CAA, blunted NMDAR-elicited Ca^{2+} transients. Cerebral artery preparations were incubated for 30 minutes with 5 μM amyloid-$\beta_{(1-42)}$, then exposed to NMDA. Our preliminary data suggests that pre-incubation of preparations with amyloid-β blunts NMDA-dependent induction of endothelial cell Ca^{2+} transients (0.21 ± 0.04, n = 10 fields of view, $p < 0.05$ vs NMDA, one-way ANOVA). In order to evaluate the effects of amyloid-β on dilation of cerebral arterioles, we then performed *ex vivo* pressure myography experiments with cerebral parenchymal arterioles from a mouse model of familial Alzheimer's disease without aging (*5x-FAD*) or wildtype littermates. Our preliminary results suggest that NMDA-elicited dilation of parenchymal arterioles may be impaired in *5x-FAD* mice (vasodilation (%): 11.06 ± 0.78 vs 6.21 ± 2.09, wildtype vs *5x-FAD*, n = 3 arterioles from 3 mice, $p = 0.067$, two-tailed *Student*'s t-test). These preliminary data suggest that NMDA receptors in the cerebrovascular endothelium of wildtype mice mediate arteriolar dilation via an increase in Ca^{2+} transients. Further, amyloid-β may impair the activity of endothelial NMDA receptors and thus contribute to neurovascular dysfunction via impaired arteriolar dilation in individuals with CAA. Funding: The National Heart, Lung, and Blood Institute (NHLBI) of the National Institutes of Health (NIH) under award number R00HL140106 to PWP, and supported in part by the Undergraduate Biology Research Program with funds from the University of Arizona Office of the Provost and the College of Medicine.

COMPLEX RODENT SPATIAL NAVIGATION WITH OBJECTS AND 3D NAVIGATION

***YUXIN QIN*, MADDIE SOUDER, JEAN-MARC FELLOUS**

Navigation is a critical skill for both humans and rats. To complete our daily activities, a neural system for complex spatial navigation is required. The hippocampus is involved in both path integration and landmark navigation, which are two strategies utilized in spatial navigation. The Traveling Salesperson Problem (TSP) is a spatial problem model in which the subject needs to find the shortest path between cities over multiple trials. In our rodent study, the size of the room is significantly larger than in previous studies, and the cities are different objects with cups attached instead of identical cups. Detached cups and objects were used as control conditions. The configuration of the object placement is designed to study path optimization. We hypothesize that an object switch would affect their choice of path. The resulting paths are compared to path obtained from the same configuration with identical cups. The results are also compared between males and females. The preliminary result show differences in moving speed between the three trials before switch and the three trials after switch. Single cell and network activity recorded from hippocampus during the task show a difference in sharp wave activity between cup and object configurations. From observing the rat's behavior around objects, we were curious about their behavior in a 3D environment. A new 3D maze with three levels was made with colorful Lego blocks accompanied with three corresponding sounds indicating the reward position. One reward at one level per trial. Previous study (Hayman et al., 2011) has shown differences in place cell firing at different heights for rodents going down or up a transparent helix or pegboard with no reward. In the current pilot study, the rat has the task to find a reward with guidance. We hypothesize the searching behavior could significantly change the

single cell and network activity. Funding: The Undergraduate Biology Research Program with funds from the Office of the Provost and the College of Science.

RED BLOOD CELL-MIMETIC ARTIFICIAL PROTEIN HYDROGELS: TRANSLATING PROTEIN NANOMECHANICS INTO FUNCTIONAL HYDROGELS

***JOCELYNE RIVERA*, DAVID KNOFF, KAREEN FAJARDO, MINKYU KIM**

Cardiovascular disease is the leading cause of death nationally and worldwide according to the National Institutes of Health. Targeted drug delivery has been an important biomedical goal for several decades to avoid debilitating side effects, however a genuinely low number of novel drug delivery systems have been applied clinically. Herein, we offer a new mechanism for targeting cardiovascular disease by designing mechanosensitive artificial protein hydrogels that mimic the spring-like nanomechanics of ankyrin, a red blood cell cytoskeletal protein. Translating protein nanomechanics to macroscopic materials has been limited to date due to topological defects in polymer networks. In this study we develop triblock self-associating proteins that utilize the rigidity of NI6C, an ankyrin-repeat protein, to reduce topological defects, as well as improve crosslinking homogeneity via hierarchical network assembly. Using genetic engineering techniques and recombinant proteins expression, we are developing a hydrogel consisting of telechelic proteins with streptavidin monomer end groups. Specific ratios of rigidness and flexibility amino acid properties will determine the efficiency of the hydrogel scaffold for long-term in-vivo applications, including drug delivery. This design to control effective crosslinking in the polymer network is being investigated as a means to improve the translation of NI6C nanomechanics to artificial protein hydrogels, in order to develop mechanosensitive micro-gels that reversibly deform under high fluid shear stress. Responding to high fluid shear stress environments, indicative of atherosclerotic regions, the proposed micro-gel will deform to deliver drug exclusively to the target tissue for patients suffering from cardiovascular diseases.

THE EMOTIONAL STROOP TASK FOR CANCER PATIENTS

***RUDOLPH RODRIGUEZ*, YING-HUI CHOU**

The Emotional Stroop Task is a psychological evaluation used to assess prevalent emotions in a subject. These are found by presenting words in different colored ink and having the subject name the color of the assigned word. The words themselves will change between neutral, positive, and negative valences. The response time is measured for each word, and a longer latency between the presentation of the word and the naming of the color points to a stronger emotional response to said word. This longer latency, also known as the Emotional Stroop Effect (ESE) can be used to measure how strongly a person responds to particular words. Using words obtained from a narrative program for cancer patients, we have designed a wordlist specific for people who have or have had cancer. Using this wordlist, we plan to observe how strongly our subjects react to cancer-specific words by measuring their reaction time to these words in the Emotional Stroop Task. Funding provided by: The Undergraduate Biology Research Program with funds from the BIO5 Institute, Dr. Ying-hui Chou Lab, and this research was supported in part by the Western Alliance to Expand Student Opportunities (WAESO) Louis Stokes Alliance for Minority Participation (LSAMP) National Science Foundation (NSF) Cooperative Agreement No. HRD-1101728.

USING TISSUE SPECIFIC HISTONE MARKERS TO ANALYZE LINCRNA REGULATION

***JAMES ROZELLE*, CECILIA MARTINEZ, MIKE FRANK, MARK BEILSTEIN**

LincRNAs are long intergenic non coding RNA transcripts that have a variety of functions often relating to genome regulation. Within the plant family *Brassicaceae*, a group packed with economically and agriculturally important species, lincRNA loci are more highly conserved than other non-coding regions of the genome. Moreover, studies of specific lincRNAs have shown that they can regulate the transcription of protein coding regions, operate as scaffold structures to integrate proteins, and can function as signals. In this study, we aim to better understand the suite of lincRNAs transcribed in different types of tissues, at different developmental stages, and in response to environmental stressors. To accomplish this a method of effectively collecting genetic material from cells that fit each of these varying conditions is required. Cell sorting accomplishes this task. To effectively sort cell types, we are developing histone bound fluorescent markers that are driven under cell and/or tissue specific promoters, allowing us to effectively mark the nucleus of desired cell types. Currently, the project is in the early stages as the plants of study are still being bred to carry the desired phenotypes for cell sorting, however, eventually this study will give new insight into what roles lincRNAs play in development and response to environmental stress. This research is supported in part by the Undergraduate Biology Research Program with funds from the Office of the Provost.

CHARACTERIZING NOVEL KINASES IN THE COLONIC EPITHELIUM

***AMANDA RUELAS*, CARLY CABEL, CURTIS THORNE**

Colorectal cancer is a fatal disease and the second deadliest cancer for men and women combined in the western world. Like many cancers, colorectal cancer exhibits uncontrolled cellular proliferation, a common hallmark in cancer. Over time, proliferative cells acquire mutations, causing further damage and progression to metastatic cancer. Kinases, proteins that phosphorylate their substrates, are highly associated with cellular proliferation. Our lab performed a kinome-wide RNAi screen in human colonic epithelial cells (HCEC), which are immortalized normal colon cells, where each kinase was individually silenced, and cellular proliferation was measured. The RNAi screen revealed a few kinases associated with decreased cellular proliferation. This would indicate that those kinases act as tumor suppressors, proteins that block aberrant cell signaling. Our lab selected 20 kinases as experimental targets based upon their anti-proliferative function, decreased expression in colon cancer and novelty. I am creating a CRISPR/Cas9 knockout bank of the 20 kinases correlated to tumor suppressor behavior in human colon cancer cells to analyze how losing the gene that encodes each kinase affects proliferation and cellular signaling. I am focusing on the understudied kinases CLK3 and CDK10. Little is known about their cellular function and role in the colonic epithelium homeostasis. I am using molecular cloning techniques to clone oligo sequences for the kinases into plasmids, which will then be used to transfect cells via lentiviral transduction. Additionally, I am optimizing the cloning procedure with varying plasmids and oligo sequences that target several exons in the CDK10/CLK3 genes. To date, I have made 14 knockout clones for CLK3 and two knockout clones for CDK10. My next steps are to transfect CLK3 and CDK10 knockout plasmids into HCECs, where I will analyze whether there is an increase, decrease, or no change in proliferation. I will measure cellular morphology and cell signaling through the Wnt pathway, a frequently used pathway in colon cells. Using immunoblotting, I will verify successful CRISPR knockout and use immunofluorescence to visualize and quantify proliferation, morphology, and signaling. My project centers on using molecular cloning and CRISPR/Cas9 to analyze the function of novelty kinases in the colonic epithelium. Funding source: This project was funded by the Margaret Bilson Endowment, the National Institutes of Health (NIH) under award number 5R00DK103126-04, and in part by the Western Alliance to Expand Student Opportunities (WAESO) Louis Stokes Alliance for Minority Participation (LSAMP) National Science Foundation (NSF) Cooperative Agreement No. HRD-1101728.

INVESTIGATING FGFR3 AS A THERAPEUTIC TARGET FOR HEAD AND NECK SQUAMOUS CELL CARCINOMAS

***ANDRES SANCHEZ*, KAY GURLEY, RUSSELL MOSER, CHRIS KEMP**

Head and Neck Squamous Cell Carcinomas (HNSCCs) are the sixth most common form of cancer worldwide. Despite its prevalence, few specific targets are known for HNSCCs. Therefore, it is imperative to identify novel therapeutic targets in HNSCCs. Previously, we performed a high-throughput, kinome wide, siRNA screen in a primary Murine Squamous Cell Carcinoma (MSCC) model and identified Wee1 kinase as a potential therapeutic target. In the same high-throughput screen, we identified the receptor tyrosine kinase Fibroblast Growth Factor Receptor 3 (FGFR3) as an additional candidate. Studies conducted by other groups showed overexpression of FGFR3 in the development of bladder cancers. We first validated FGFR3 in lab with a pool of siRNAs from a secondary source. The goal of this study is to investigate FGFR3 as a clinically relevant target using small molecule FGFR inhibitors, AZD4547 and BGJ398, by determining their impact on MSCC viability and proliferation. In two MSCC lines, one containing a p53 null mutation and another containing wild type p53, we performed Cell Titer-Glo (CTG) assays and determined the IC50 of AZD4547 to be 1.57 M and 2.16 M for the p53-null and wild type p53 cell line, respectively. We performed the CTG assay with BGJ398 and determined the respective IC50 values to be 2.82 M and 3.09 M. Additionally, anti-proliferative effects were observed with increasing concentrations of each FGFR3 inhibitor in clonogenic assays. Finally, Western blot analysis showed a reduction of phosphorylated FGFR3 and decreased AKT signaling, but not AKT expression, after treatment with AZD4547. Our results not only confirm FGFR3 as a possible clinically relevant target in HNSCCs, but also provide evidence that the high-throughput siRNA screen employed is an effective method of finding new therapeutic targets in HNSCCs. This work is funded by the National Cancer Institute (NCI) of the National Institutes of Health (NIH) under award number UO1CA217883. The Summer Undergraduate Research Program is supported in parts by the Cancer Center Support Grant (CCSG) CURE Supplement: NCI 3P30-CA015704, the Fred Hutch Internship Program, and individual labs/research groups.

OPTIMIZATION OF PROTEASE ACTIVATED RECEPTOR-2 COMPETITIVE BINDING ASSAY

***ESTEVAN SANDOVAL*, JOY PRISCO, MATTHEW KAPLAN, JUSTIN HOFFMAN, MARISSA LOVETT, SCOTT BOITANO**

Protease Activated Receptor-2 (PAR2) is a G-protein coupled receptor that is expressed in a variety of cells, including those of the airway epithelium, where its activation is associated with asthma. In the airway, PAR2 is activated by exogenous (e.g. from asthma-inducing allergens) and endogenous (e.g. epithelial or immune cells) proteases. These proteases cleave the extracellular amino terminus of PAR2 exposing a tethered ligand that binds the remaining receptor and initiates cellular signaling. PAR2 activation can lead to several asthma indicators in vivo including: increased mucus production, proinflammatory proteins, and airway hyperresponsiveness. Thus, PAR2 is a potential target for novel drugs to treat asthma. To this end, our laboratory has developed PAR2 ligands that can directly activate PAR2 or antagonize protease-activation of PAR2. In order to better characterize these ligands, we have developed a competitive, europium-based, time resolved fluorescent PAR2 binding assay with the novel PAR2 peptidomimetic agonist, 2-furoyl-LIGRLO-(diehtylenetriamminepentaaceticacid)-NH2 (2f-dtpa). In this project, I have worked with the Functional Genomics Core Facility at the University of Arizona to modify this assay for high capacity screening. Integrating automatic pipetting into the assay has led to more precise competition timing across the replicates resulting in higher reproducibility along with an improved window for fluorescent detection. This improved assay will be used to better screen novel compounds as PAR2 ligands and thus, improve the initial step in our PAR2 drug development program. This research was supported in part by the Western Alliance to Expand Student Opportunities (WAESO) Louis Stokes Alliance for Minority Participation (LSAMP) National Science Foundation (NSF) Cooperative Agreement No. HRD-1101728, and the Environmental Health Sciences – Transformative Research Undergraduate Experience (EHS-TRUE) through the National Institute of Environmental Health Sciences Grant #1-R25-ES025494.

WORKING MEMORY AND READING IN SCHOOL-AGED CHILDREN

***SIENA SCHOELEN*, MARY ALT**

Purpose: To investigate how each component of working memory relates to the decoding portion of reading. Method: Thirty-one monolingual English-speaking kindergarten children participated in this study including children with and without typical language skills. The children completed standardized assessments for language (Celf-5), intelligence (KABC II), and measures of reading decoding (Dynamic Decoding Measures (DDM)). They also completed the Comprehensive Assessment Battery for Children - Working Memory (CABC-WM). The CABC-WM included 14 subtests spread over two testing sessions, although we selected four subtests for analyses. We used statistical analyses of correlation and regression to compare the reading and working memory measures. Predictions: We hypothesized that the scores on the CABC-WM tasks would predict performance on the DDM decoding measure. We expected that children who had higher scores on the CABC-WM tasks would perform better on the DDM decoding measure. Results: One phonological working memory task predicted reading decoding score. However, the other working memory tasks did not. The other tasks had a high percentage of children who performed at floor levels. Conclusions: Phonological working memory predicted decoding, which is an important part of reading. Other parts of working memory may contribute to reading, but the tasks we used were too difficult for many kindergarteners, leading to inconclusive results. Working memory may explain individual variability in decoding. Acknowledgements/funding: This research included tasks from the Profiles of Working Memory for Educational Research (POWER). The authors acknowledge POWWER/POWER investigators Shelley Gray, Mary Alt, Tiffany Hogan, Sam Green, Roy Levy and Nelson Cowan for their contributions to this research. POWWER was funded by the National Institute on Deafness and Other Communication Disorders (NIDCD) of the National Institutes of Health (NIH) under award number R01DC010784. Additional funding is provided by the Western Alliance Expanding Student Opportunities, and the University of Arizona Office of Provost and College of Science.

ENGINEERING MOLECULAR SWITCH

***PIPER SEELEY*, JULIE CHEUNG, MARK BEILSTEIN**

Our goal is to employ modern genetic engineering to create a molecular switch within cotton that will allow for the canopy to be able to sustain itself by maintaining water. The three techniques we will use to construct the molecular switch: pollen magnetofection, *Arabidopsis* PYR1 receptor engineered to bind agrochemicals, and conditionally stable ligand binding domains. We started with a yeast codon optimized *Arabidopsis thaliana* PYR1 and utilized PCR to generate a mixed population of mutants. This library will then be screened by fluorescence activated cell sorting (FACS) to find a conditionally stable mandipropamid binding PYR1. The overall goal is to utilize those three techniques to engineer a protein cassette allowing for better water sustainability.

PROBING PROTEIN INTERACTIONS OF RNA HELICASE DED1 USING BIMOLECULAR COMPLEMENTATION

***ASHWIN SIBY*, TELSA M. MITTELMEIER, TIMOTHY A. BOLGER**

Ded1 is a DEAD-box RNA helicase important for translation initiation in budding yeast, *Saccharomyces cerevisiae*. Ded1 is essential in yeast, and the human homolog, DDX3, is implicated in many human diseases. The Ded1 C-terminus is a predicted intrinsically disordered domain that is not absolutely required for helicase activity but plays an important role in the cellular response to stress. Previous in vitro studies and immunoprecipitation assays have shown that Ded1 can bind to the initiation factor eIF4G1 and homo-oligomerize through its C-terminal domain in what is believed to be a mutually-exclusive fashion. However, the extent to which Ded1-Ded1 or Ded1-eIF4G1 interactions occur in vivo, and whether these interactions are altered by stress, remains unknown. Here we use a split luciferase complementation assay to study Ded1 protein-protein interactions within the cell. Initial data from stress-free cells show signs of Ded1-Ded1 interaction that is lost in Ded1 C-terminal mutants. Ded1 interaction with eIF4G1 is also detectable. Future experiments will focus on monitoring these interactions under various stress conditions. This research was funded in part by the Margaret Bilson Endowment.

INVESTIGATION OF THE SIGNALING MECHANISMS OF THE MU-DELTA OPIOID RECEPTOR HETERODIMER

***MADISON SIEFFERT*, ATTILA KERESZTES, NATALIE BARKER, PAUL LANGLAIS, JOHN M. STREICHER**

It has been hypothesized that the mu- and delta-opioid receptors (MOR and DOR) can form a heterodimer (MDOR) and that the resulting heterodimer has a distinct pharmacology. Limited evidence from the literature suggests that MDOR-selective antagonists could be a novel therapy to enhance opioid efficacy while decreasing side effects. However, due to the lack of selective tools, it is very difficult to study this complex system. We thus recently developed D24M, a novel, first-in-class selective MDOR antagonist. When D24M was combined with oxymorphone in vivo, we found potentiation of antinociception and reduction of withdrawal symptoms, validating the hypothesis of the MDOR as an anti-opioid negative feedback loop and that MDOR antagonists could be novel therapeutics. These striking results prompted us to investigate the underlying signaling mechanisms of the MDOR. First, we treated male and female CD-1 mice with oxymorphone and/or D24M, analyzed the brainstem using quantitative phospho-proteomic analysis. Out of the thousands of hits, we found dozens of potential targets that became phosphorylated or dephosphorylated upon exposure to both oxymorphone and D24M. Based on this phospho-proteomic database, we selected the signaling kinases CaMKII and Src to further investigate in vivo. We ran Western blot and tail-flick anti-nociception experiments in male and female CD-1 mice in the presence or absence of specific kinase inhibitors (KN93 and Src I1) combined with oxymorphone and/or D24M. We found that both Src and CaMKII inhibitors blocked the enhanced anti-nociception caused by D24M, while not impacting baseline opioid response. These results suggest that Src and CaMKII are both repressed by the MDOR to repress opioid anti-nociception, which is reversed by D24M treatment. However, Western blot analysis did not show phosphorylation differences in the canonical activation loop sites of either kinase, suggesting that alternate phosphorylation sites identified in our proteomic analysis may be responsible for the signaling effects of these kinases downstream of the MDOR. Our results have thus identified two key nodes in the MDOR signal transduction cascade by which the MDOR blocks opioid anti-nociception and begun to explore their molecular signaling mechanisms. Future work will expand on the signaling cascades of the MDOR, as well as further establish MDOR antagonists as novel opioid therapeutics. Funding Information: These studies were funded by the National Institutes of Health (NIH) under award number R21DA044509 and UG3DA047717 to JMS, and the Undergraduate Biology Research Program with funds from the Office of the Provost. The authors have no relevant conflicts of interest to declare.

CIRCADIAN FUNCTION IN RETINAL PIGMENT EPITHELIUM

***SARA SILLIK*, NICOLE R. CONGROVE, RORY COLVIN-MORRISON, BRIAN S. MCKAY, ROBERT W. SNYDER**

Background: Circadian rhythm is the 24-hour cycle that many physiological processes follow. Circadian rhythm occurs naturally within organisms but can be influenced by outside factors, such as sunlight. Dopamine and L-DOPA have been widely studied and connected to the innate circadian rhythm. The goal of this study was to expose monolayers of retinal pigment epithelium (RPE) produced from induced pluripotent stem cells (iPSC) to either dopamine or L-DOPA in a contained system. A perfusion system was developed that would expose the iPSC cells sequentially to both drugs; with the purpose of mimicking how L-DOPA is released in the body in response to day/night cycles. The overall goals of this project were to test whether RPE function follow a circadian rhythm driven by L-DOPA and dopamine. Methods: The perfusion system alternates delivering L-DOPA then dopamine for 12-hour intervals over a three-day period. The first steps taken to begin running test with the perfusion system were to create monolayers of induced pluripotent stem cells (iPSC) and porcine RPE. The porcine RPE cultures were obtained from freshly dissected eyes. We used the transepithelial resistance (TEER) measurements of the monolayers, taken weekly, to assess monolayer integrity. Once the monolayers were patent, and exhibit a tight barrier, they were placed into the perfusion system and exposed to alternating L-DOPA and dopamine in 12-hour intervals. Conditioned media samples in the effluent were collected every two hours throughout the time course. Results: The data indicates the perfused RPE function in a circadian manner following drug exposure. Samples from both apical and basal compartments were collected and the amount of VEGF and PEDF was measured by ELISA. After the baseline for VEGF and PEDF was established, the results show that the two prominent neurotrophic factors in the eye develop a circadian rhythm in response to alteration between L-DOPA and dopamine. Conclusion: The results show a relationship between L-DOPA and dopamine signaling pathways to establish circadian function in RPE. Previous studies have shown that L-DOPA may treat or prevent Age-related Macular Degeneration (AMD). This study illustrates a potential mechanism for L-DOPA and dopamine to affect AMD pathobiology. Support for this research is provided by The National Institutes of Health (NIH) under award number R01EY026544-01 (McKay), generous support from “Friends of Yuma,” the Western Alliance to Expand Student Opportunities (WAESO) Louis Stokes Alliance for

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CX43 PHOSPHORYLATION IN CARDIAC ISCHEMIC PRECONDITIONING AND INJURY

***DIEGO SILVA-MENDOZA*, TASHA K. PONTIFEX, JOSÉ F. EK-VITORÍN, JOHN KANADY, JANIS M. BURT**

Heart tissue beats synchronously due to the electrical coupling provided by intercellular channels termed gap junction channels. In the ventricles of the heart, these channels are composed of connexin 43 (Cx43); large numbers of these channels localize at the intercalated disks, the site of mechanical as well as electrical coupling in the heart. The colligative properties of these channels support passage of electrical signals from cell to cell, signals that lead all cells of the ventricles to contribute simultaneously to each contraction. Heart attacks, blood clots, or other events that restrict blood flow to (inadequate oxygen supply; ischemia) the cells (myocytes) of the heart for 30 minutes or more, disrupt electrical coupling in the affected (ischemic) region and negatively impact coordinated contraction in the heart, often with deadly effect. Interestingly, loss of blood flow for a brief period of time can protect the heart and electrical coupling from subsequent longer periods of lost blood flow. This protection may, at least in part, reflect phosphorylation, the addition of a phosphate group, of a specific amino acid residue, serine 368 (S368) in the Cx43 protein. Phosphorylation at this residue changes the function of Cx43 channels in a manner that minimizes ischemia-induced damage. In the current project we aim to delineate an accurate time course for phosphorylation of S368. To do so, we are using an antibody that specifically detects only the phosphorylated S368 (pS368) in hearts ischemic for 0, 5, 10, 20, and 30 minutes. Results are compared to total Cx43 in the same hearts. Our preliminary data suggest that pS368 appears in the first 5-10 minutes of ischemic time. Funding: National Institutes of Health (NIH) under award number R01HL131712, and the Undergraduate Biology Research Program with funds from the Office of the Provost and the College of Medicine.

MODULATION OF VEGF SIGNALING IN ATRIAL-LIKE INDUCED PLURIPOTENT STEM CELL DERIVED CARDIOMYOCYTE DIFFERENTIATION

***KRISTINA SIN*, ZOEY NUNN, JARED CHURKO**

The advent of human induced pluripotent stem cells (hiPSCs) and ability to differentiate hiPSCs into cardiomyocytes (hiPSC-CMs) offers a promising and inexhaustible source of cells for creating in vitro cardiac models that may faithfully replicate human phenotypes and the cellular responses exhibited in vivo. Current cardiomyocyte differentiation protocols display cell type heterogeneity and immaturity, which impede upon the conception of cost-efficient, homogenous models. To define novel and more efficient protocols for defined cardiomyocyte generation to the atrial, ventricular, and nodal cell subtypes, it is critical to understand the spatiotemporal environment that distinguishes the embryonic development of these cell types, so to recapitulate conditions that target for generation of the specific cardiomyocyte subtypes. We have recently identified two subpopulations of hiPSC-CMs that are regulated by atrial marker NR2F2 and atrial identity suppressor HEY2. Both markers are opposingly regulated by VEGF-Notch signaling as well as strongly implicated in governing the atrial versus ventricular gene signature programming, as active VEGFR2/VEGF signaling inhibits HEY2 expression and promotes NR2F2. It is hypothesized that by inhibiting the VEGF decoy receptor VEGFR1, atrial differentiation may be promoted by way of enacting the downstream effects of NR2F2 to thus suppress the ventricular program and favor atrial cardiomyocyte generation. This research is supported in part by the Undergraduate Biology Research Program with funds from the Office of Research, Innovation & Impact.

ASSESSING MEXICAN WOODRAT (*NEOTOMA MEXICANA*) MOVEMENT OVER PATCHES OF DIFFERENT BURN SEVERITY

***SANDY SLOVIKOSKY*, JOHN KOPROWSKI**

The use of small mammals as indicators of disturbance and effects on wildlife has become popular in the field of ecology. Population densities, distributions, foraging behavior, and habitat use may change as a result of floods, disease outbreaks, extreme weather conditions, or fire. We examined how Mexican woodrats (*Neotoma mexicana*), a common but understudied species, respond to fire as measured by changes in path tortuosity when translocated over patches of different burn severity. The study was conducted on Mt. Graham, the highest peak of Southern Arizona's Sky Islands, and which experienced the severe 2017 Frye Fire that burned over 19,400 hectares. Woodrats were captured, sexed, weighed, tagged, covered in fluorescent powder, and released 50 meters away from their middens across areas of low or moderate burn severity. The resulting powder trail was marked with pin flags and bearing, and vegetation were noted for each segment of the trail. Vegetation proportions were compared to proportions from randomized vegetation transects for both low and moderate burn severities. Preliminary analyses indicated higher tortuosity over areas of moderate burn severity as demonstrated by increased numbers of turns per straight-line distance. No difference of path tortuosity existed between male and female woodrats. Logs were heavily selected in both areas, whereas grasses were avoided. This may be attributed to lower visual perception in dense vegetation, especially owing to the woodrats large size and preference to move freely over the landscape. With the potential for increased wildfires in the future due to climate change, this work presents an understudied approach to understanding these disturbances and their effects on ecological communities. This research was supported by the Undergraduate Biology Research Program with funds from the Office of Research, Innovation & Impact and the College of Agriculture and Life Sciences, and the United States Department of Agriculture (USDA) Forest Service.

FUNCTIONAL CONNECTIVITY OF PRELIMBIC PREFRONTAL CORTEX AND ROSTRAL VENTROMEDIAL MEDULLA FOR DESCENDING PAIN MODULATION

***ANGELA SMITH*, AUSTIN FLOHRSCHUTZ, CHRISTOPHER CAMPBELL, ZACHARY POLITE, ARTHUR RIEGEL, TALLY LARGENT-MILNES, TODD VANDERAH**

Chronic pain is a health condition that can seriously impact quality of life. To develop better treatments for chronic pain, a better understanding of the mechanisms behind pain perception is needed. Current models of descending pain modulation show the prelimbic prefrontal cortex (plPFC; area 32V) synapsing onto cells within the periaqueductal grey (PAG) which then synapses onto the rostral ventromedial medulla (RVM). The RVM area contains on and off cells which respond to pain stimuli by respectively activating or inhibiting pain perception. We aim to functionally characterize a hypothesized direct projection from the plPFC to the RVM first described in 1997. Anterograde tracing was performed in the plPFC to determine the anatomical connectivity of the two regions. Similarly, we transfected the plPFC with hM4Dq DREADDs fused with mCherry under the control of CaMKIIa promoters to label glutamatergic Layer 5 projection neurons to determine functional connectivity of the purported circuit in rats with spared nerve injury. These receptors are inert unless acted on by a designer drug, such as CNO. We used reverse microdialysis to locally deliver CNO into the RVM and activate DREADDs on neurons with direct projections from the plPFC while collecting released neurotransmitter and performing reflexive pain behavior assays. This strategy allowed for avoidance of systemic/off target activation of plPFC neurons projecting to a different CNS region. Anatomical and microanalysis pilot data indicate a direct connection from plPFC to the RVM. Funding provided by the University of Arizona Department of Medicinal Pharmacology, and the American Society for Pharmacology and Experimental Therapeutics (ASPET) Summer Undergraduate Research Fellowship and the College of Pharmacy.

RECORDS: ASSESING CHANGE IN TEMPERATURE AND OTHER DATA SETS

***TRISTIN SOLORZANO*, ALEXIS GARRABRANT, WILLIAM LIPPITT, SUNDER SETHURAMAN**

We statistically examined the climate in Tucson over the last 70 years. By analyzing average annual temperatures for Tucson using non-parametric methods, we judge whether the temperature in Tucson is warming year to year. Specifically, we were concerned with the mathematics of records. A record is a value which is greater than all previous values in the data set. The mathematical theory of records is understood through Renyi's theorem. On the other hand, records can be understood statistically via the Rosenbaum T-sample test, which measures if the record structure of a time series data set changes. In a data set examined, we studied the average temperature data from 1949 to 2019 at the Tucson International Airport available through NOAA. We divided the data into two equal sets to see how the distribution of the temperatures has changed from the first to the second half. By putting the data through code, we checked how many times the record temperature in the first half was broken in the second half of the data set and applied the aforementioned Rosenbaum test. Through estimation of Type I and Type II error, we ascertained that there were many record-breaking values in the second half of the data, more than would be probable if the temperature each year had the same distribution.

INVESTIGATING CRHID1

***JULIAN SOMERS*, MARK BEILSTEIN**

Plant genomes can be separated into two main categories: protein coding DNA regions and noncoding ones. Researchers have found that transcription occurs in both regions, but only transcripts produced from coding regions are translated into proteins. Since the discovery of DNA, RNA, and proteins, coding RNA and their respective proteins have been studied in depth. However, noncoding RNA have yet to reach similar levels of characterization in the literature despite growing evidence showing critical regulatory functions. One goal of our lab is to identify noncoding RNAs that affect plant growth, development, and survival. A specific noncoding RNA we are focusing on, designated HIDDEN TREASURE 1 (HID1) (Wang et al. 2014), was first identified in *Arabidopsis thaliana* and characterized as a mediator of photomorphogenesis by red light. We subsequently discovered three homologs of HID1 in *Capsella rubella*, a close relative of *Arabidopsis thaliana*. These homologs include highly conserved regions correlating to those important for the predicted structure of HID1 in Arabidopsis thaliana. Currently we are using CRISPR-Cas genome editing to generate mutants that will allow us to decipher the molecular roles of the HID1 homologs in *Capsella rubella* and determine which homologs, if any, share the function of *Arabidopsis thaliana* HID1. We plan to characterize our CRISPR-Cas mutants and perform phenotypic analyses under red light. Results from this research will allow us to refine our understanding of the role long non-coding RNAs play in the perception of light information by plants. This research is supported in part by the Undergraduate Biology Research Program with funds from the Office of Research, Innovation & Impact.

THE ROLE OF ATG5 IN *TOXOPLASMA* CLEARANCE IN PRIMARY MURINE NEURONS

***TESSA SPANGLER*, CHANDRASEKARAN SAMBAMURTHY, ANITA KOSHY**

Toxoplasma gondii is a common intracellular parasite that naturally causes a chronic, asymptomatic infection in the brain of humans and rodents. Within the brain, *T. gondii* primarily persists or encysts in neurons. While this neuron persistence was thought to be secondary to neurons being unable to clear intracellular parasites, recent work from the Koshy lab questions this dogma. Using a novel mouse model that enables the permanent marking and tracking of brain cells injected with parasite protein, the Koshy lab has shown that *T. gondii* almost exclusively interacts with neurons. This exclusivity is most likely why the parasite encysts in neurons. In addition, about 95% of these interacted neurons do not harbor parasites, raising the possibility that neurons may be clearing intracellular parasites. Our preliminary data suggests that, akin to IFN-γ stimulated non-neuronal cells, IFN-γ stimulated murine neurons can also clear intracellular parasites. In non-neuronal cells, Atg5, a protein involved in the autophagy pathway, is essential for clearance of intracellular parasites. The goal of this project is to determine the role of Atg5 in neuronal clearance of *T. gondii* in vitro. We hypothesize that altering the expression of the Atg5 gene in neurons will impact the parasites ability to persist in neuronal cells. To test this hypothesis, I will be utilizing plasmid DNA cloning to

engineer plasmids capable of deleting or overexpressing the Atg5 gene. Lentivirus transduction will then be used to package these plasmids and deliver them into the primary neurons. After validating the knockout or overexpression of Atg5 in neurons, neuronal cultures will be IFN-γ stimulated and infected with parasites. After 24 hours, I will analyze these cultures for the percentage of infected neurons that harbor parasites. Overall, this work will demonstrate how altering the expression of the Atg5 gene in neuronal cells affects the clearance of intracellular *T. gondii* parasites. This project is funded by the Office of the Provost through the Undergraduate Biology Research Program.

HUMAN PAPILLOMAVIRUS EARLY GENES AS A MECHANISM FOR INNATE IMMUNE SYSTEM EVASION THROUGH THE CGAS-STING PATHWAY

***ERICA SPENCE*, BRITTANY UHLORN, SAMUEL K. CAMPOS**

Human Papillomavirus (HPV) is the most common sexually transmitted infection in the United States and in the top three worldwide, infecting up to 80% of the population, and causing around 5% of cancers including almost all cases of cervical cancer. Previous work has found that HPV E6 and E7 oncogenes interfere with p53 and pRb function. E6 and E7 inhibition of p53 and pRb lead to uncontrolled cell growth which underlies HPV's oncogenicity. The cGAS-STING pathway is an innate immune pathway that senses cytosolic DNA causing the phosphorylation and activation of STING and IRF3 to initiate antiviral interferon (IFN) responses. Our preliminary data suggest that HPV18-immortalized keratinocytes, which express physiological levels of HPV oncogenes, have a defective cGAS/STING response. We hypothesize that E6 and/or E7 alone may inhibit cGAS/STING antiviral responses, in addition to p53 and pRb. To test this hypothesis, we are generating a panel of HaCaT keratinocyte lines that will stably express PV oncogenes either alone (HPV16 E6, HPV16 E7) or in combination (HPV16 E6/E7, HPV18 E6/E7, MusPV1 E6/E7) and testing cGAS/STING responses to exogenous dsDNA plasmid. If our hypothesis is correct, there will be lower levels of pIRF3 and pSTING in some E6 and/or E7-expressing cells compared to GFP-expressing- or parental HaCaT controls. This approach will hopefully enable us to assign cGAS/STING antagonisitic functions to E6 and/or E7 oncogenes and lay the foundation for future mechanistic studies. Understanding mechanisms of specific oncogene inhibition of cGAS/STING innate immune responses will advance our understanding of how high-risk HPV may evade the innate immune system to establish persistent infections, the risk factor for HPV-associated cancers. This work was funded by the National Institute of Allergy and Infectious Diseases (NIAID) of the National Institutes of Health (NIH) and the Alfred P. Sloan Foundation. My Undergraduate Biology Research Program position was funded by the Office of the Provost and the College of Science.

DEVELOPING A HUMAN IPSC-BASED NEURONAL MODEL TO STUDY CHILDHOOD EPILEPSY

***SHRUTHI SRINIVASAN*, MANDI J. CORENBLUM, MICHAEL F. HAMMER, LALITHA MADHAVAN**

Epilepsy is the most common childhood neurological disorder in the United States, affecting approximately 450,000 children under the age of 17. While over 50% of these cases are idiopathic, recent studies from the Hammer lab have elucidated a single missense mutation in the SCN8A gene as the genetic cause of a rare form of early infantile epileptic encephalopathy. In order to study the effects of this mutation, we have generated induced pluripotent stem cell (iPSC) lines from the cryopreserved cord blood of an index patient with a known gain of function mutation in the SCN8A gene, along with age and sex matched controls. Specifically, the iPSCs were generated using a sendai viral transduction method and fully characterized for their capacity for self-renewal and differentiation. We have also established protocols to convert the iPSCs into cortical neurons in order to study SCN8A mechanisms at a neuronal level. Using a highly efficient, serum-free, SMAD inhibition monolayer culture method, iPSCs were first induced into neural precursor cells (NPCs) and then driven to become a mixed excitatory (glutamatergic) and inhibitory (GABAergic) population of forebrain-type cortical neurons. The cells were assessed using multiple markers to confirm their NPC, neuronal, cortical and subtype-specific identity. Currently, we are in the process of comparatively analyzing the transcriptional profile and phenotype of SCN8A and control cell lines at different stages of development during the iPSC to cortical neuron continuum. In particular, we are using RNA sequencing, morphological analysis, as well as viability, mitochondrial, redox and immunocytochemical assays to infer molecular processes that may contribute to SCN8A epileptogenesis. We expect that these studies will provide novel information on the mechanisms underlying SCN8A epilepsy,

which will have broad relevance to advancing our understanding of, and development of treatments for epileptogenesis as a whole.

SELECTIVE DOMAIN INSERTION CONTROL OF SRC KINASE

***DESERAE STANERSON*, SAM SUGERMAN, INDRANEEL GHOSH**

There are hundreds of kinases in the body that send signals to control important cellular processes. These kinases all have structurally similar ATP binding sites even if they signal cellular functions that may be vastly different. One method to inhibit the activity of kinases is to use a small molecule inhibitor that inhibits the binding of ATP to these kinases, but this often inhibits many kinases since the ATP binding sites are structurally similar. The current need is a way to study specific kinases in a selective and titratable fashion such that a specific cellular pathway may be controlled and studied. A loop insertion site in human Src kinase was determined by using a sequence alignment homology strategy. Our approach to achieving specific, titratable control of Src is to clone a peptide into a loop insertion site on the kinase to indirectly control Src activity through a specific binder of the inserted peptide. The current work on this project is to create and validate a protein, BfL-1 mutant, that does not bind any endogenous BH3 peptide domains. I am currently using phage display to select for a mutated BH3 peptides that specifically bind the mutated Bfl-1 protein. The new selected peptides will be tested in the context of Src kinase. This system can be potentially applied to study cell signaling. This research was supported in part by the Undergraduate Biology Research Program with funds from the BIO5 Institute and the Department of Biomedical engineering.

APEX2-MEDIATED PROXIMITY LABELING IN *DICTYOSTELIUM DISCOIDEUM*

***JAMIE TAKASHIMA*, A.F.M. TARIQUL ISLAM, PASCALE G. CHAREST**

Characterizing the signaling mechanisms controlling directed cellular migration, or chemotaxis, is critical for understanding and combating the spread of metastatic cancer. *Dictyostelium discoideum* is an excellent model organism for the study of chemotaxis due to its powerful chemotactic response to the signaling molecule cAMP. However, methods for the identification of new protein-protein interactions in *Dictyostelium's* signaling pathways are limited. Here, we describe progress towards a method for interrogating protein-protein interactions in *Dictyostelium* using the APEX2 proximity-dependent labeling system. The work described here demonstrates that in *Dictyostelium*, exposure to osmotic stress permits APEX2-mediated labeling. These preliminary findings pave the way for large-scale studies of chemotactic signaling networks in *Dictyostelium*. The described research was supported by the National Institutes of Health (NIH) under award number R01GM131200-01A1, the American Cancer Society award number 127940-RSG-15-024-01-CSM, and the Arnold and Mabel Beckman Foundation.

DETERMINING THE IMPACT OF SURFACTANT PROTEIN A DURING HOUSE DUST MITE CHALLENGE

***ASHLEY TOLTON*, KENNETH J. ADDISON, JULIE G. LEDFORD**

Human lung surfactant protein A (SP-A) is an oligomeric octadecamer comprised of products encoded by the two functional SP-A genes, SP-A1 and SP-A2. Previous studies have shown that the two main allergens from House Dust Mite (HDM), Der p1 and Der f1, degrade and inactivate SP-A. Previously we found that SP-A1 and SP-A2 differed in their ability to reduce HDM-induced inflammation. As compared to their saline controls, all groups of HDM-challenged mice had significant increases in eosinophils, a type of white blood cell associated with asthma. The SP-A2 expressing mice, compared to the SP-A -/- and WT groups, had the highest percentage and number of eosinophils in the BAL; in contrast, they had the lowest number of eosinophils in the lung

tissue and significantly decreased Muc5AC gene expression in the lungs. In the mediastinal lymph nodes, SP-A2 expressing mice had significantly reduced type-2 markers of inflammation (IL-4:IFN-γ ratio) as compared to SP-A1 expressing mice. Therefore, it appears that in our mouse model, SP-A2 may be more protective than SP-A1 in attenuating the type-2 immune response to HDM challenge. We hypothesized that this difference in activity may be attributed to amino acid variation within the sequences that result in cleavage and breakdown of SP-A by HDM-associated proteases. Over a 48-hour incubation, we discovered that recombinant SP-A2 expressing a lysine at amino acid position 223 was degraded more than if a glutamine was in position 223. In extracted human SP-A, made up of both SP-A1 and SP-A2, we found that neither the Q/Q nor the Q/K variations were broken down, suggesting that SP-A1 may play a role in stabilizing SP-A2. For use in future mechanistic studies, we want to look at how HDM exposure impacts the production, secretion, and breakdown of SP-A using an ex vivo system in which we culture human lung alveolar type II cells that express both SP-A1 and SP-A2 gene products. Grant funding: National Institutes of Health (NIH) under award number HL125602 and the Environmental Health Sciences – Transformative Research Undergraduate Experience (EHS-TRUE) through the National Institute of Environmental Health Sciences Grant #1-R25-ES025494.

STRESS ALTERS THE EXPRESSION OF CRITICAL STRESS GRANULE COMPONENTS IN AGING RATS

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RNA stress granules (SGs) are non-membranous organelles that condense during cellular stress, such as oxidative and osmotic pressures, and inhibited translation initiation. Depending on the cell type and stress, SGs sequester translation initiation factors and non-translating RNA protein complexes. SGs have been proposed to modulate signaling pathways in normal stress response, and shown to increase fitness, protect RNA from damage, and delay the aggregation of proteins linked to neurodegeneration during stress. However, dysregulation of SG formation and clearance has been linked to neurodegenerative diseases and is also disrupted in aging. Because age is the biggest risk factor in neurodegeneration, we are interested in how SG dynamics are affected in aging. To understand these mechanisms, we profiled the changes in SGs during aging and stress by examining the expression of genes critical to altering SGs dynamics and translation initiation in young (6 months, n=6), middle-aged (11-12 months, n=8), and old rats (24-25 months, n=8). The stress condition for the rats was one second of 85mA shock with a 1-hour recovery. Some of the genes profiled were G3BP1, critical for SG formation, TIAR, known to modulate stress granules, PABP, TDP-43, EiF2alpha, and Ataxin-2, all known to associate with SGs. Western blots and qPCRs found variable expression of these genes in the hippocampus, prefrontal cortex, and cerebellum throughout aging and stress. These varying expressions could impact the formation and dynamics of SGs in the cells even though we cannot directly quantify SGs. In aging, varying dynamics could contribute to decreased stress resiliency. Following stress, alterations in SG components could represent a feedback where long-term stress alters future stress responses. In *Drosophila*, SG dynamics in response to stress appears to vary with age compared to the non-stressed control group. Further investigations will isolate SGs from *Drosophila* to examine how components associated with these RNA/proteins granules change with aging and stress, and how those conditions impact SG dynamics. Funding: The National Institutes of Health (NIH) under award number RO1NS091299, ADC pilot P30 AG019610 to DCZ, the McKnight Foundation to CAB, the Undergraduate Biology Research Program with funds from the Office of Research, Innovation & Impact to MDT, and the Arnold and Mabel Beckman Foundation to RE.

DETERMINING THE ROLE OF BRAIN-TYPE CREATINE KINASE IN ASTHMA

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Creatine kinase (CK) is an enzyme that regulates cellular energy homeostasis by catalyzing the reversible transfer of phosphates from ATP to creatine, allowing for energy to be stored in phosphocreatine. There are several cytosolic and mitochondrial isoforms of creatine kinase, and the three dimeric cytosolic isoenzymes are CK-MM (muscle), CK-BB (brain), and CK-MB. While creatine kinase is present in all tissues, the distribution of isoenzymes varies across tissue types. Brain-type creatine kinase

(CKB) has been found to be the predominant isoenzyme present in normal human lung tissue. Its role in the lungs is not yet well understood, however, CKB has been implicated as a possible biomarker for lung disease. Using cell and mouse models of asthma, this study investigates the role of creatine kinase in asthma. We show that CKB expression is limited to ciliated cells in the airway, and that CKB expression initially decreases following house dust mite (HDM) exposure to induce allergic conditions but is significantly upregulated after five days in mice and in human nasal epithelial cells. Inhibition of CKB using BU99006 during HDM exposure in mice resulted in enhanced mucus production, which was not dependent on inflammatory cell populations. In pulmonary function tests conducted on the FlexiVent to methacholine challenge, we observed significantly increased total respiratory system resistance (Rrs), total airway elastance (Ers), and resistance of the conducting airways (Rn) in mice exposed to HDM and treated with CKB inhibitor as compared to HDM challenged mice without CKB inhibitor. These results suggest that CKB may play a role in regulating mucociliary clearance, which could aid in restoring normal pulmonary function in the context of asthma. Funded in part by the Environmental Health Sciences – Transformative Research Undergraduate Experience (EHS-TRUE) through the National Institute of Environmental Health Sciences Grant #1-R25-ES025494 and the Western Alliance to Expand Student Opportunities (WAESO) Louis Stokes Alliance for Minority Participation (LSAMP) National Science Foundation (NSF) Cooperative Agreement No. HRD-1101728.

INTERGENIC BUREAUCRACY: AN INSIDE LOOK AT SEGMENTATION GENE REGULATION IN *TRIBOLIUM CASTANEUM*

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Vertebrates, annelids, and arthropods, while in evolutionarily distant clades, all go through segmentation during development. This process is driven by a cycle of periodic transcription factor (TF) gene expression. In *Tribolium castaneum*, there is a three gene pair-rule oscillator of *even-skipped* (*eve*), *odd-skipped* (*odd*), and *runt* (*run*) with a posterior activating gradient from the maternal effect gene *caudal* (*cad*). This forms a bare-bone negative feedback loop with no known secondary regulators. However, it is currently unknown if characteristics of other segmentation models, such as an anterior wavefront or a method of local synchrony exist in *Tribolium*. In addition, it has been shown that *Tribolium* segmentation exhibits variable periodicity. This suggests that there are additional uncharacterized regulators of this system. Previously, an MCAST analysis created a map of predicted binding sites of clusters of TF's based on *Drosophila melanogaster* models. Our goal is to create mCHERRY promoter fusions, using Gateway cloning, of enhancer regions previously identified using MCAST. By creating these constructs, we hope in the future to create transgenic lines of *Tribolium* using p-element transposition. Then, by observing the mCHERRY expression we will be able to characterize what enhancer regions are important for segmentation gene expression. This will allow for future analysis into novel upstream regulators of the *Tribolium* segmentation process.

ALPHA-SYNUCLEIN OVEREXPRESSION LEADS TO REDUCED SINGING IN A ZEBRA FINCH MODEL OF PARKINSON'S DISEASE

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Overexpression (OE) of the gene that encodes alpha-synuclein (α-syn), SNCA, has been causatively linked to Parkinson's disease (PD) which is a disease that results in impaired motor functioning, including vocal deficits. Most PD patients' vocal deficits manifest as monotonous voice, reduced loudness, and decreased pitch, but the molecular and cellular mechanisms that underlie these changes are not well understood. The α-syn OE models in rodents have reproduced some of the vocal symptoms seen in PD but lack of a well-characterized rodent vocal circuit makes interpretation difficult. The zebra finch songbird, however, has a well-characterized vocal circuit that is similar to the human vocal circuit. Here, we use the zebra finch to study the underlying molecular and cellular mechanisms that lead to vocal deficits in PD. To do this, we drove OE of human wild-type α-syn in Area X, a song-dedicated region, using an adeno-associated virus (AAV) in one group of birds and green fluorescent protein in a control group of birds. Song was collected and analyzed before and after injection of the virus. Our findings show that AAV-α-syn birds sang less two months post-injection compared to control birds. Current efforts are focused on validating

that α-syn is overexpressed in Area X neurons. Vocal dysfunction in our AAV-α-syn zebra finches supports using the finch model to identify novel molecular targets of α-syn OE. This research was supported in part by the Undergraduate Biology Research Program with funds from the Office of the Provost.

THE EFFECT OF SILENCING PKC-δ+ NEURONS ON THE LATENCY TO APPROACH FOOD AND TERMINATION OF FEEDING

***KEVIN VO*, HAIJIANG CAI, MATTHEW SCHMIT**

Protein Kinase C-delta (PKC-δ) neurons are GABAergic cells located in the lateral portion of the Central Amygdala (CeA), these neurons have a role in a neural circuit involved in feeding behaviors. Activation of PKC-δ+ neurons cause a decrease in the total intake of food in mice. Additionally, in vivo calcium imaging reveals PKC-δ+ cell activity increases before the approach to food and during the end of a feeding bout. These neurons therefore may be involved in the initiation of feeding as well as the termination of a feeding bout. We hypothesized that silencing PKC-δ+ neuron activity decreases latency to approach food in both normal and anorexigenic conditions. For the termination of feeding we hypothesized silencing PKC-δ+ would increase the total time of feeding in fasted mice. In current work, we expressed halorhodopsin (NpHR3.0) in only the PKC-δ+ neurons of the CeA, allowing these neurons to be quickly silenced with light through an implanted optic fiber. We studied the effect of silencing these neurons on the latency to approach three types of food: normal food, novel food (a water-soaked pellet), and previously aversive food (novel food pellets presented after Lithium Chloride injection). To study the termination of feeding we silenced PKC-δ+ neurons only during a feeding bout. Data indicates that silencing these neurons decreases the latency to approach food for all three conditions for 24-hour fasted Halorhodopsin mice compared to control mice and that there is no difference for the length of a feeding bout between both groups. This suggests that PKC-δ+ neurons have control over latency to approach food, supporting our previous hypothesis. However, the termination data contradicts our previous predictions. Further exploration of PKC-δ+ neurons' role in the initiation of a feeding will be conducted to reveal how their activity negatively regulates feeding. This work is supported by the National Alliance for Research on Schizophrenia & Depression (NARSAD) Young Investigator Award from the Brain & Behavior Research Foundation, the Foundation for Prader-Willi Research, the Klarman Family Foundation, and by the Undergraduate Biology Research Program with funds from the Office of Research, Innovation & Impact.

INVESTIGATING CYCLOSPORINE A-INDUCED KIDNEY TOXICITY IN MICE WITH DECREASED EXPRESSION OF CYTOCHROME P450 REDUCTASE

***AUSTIN RAJ WATSON*, XINXIN DING, XIANGMENG WU, ARIELLE JOEY DE LA CRUZ**

Cytochrome P450 enzymes are necessary monooxygenases that play important roles in the biotransformation of xenobiotic substances, like medications, into metabolites that are more easily removed from the body. Some of these metabolites can be toxic, which leads to the drug's adverse effects, whereas others may have therapeutic effects, as in the cases of prodrugs. Most of these P450 enzymes are located within the liver, the main site of drug metabolism; however, P450s are also expressed in other parts of the body, including the intestinal tract, kidneys, and the brain. Controlled by a myriad of different factors including genetic polymorphisms, hormone levels, age, and sex, cytochrome P450 expression in humans can vary greatly, which results in a diverse range of drug metabolizing capabilities. This study aims to look at one of the essential factors shaping the functionality of cytochrome P450 enzymes: the cytochrome P450 reductase enzyme (CPR), which catalyzes the electron transfer from NADPH to cytochrome P450. According to a previous study, a substantial reduction in CPR expression had significant impacts on P450-mediated xenobiotic metabolism in mouse tissues demonstrated by decreases in the clearance rates of pentobarbital. With CPR deficiency being an under-reported and under-investigated condition, there is a need to reveal additional phenotypes in the context of xenobiotic metabolism. This study examined the nephrotoxicity of the immunosuppressive drug cyclosporine A in a mouse model with significant CPR deficiency (named Cpr-low), which is an animal model for CPR deficiency in human patients. Mice were given a dose of 20 mg/kg/day of cyclosporine A for seven days and plasma samples were taken at zero, five, and seven days for analysis. A blood urea nitrogen (BUN) assay was conducted to

monitor kidney function of drug-treated mice. Our initial results indicate a dose of 20 mg/kg/day for seven days was insufficient to induce kidney toxicity in CPR-low mice. Future studies are needed to identify a proper dosage of cyclosporine A to induce kidney toxicity in either wild-type or CPR-low mice. This research was supported by an American Society for Pharmacology and Experimental Therapeutics (ASPET) Summer Undergraduate Research Fellowship and the College of Pharmacy.

MORE EXQUISITELY ADAPTED SPECIES HAVE LOWER STRUCTURAL DISORDER IN VERTEBRATE PROTEIN DOMAINS

***CATHERINE WEIBEL*, JENNIFER JAMES, SARA WILLIS, PAUL NELSON, JOANNA MASEL**

Past work in the Masel lab suggests that protein domains become more structurally ordered with age. We studied the relationship between the effectiveness of selection and the Intrinsic Structural Disorder (ISD) of Pfam domains across 120 vertebrate species. Using the phylostratigraphy database of James et. al, we compiled the IUPred2 ISD estimates for the Pfams of 120 vertebrate species and estimated ISD Species effects using a mixed linear model with species as fixed effect and Pfam as random effect, thus controlling for Pfam composition across each species genome. We then calculated each species codon adaptation index (CAI) from genomic codon frequencies and calculated each species total GC content (from genic and intergenic regions) using in-house scripts, and linearly modeled CAI vs ISD and Total GC vs ISD. Initial correlations between ISD and CAI indicate that well adapted species tend to have low ISD (Spearman's R = -0.81, p value < 2e-16). Surprisingly, the initial GC~ISD relationship is weak and not significant (Spearman's R = 0.27, p value = 0.07). To correct for phylogenetic confounding and pseudo replication, we transformed CAI, Total GC, and ISD Species Effect data using Phylogenetic Independent Contrasts (PIC) and re-plotted. Phylogenetically controlled linear models found a strengthened relationship between Total GC content and ISD (Spearman's R = 0.37, p value = 4.6e-6), indicating that either PIC is artificially amplifying a small %GC signal or that mutation bias may have a role in driving structural disorder in protein domains. We plan future work to remove the effect of mutation bias. Phylogenetically controlled linear models soundly confirmed that well adapted species have lower ISD domains than poorly adapted species (Spearman's R = -0.58, p value = 8.5e-8), thus adding evidence to the case of directional protein evolution. This research is supported by the Arnold and Mabel Beckman Foundation Scholars Program and the John Templeton Foundation.

A MOUSE MODEL TO EXAMINE MECHANISMS UNDERLYING POST-CONCUSSION SYNDROME

***TROY WEINSTEIN*, JILL RAU, JANICE OYARZO, FRANK PORRECA**

Post-Concussion Syndrome can develop secondary to mild traumatic brain injuries. Symptoms include severe headache, dizziness, mental fogginess, memory difficulty, anxiety, and depression and can linger anywhere from days to years. In the lab of Dr. Frank Porreca, the team developed a translational model of concussion in the mouse which will aid in the understanding and treatment of many of these debilitating symptoms. An unrestrained free weight-drop system was employed. This model allows for free rotation of the head, which is consistent with human concussions from direct hits such as in sports injuries, motor vehicle collisions, and falls. This novel mouse model goes further to illicit latent stress-evoked responses by utilizing bright light as a stressor after resolution of the initial concussive symptoms. Again, this reflects the human condition in that a variety of influences such as physical, cognitive, and emotional stress can worsen or provoke symptoms in an individual with Post-Concussion Syndrome. All mice were anesthetized using isoflurane and either received a mild traumatic brain injury (mTBI) via the free weight drop model (mTBI group) or received no treatment (sham group). Following the mTBI, effects of concussion were assessed using the open field assay. Evoked pain responses were also evaluated in both the face and hind paw using the von Frey apparatus. mTBI mice spent significantly more time in the perimeter (2-inch peripheral border) of the open field apparatus and reared more, representing an increase in anxiety. mTBI mice also showed significantly more facial and hind paw allodynia than sham mice in the days just after mTBI (or sham). Bright light stress (BLS) thirteen days after mTBI re-evoked initial behaviors in the open field assay and allodynia testing. Utilizing this novel mouse model could prove useful when testing future pharmaceuticals for the treatment of Post-Concussion Syndrome. The project was supported by the College of Medicine

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INVESTIGATING THE EFFECTS OF FUSED IN SARCOMA (FUS) ON IN-VITRO T7 TRANSCRIPTION

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Fused in Sarcoma (FUS) is an RNA-binding protein where mutations in its polypeptide chain have been linked to the development of frontotemporal dementia and amyotrophic lateral sclerosis (ALS), a rare and fatal neurodegenerative disease. Previously, FUS has been shown to exhibit RNA-dependent binding with the carboxy-terminal domain (CTD) of RNA Polymerase II in addition to regulating CTD phosphorylation during transcription. Our study focuses on the effects of FUS on in-vitro transcription in a novel environment containing the phage T7 Polymerase. Here we present an increase in RNA product in these transcription reactions in presence of FUS despite having no direct interaction with T7 as confirmed via pulldown. Interestingly, transcription reactions over longer periods of time have also exhibited a greater abundance of R-loops, which are unique assemblies where the nascent RNA can hybridize to the template DNA forming a 3-stranded structure. These are thought have a role in transcriptional stalling, genomic instability, and disease formation. Our future direction is to directly characterize how FUS may behave as an intrinsic transcription factor through its potential regulation of R-loop formation. This work is supported by the American Cancer Society award RSG-18-237-01-DMC, the National Institutes of Health (NIH) under award number R21CA238499-01, and in part by the Undergraduate Biology Research Program with funds from the BIO5 Institute and the Department of Biomedical Engineering.

EVALUATING NOVEL THERAPIES FOR NEURODEGENERATIVE DISEASE USING *DROSOPHILA* MODELS

***HALEY WILLIAMS*, JUDITH TELLO, MAY KHANNA**

With improvements in healthcare, age-related diseases are becoming increasingly common. Two of the major neurodegenerative diseases are Amyotrophic Lateral Sclerosis (ALS) and Alzheimer's disease (AD). Surprisingly, these two diseases appear to be related through the involvement in CD44 proteins. CD44 is a gene encoding a large family of glycoproteins involved in inflammation and neuronal injuries due to inflammation. In AD, C-terminal ends of CD44 proteins are known to be bound by FERM. The FERM protein family is involved in cell-membrane targeting of other proteins and lipids, especially important during wound healing. However, in ALS, the involvement of CD44 is not extensively studied but initial studies indicate the CD44 inflammatory pathway is also involved in ALS pathology. Previous work in the lab identified potential therapeutic compounds for each of the proteins involved in these diseases. We hypothesized that by targeting interactions between proteins implicated in each disease's pathogenesis, the disease progression could be modified. Using *Drosophila melanogaster* models of each disease expressing the human proteins, the effect of each compound on the disease phenotype was assessed using survival curves, Western blot, immunoprecipitation, immunostaining, confocal microscopy, and locomotor assays in both adults and larvae. In ALS models, survival data indicates the compound rTRD01 improves the lifespan of adult flies expressing mutant TDP-43, improves larval locomotion, and decreases aggregation of TDP-43 in larval neurons. Another compound, Pharmacomimetic01, was used to treat AD models and has shown to improve the lifespan of adult flies. Ongoing work investigates the effect of each compound on adult locomotion and aggregation of diseased proteins in larval brains of AD models. Further testing of these compounds as well as the others of interest will elucidate the true therapeutic properties of the compounds and their potential to proceed through the drug discovery-to-market pipeline. This research was supported in part by the Undergraduate Biology Research Program with funds from the Office of Research, Innovation & Impact.

THE DYNAMICS OF EXPLORE-EXPLOIT DECISIONS REVEALS A SIGNAL-TO-NOISE MECHANISM FOR RANDOM EXPLORATION

***SYLVIA ZARNESCU*, S.F. FENG, S. WANG, R.C. WILSON**

Let’s say you go out to a restaurant to eat: you can either order the pizza that you always get-you know for sure you would have a good meal with the pizza, or you could try the new spaghetti on the menu, which could potentially be your new favorite dish. If you choose to exploit too often, you might end up eating pizza for the rest of your life. Conversely, if you are too explorative, you might experience a lot of bad meals. This simple conundrum choosing between what you know and what you don't is called the explore-exploit dilemma. Research has suggested a number of heuristics which people can use to solve explore-exploit decisions in practice. These include an explicit bias for information (directed exploration) and the randomization of choice (random exploration). In this work we focus on random exploration, asking in particular, how can behavioral variability be controlled by the brain? By modeling the explore-exploit choice using a drift diffusion process, we identify two distinct mechanisms by which behavioral variability could be controlled, and by fitting the diffusion model to human behavior, we determine which of these mechanisms humans actually use. More specifically when applied to an explore-exploit choice the drift diffusion model assumes that the decision is made by accumulating a noisy value-based signal towards one of two bounds: one for explore and one for exploit. Behavioral variability can then be controlled by two different parameters: the signal-to-noise ratio of the accumulation process (drift rate) and the separation between the decision bounds (threshold). By fitting the model to human choices and reaction times in a Horizon Task commonly used in the field, we determined that numerically, the change in drift rate has the main effect on random exploration. This provides evidence that random exploration is primarily driven by changes in the signal-to-noise ratio, with which reward information is represented in the brain. Funding Source: The Undergraduate Biology Research Program with funds from the Office of the Provost, and the National Institutes of Health (NIH) under award number R563030210.