University of Central Florida
College of Medicine
Burnett School of Biomedical Sciences

Immunity and Pathogenesis

Molecular Microbiology
Cardiovascular

Cancer

Neuroscience

11th Annual Graduate Research Colloquium
August 21, 2019
College of Medicine, Lake Nona Campus
Images on the cover

Immunofluorescent staining of HeLa cells infected for 1 hour with *Chlamydia trachomatis*. Anti-Chlamydia MOMP (Alexa 488, green) and actin (phalloidin, red). Image submitted by Dr. Susmita Ghosh from the T. Jewett Lab.

Multiplex immunofluorescence analysis of human skin tissue to identify epidermal stem cells. Ki67 (PerpCP7.7, cyan), XPC (FITC, green), MECP2 (Cy3, red), VIM (Alexa647, yellow), ITGB1 (Coumarin, blue), KRT14 (Pacific Orange, grey), DNA (BOBO1, magenta). Submitted by Dr. Thomas Andl.

Immunofluorescence analysis of Murine Tracheal Epithelial Cells (MTECs) showing a well organized f-actin network. Actin (Phalloidin, red), nucleus (DAPI, blue). Submitted by the laboratory of Dr. Tigno-Aranjuez.

Immunofluorescence analysis of Murine Tracheal Epithelial Cells (MTECs) showing a well-defined tight junction. ZO-1 (Alexa 488, yellow-green), nucleus (DAPI, blue). Submitted by the laboratory of Dr. Tigno-Aranjuez.

Gross analysis of colon tissue from mice subjected to an inflammation-induced cancer model (AOM/DSS) showing polyp formation. Methylene blue was used to define the borders of the polyps. Submitted by the laboratory of Dr. Tigno-Aranjuez.

Multiplex immunofluorescence analysis of human oral tissue to identify epithelial stem cells. Ki67 (Pacific Orange, cyan), MECP2 (Cy3, red), ITGB1 (Alexa647, yellow), KRT15 (Coumarin, blue), and TP63 (FITC, green). Submitted by Dr. Thomas Andl.

Immunohistochemistry of lung tissue from a mouse subjected to an allergic airway asthma model showing a mucus plug in one of the airway bronchioles. Periodic Acid Schiff (PAS) stain to detect mucosubstances was performed. Glycoproteins, glycolipids, and mucins appear magenta. Submitted by the laboratory of Dr. Tigno-Aranjuez.
Program

10:30 – 11:30 Registration and Poster Setup (COM 1st floor)
11:30 – 12:00 New Graduate Student Introduction (COM 102)
12:00 – 1:00 Welcoming Luncheon (Burnett Building 103)

1:00 – 2:00 Plenary Talk (COM 102) Dr. Pawan K. Singal

“Oxidative/nitrosative stresses and heart failure”

2:00 – 3:30 Poster Session

3:30 Faculty Wine and Cheese Reception with Speaker
(Burnett Building Lobby – Faculty only)
UCF College of Medicine
11th Annual Graduate Research Colloquium
August 21, 2019

Keynote Speaker

Pawan K. Singal, PhD, DSc, LLD (Hon) (Naranjan Dhalla Chair)

Professor of Physiology and Pathophysiology
Institute of Cardiovascular Sciences,
St. Boniface Hospital and the University of
Manitoba, Winnipeg, Canada.

“Oxidative/nitrosative stresses and heart failure”
Medical Education Building COM 102
College of Medicine, Lake Nona Campus
1:00 - 2:00 pm

Internationally known for his work on oxidative stress and heart failure, Dr. Singal has made significant contributions in our understanding of the sequale of heart failure due to doxorubicin, chronic pressure overload as well as myocardial ischemia/reperfusion. He has published 280 papers, has co-edited 31 books and trained more than 100 students, fellows and visiting scientists. He has received more than 90 national and international recognitions. The University of Manitoba has established an award in his name called ‘Pawan K. Singal Award for Graduate Students in Cardiovascular Sciences’. His name has been added to the Wall of Fame in the University Centre at the University of Manitoba recognizing his outstanding teaching skills and research. Additionally, the University of Winnipeg has bestowed to him an Honorary Doctorate of Law.
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MD-PhD Biomedical Sciences Program

Amanda Cox

Education: B.S. Biochemistry and Biomedical Sciences, UCF, 2016; UCF Medical School 2017
Growing up: Born in Cape Canaveral, Florida and raised in Titusville, Florida.
Research/Professional Interests and Future Goals: In undergrad, I worked for Dr. Kolpashchikov in Biochemistry using deoxyribozyme binary sensors as a diagnostic tool for Tuberculosis. I also worked one year with Dr. DeCampli at Arnold Palmer Children’s Hospital in the Pediatric Cardiothoracic Surgery Department. Both of these have increased my interest in genetic and congenital research. My goal as a future research physician is to run my own lab at a medical school where I can bridge the gap between research and medical practice.
Hobbies: Painting, watching movies, playing board games and various outdoor sports.
Faculty Mentor: Dr. Annette Khaled

Alexander McClanahan

Education: B.S. Biochemistry, Florida Southern College; UCF Medical School 2017
Growing Up: Born and raised in nearby Maitland, FL
Research/Professional Interests and Goals: I am an MD-PhD student researching brain-computer interfaces in neuroscience; I hope to go into neurosurgery or neuroradiology as a physician-scientist.
Hobbies: Playing basketball, learning new things, reading, traveling
Faculty Mentors: Dr. Kiminobu Sugaya & Dr. Brian Kim
PhD Biomedical Sciences Program

Abha Banerjee
Education: B.S. Biotechnology, PSU, 2015; M.S. Biotechnology, USF, 2018
Growing Up: I was born in Virginia. I have also lived in California, India, Pennsylvania, and Florida.
Research/Professional Interests and Goals: I did neurobiology research during my graduate degree, investigating XLID (X-linked intellectual disability). I am interested in studying immune response to cancer cells and pathways involved in tumor development and resistance.
Upon obtaining my Ph.D., I plan to pursue a career in R&D.
Hobbies: Digital graphics and photography.

Raúl Baños
Education: B.S. Biology. St. Thomas University, 2018
Growing Up: Miami, Florida
Research/Professional Interests and Goals: I am driven to study the complexities of the human brain to obtain vital knowledge of neurodegenerative disease pathology. With prior experience in spinal cord and traumatic brain injury rejuvenation and Alzheimer’s disease, I plan to continue my research beyond my graduate studies in search of life changing treatments or potential cures. I also intend on educating and sharing knowledge with future scientists to ensure the progression of high-quality accurate science.
Hobbies: Martial arts, fitness, outdoor activities, reading, and learning to cook.

Jordan Beardsley
Education: B.S. Biomedical Sciences, UCF, 2017
Growing Up: Jupiter, Florida
Research/Professional Interests and Goals: I am interested in the metabolic and immunological impact on tumor microenvironment, particularly in the context of a pre-metastatic niche. I would like to use murine models to understand disease progression and determine potential points of therapeutic intervention. After graduate school, I intend to diversify my research experiences before entering academia.
Hobbies: Music, road trips, and spending time with my dogs
Ariege Bizanti
Education: B.S. Biomedical Sciences, UCF, 2016; M.S. Biomedical Engineering, UCF, 2018
Born: Scotland. I grew up in Libya.
Research/Professional Interests and Goals: I completed a cellular-biomechanics research project with Dr. Robert Steward during my master’s degree, investigating the effects of different substrate stiffness on astrocytes biomechanics. The chosen substrate stiffness mimicked brain tumors and the goal was to observe how they affect the healthy brain environment and mechanical cues. I aspire to obtain a Ph.D and find a career at a scientific or medical field.
Hobbies: Ping Pong, biking, video editing, swimming.

Charles “Chuck” Didier
Education: B.S. Biomedical Sciences, B.S Biotechnology, UCF 2015; M.Sc. Nanotechnology, UCF, 2019
Growing Up: Born in Dunedin, FL. I moved to Orlando in 2011.
Research/Professional Interests and Goals: During my undergraduate career, I helped perform research on silencing miRNA and their role in regulating prostate cancer. Currently I am focused on researching novel methods for producing Microelectrode Arrays (MEAs) to better interface with neural tissue for more effective prosthetic implants. After obtaining my PhD, I plan to aid industries in bettering their prosthetic technologies.
Hobbies: Cooking, baking, and creative storytelling
Faculty Mentor: Dr. Swaminathan Rajaraman

Joseph Goode
Education: B.S. Biochemistry, FIT, 2019
Growing Up: Melbourne, Florida
Research/Professional Interests and Goals: During my undergraduate career, I investigated the ability of the unicellular alga Chlamydomonas reinhardtii to exhibit quorum sensing as well as worked towards discerning the signaling molecules involved in this process. I plan to further my experience in elucidating biological mechanisms and the molecules involved in order to translate this knowledge towards pharmacological applications. I aim to expand the current understanding of pathological pathways in order to discover treatments that focus on preventative measures.
Hobbies: Cooking, woodworking, and video games
**Noah Greenman**

**Education:** B.S. Biology, James Madison University, 2016; M.S. Biology, James Madison University, 2019.  
**Growing Up:** Loudoun County, Virginia  
**Research/Professional Interests and Goals:** I completed research studying the morphology of the *Limnonectes kuhlii* during my undergraduate degree, and then transitioned to studying multidrug resistant *Salmonella enterica* isolated from stream sediment and poultry litter around the Shenandoah Valley during my master’s. I am interested in pursuing research in the field of systems biology using ‘omics approaches and eventually going on to work in the biotechnology sector.  
**Hobbies:** Lifting, drawing, cooking, hiking, and writing.

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**Md Faqrul Hasan**

**Education:** B.S. Microbiology, University of Dhaka, Bangladesh, 2012; M.S. Microbiology, University of Dhaka, Bangladesh, 2014; M.S. Biotechnology, UCF 2019  
**Growing Up:** Dhaka, Bangladesh.  
**Research/Professional Interests and Goals:** I was involved in prostate cancer and long non-coding RNA research during my master’s study in UCF. I am interested in studying the functional role of non-coding RNAs in cancers and other diseases. After completing graduate study, I want to pursue research career in academia/biopharmaceutical industries.  
**Hobbies:** Reading, playing cricket, and watching movies.

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**George Robb Huhn III**

**Education:** A.A in General Studies, Valencia, 2010; B.S. in Biomedical Sciences, UCF, 2016; M.S. in Biotechnology, UCF, 2019  
**Growing Up:** Venice, Florida  
**Research Interests and Goals:** I am interested in toxin-cell biology and the interactions inside target cells. My goal is to graduate and teach at the university level.  
**Hobbies:** Fishing, shooting, and working on cars.  
**Faculty Mentor:** Dr. Ken Teter
Michael Johnstone
Education: B.S. Biomedical Sciences, UCF, 2017; M.S. Biotechnology, UCF, 2019
Growing Up: Orlando, FL
Research/Professional Interests and Goals: My research interests primarily include bacteriology, enzymology, and the role of trace elements in microbial metabolism and pathogenesis. During my master’s in Dr. Self’s lab, I studied the selenium-vs.-sulfur specificity of a selenocysteine lyase in Enterococcus faecalis. Upon completion of my PhD, I plan on working in industry.
Hobbies: Drumming, tennis, video games
Faculty Mentor: Dr. William Self

Arash Keshavarzi
Education: M.S Biotechnology, UCF, 2019
Growing Up: Tehran, Iran
Research/Professional Interests and Goals: My master project has been discovering new compounds for malaria using new technologies like Artificial Intelligence in Dr. Chakrabarti lab. The model I developed is capable of predicting antimalarial hits’ activity with no need for robots and expensive compound libraries. In the future, I will try to encode gene sequence using new technologies. My plan after Ph.D. is to enter big pharma or start my own biotech company.
Hobbies: Hiking, gym, photography, hanging with friends

Anamaria Morales Alvarez
Education: B.S. Biology, National University of Colombia, 2012; M. Sc. Biochemistry, National University of Colombia, 2017
Growing Up: I was born and raised in Bogotá, Colombia.
Research/Professional Interests and Goals: I did my master’s work on the immuno-monitoring of gastric carcinogenesis. I am passionate about cancer: the etiology, the treatment and the prognosis, but above all I enjoy the topic of cancer immunotherapy. By obtaining my Ph.D., I plan to continue my research and become a PI of my own laboratory.
Hobbies: Cultural events like concerts, theater, cinema
Sarah Noureddine
Education: B.S. Biology, UCF, 2019
Growing Up: Born in Alexandria, Virginia, but I spent a good majority of my life moving from country to country.
Research/Professional Interests and Goals: In the past year, my undergraduate research revolved around generating a Difluoromethylornithine-resistant cell line in a polyamine depletion strategy and investigating the mechanisms by which resistance has transpired. I am interested in applying what experience I have gained to a different field within research – longevity and aging.
Hobbies: Reading, baking, boxing, and photography

Kaylyn Scanlon
Education: B.S. Biology, GCSU, 2019
Growing Up: Raised in Brunswick, GA
Research/Professional Interests and Goals: I worked in a virology research lab during my undergraduate career studying the effects of adenovirus on cellular proteins. I am interested in using viruses as a way to target and terminate cancer cells in the human body. Upon completion of my Ph.D., I plan to either continue in academia as a professor, move into government sector research, or work with the EIS.
Hobbies: Hiking, camping, gardening, painting/photography, and playing guitar

Albert Serrano
Education: B.S Biomedical Sciences, UCF, 2013
Growing Up: Taunton, Massachusetts
Research/Professional Interests and Goals: Via the UCF Summer Research Academy, I gained my first publication on Cholera Toxin trafficking through Lipid Rafts in Dr. Teter’s lab. Upon graduation, I continued working and finalized work on Parkinson’s Disease (α-Synuclein) aggregation via Thioflavin Spectroscopy experiments in collaboration with Dr. Tatulian. Later, I worked on testing vaccines through ELISAs for Sanofi Pasteur as a Senior Lab Analyst and acted as Chemical Hygiene Officer for safety audits. My future aspirations involve further work in industry revolving around bioinformatics and infectious diseases.
Hobbies: Science fiction, programming, cooking.
Faculty Mentor: Dr. Ken Teter
Jonhoi Smith
Education: B.S. Biology, UCF, 2016; M.S. Biotechnology, UCF, 2019
Growing Up: Far Rockaway, New York
Research/Professional Interests and Goals: For my master’s. I investigated NanogP8’s role in Glioblastoma cancer stem cells. For research, I am interested in furthering studies on stem cell genes and their role in cancer stem cells.
Hobbies: Working out, watching TV
Faculty Mentor: Dr. Kiminobu Sugaya

Joseph Vaccaro
Education: B.S. in Microbiology, University of Pittsburgh
Growing Up: Born in Rhode Island, but grew up in rural Pennsylvania in a town a fortieth the size of UCF.
Research/Professional Interests and Goals: Vaccine development
Hobbies: Reading, writing and learning languages
Gretel Arcia Gonzalez
Education: B.S. Biology and Certificate in Forensic Science, FIU, 2017
Growing Up: Miami, Florida
Research/Professional Interests and Goals: My research interests include tissue engineering and regenerative medicine, human stem cell research, and cancer nanotechnology. In the next few years, I seek to expand my knowledge and laboratory experience in the field of biomedical science. My ultimate goal is to help develop groundbreaking nanobiotechnology for the diagnosis and/or treatment of various human diseases.
Hobbies: Fitness, cars, traveling.

Sarah Ashiqueali
Education: B.S. Biology, Boston College, 2017
Growing Up: Cooper City, Florida
Research/Professional Interests and Goals: I plan to conduct research in the Cardiovascular Division. My research will concern cardiotoxicity induced by chemotherapeutic drugs and the potential of cannabinoids in treating this adverse effect. My future academic endeavor includes completion of a dual MD-MBA or MD-PhD program. I aspire to give back to the community with the invaluable scholarship that I will acquire from this university.
Hobbies: Personal training, running, watching the NBA, writing, and traveling. As an undergraduate student, I participated in the Boston Marathon on behalf of the Dana Farber Cancer Institute.
Faculty Mentor: Dr. Dinender Singla

Samjhana Bhandari
Education: B.Tech in Biotechnology, Kathmandu University, 2017
Growing Up: Kathmandu, Nepal
Research/Professional Interests and Goals: During my undergraduate study, I investigated what phytochemicals are present in bamboo leaves and the antimicrobial properties of these phytochemicals. For my master's, I am interested in studying the mechanisms of antimicrobial resistance and development of antimicrobial drugs.
Hobbies: Travelling, reading novels and poetry, yoga
Kevin Borges
Education: B.S. Biology, FIU, 2017
Growing Up: Miami, Florida
Research/Professional Interests and Goals: My research interests include gene therapy, nanotechnology, and immunology. Particularly, I am interested in studying methods of genetic engineering and immune system manipulation to fight off disease. My long-term goal is to further my career through the development and employment of new immunological or genetic techniques to help patients with various types of cancer.
Hobbies: Weightlifting, playing video games, going to the movies, and volunteering to help the elderly.

Erij Elkamel
Education: BSc. Hons. in Biomedical Sciences with minor in Psychology, University of Waterloo, 2019
Growing Up: Waterloo, Ontario, Canada
Research/Professional Interests and Goals: I had the opportunity to contribute to research in nanomedicine and biotechnology focused on drug delivery methods. I would like to continue in research that applies principles of molecular biology and genetics to better understand the mechanisms of diseases to assist in the development of therapies. After completing a master’s degree, I plan to join an MD-PhD or PhD program and later conduct clinical research.
Hobbies: Cinematography, karate, and travel

Omonzejie Imaralu
Education: B.Sc Biochemistry, Unilorin, 2015
Growing Up: Nigeria
Research/Professional Interests and Goals: I am currently interested in studying the molecular mechanisms underlying cancer development and metastasis. Upon completion of my M.Sc, I plan to either pursue further graduate studies to obtain a Ph.D or consider medical school.
Hobbies: Coding, traveling, playing musical instruments
Akhmetzada Kargazhanov
Education: B.S. Biotechnology, Swinburne University, 2017
Growing Up: Almaty, Kazakhstan
Research/Professional Interests and Goals: During my undergraduate career, I was involved in research focused on the analysis of secondary metabolites from endophytic fungi. Later, I participated in research that was focused on cancer immunotherapy (effects of regulatory cells on lymphoma cells) as a laboratory technologist in a research institute in Kazakhstan. I am interested in studying bacteriophages of the antibiotic-resistant bacteria that could be used in phage therapy. Upon completion of my master’s program, I am planning to pursue PhD. Later, I see myself working as a researcher either in academia, non-profit organizations or private companies.
Hobbies: Soccer, table tennis, hiking and trekking.

Piotr Lagod
Education: B.S. Biomedical Sciences, UCF, 2019
Growing Up: Andrzejów, Poland
Research/Professional Interests and Goals: I have a broad interest in the biotechnology field as a whole. I am especially interested in studying induced pluripotent stem cells and their applications in both regenerative medicine and drug screening. I would like to apply my knowledge and experience to advancements in both industry and academia.
Hobbies: Traveling, science fiction movies and books, vintage watches, and consumer electronics

Duyen Nguyen
Education: B.S. Biotechnology, UCF, 2019
Growing Up: Bao Loc, Lam Dong, Vietnam
Research/Professional Interests and Goals: I completed research during my undergraduate degree investigating hyperaccumulation of Helianthus genus (sunflower) via mutation breeding. I am interested in artificial insemination of queen bees and conservation genetics of bees, as well as how bee products can prevent many physical ailments and chronic diseases such as HIV, infections, ulcers, etc. Upon graduation, I plan to either obtain a Ph.D that closely relates to my interests or work in a laboratory where my skills can become handy.
Hobbies: Playing musical instruments, learning new languages, cooking, and hiking.
Joseph Peterson  
Education: Bachelor of Science in Microbiology and Cell Science with a minor in Bioinformatics, UF, 2019  
Growing Up: East Haven, Connecticut  
Research/Professional Interests and Goals: During my time at UF, I have done research in the field of environmental and human toxicology through metal exposures on the organism *Daphnia Magna*. However, my research interests are currently in gene therapy and stem cells. After obtaining my master’s degree, I plan to find a job in a pharmaceutical biotech company for research and development. Eventually, I plan to obtain my Ph.D. and go into stem cell research.  
Hobbies: Running, reading, drawing, playing video games, swimming

Kayli Rohal  
Education: BS Biochemistry, Coastal Carolina University, 2018  
Growing Up: Westerville, Ohio  
Research/Professional Interests and Goals: In my undergraduate career, I completed my research in natural product synthesis. We created Phidianidine derivatives to be used to treat a variety of diseases including Alzheimer’s, cancer, arthritis as well as chronic pain. Moving forward in my career, I am interested in studying the progression and treatment of disease and after obtaining my Ph.D., I would like to work in a research lab.  
Hobbies: Traveling, reading, watching movies and scuba diving

Smitha Shambhu  
Education: B.E. Biotechnology, BMS College of Engineering  
Growing Up: Newbury, England and Bangalore, India  
Research/Professional Interests and Goals: During my undergraduate career, I carried out research on the synthesis and properties of carbon nanoparticles. I am interested in studying about the molecular mechanisms of gene expression and their applications in diagnostics. Upon obtaining my MS Biotechnology degree, I hope to work as a researcher in the biotechnology industry.  
Hobbies: Reading, cycling, baking.
Ameera Shaw
Education: B.S. Biology, Minor Public Health Studies, Stetson University, 2019
Growing Up: Deltona, Florida
Research/Professional Interests and Goals: I completed Lyme disease research during my undergraduate degree and utilized CRISPR-cas9 to investigate the role of specific genes in the development of Ciona embryos. I am interested in studying infectious processes and immunology in relation to infectious diseases. I plan to pursue a Ph.D. in Biomedical Sciences and work for a government or academic institution to continue conducting research in my field.
Hobbies: Reading, photography, traveling, and snorkeling

Ayushi Srivastava
Education: B.Tech and M.Tech Biotechnology (Dual Degree), Kalinga Institute of Industrial Technology, 2019, Bhubaneswar, Odisha, India
Growing Up: Lucknow, Uttar Pradesh, India
Research/Professional Interests and Goals: Having worked on Mesenchymal Stem Cells during my M.Tech thesis dissertation, I'm interested in regenerative medicine. I would like to pursue PhD in this field after my master’s.
Hobbies: Writing, Reading and Poetry
MS Biomedical Sciences

Lauren Minnear

Education: B.S. Biology, UF, 2019
Growing Up: Titusville, Florida
Research/Professional Interests and Goals: My goal is to attend dental school and become a DMD. My hopes are that the knowledge I learn from this program will help me achieve this goal. Currently, I am also a certified dental assistant.
Hobbies: Playing guitar, calligraphy, going to the beach

Alexandra Ware

Education: B.S. Psychology, University of Miami, 2018
Growing Up: Longwood, FL
Professional Interests: At the University of Miami, I completed research in the child psychology department studying the influences most prominent to the categorization of well-known objects and novel objects in young children. I am interested in broadening my spectrum of research. Upon completion of my master’s, I plan to attend medical school in quest of becoming an ER physician or trauma surgeon.
Hobbies: Exercising, dogs, equestrian, Division I Women’s Rowing Athlete in undergraduate

Raven Wright

Education: B.A. History of Science, Medicine and Technology, Johns Hopkins University, 2019
Growing Up: Jacksonville, FL
Research/Professional Interests and Goals: I completed urology research as an undergraduate, studying prostate cancer proliferation and testing of new treatments. I plan to obtain my M.D. and pursue a career as a neonatologist. I am also considering serving as a professor to help guide and inspire young minds.
Hobbies: Reading, listening to music-going to concerts, bowling, trying new restaurants
MS Biomedical Sciences - Neuroscience

**Andrei Gheorghe**

**Education:** B.S. Biomedical Sciences, UCF, 2019; minor in Health Sciences  
**Growing Up:** I was born in Bucharest, Romania and raised in Fort Myers, Florida.  
**Professional Interest:** ER Physician  
**Hobbies/Interests:** Gardening, personal fitness, cars, and videogames.

**Devin Inthavongsa**

**Education:** B.S. Biomedical Sciences, UCF, 2018  
**Growing Up:** Deltona, Florida  
**Research/Professional Interests and Goals:** I conducted migraine research during my junior and senior years of college. I want to further my research on migraines, as well as exploring neurodegenerative diseases like Alzheimer's disease, Huntington's disease, and Parkinson's disease. After obtaining my master's degree, I plan to focus on applying to medical schools.  
**Hobbies:** Cooking, lifting, playing guitar

**Nazifa Khan**

**Education:** B.S. Neuroscience, Christopher Newport University, 2017  
**Growing Up:** Leesburg, Virginia. Born in Bangladesh.  
**Research/Professional Interests and Goals:** I have a research background in studying 3xTg-AD mice and the biochemical effects of methylene blue on the onset and progression of Alzheimer’s disease. I would like to continue research on Alzheimer’s disease, focusing on the pathology and its effects on the hippocampus. My professional goal is to become a physician assistant.  
**Hobbies:** Binge watching Netflix, hiking, kayaking, cooking, and reading
Nicole Kogut
Education: B.S. Health Sciences, UCF, 2019
Research/Professional Interests and Goals: Upon completion of my master’s program, I aspire to attend medical school and become a physician. I particularly enjoy the discipline of neuroscience in addition to pathology and pathophysiology in relation to neurology. I hope to pursue these interests further via research while obtaining my master’s degree.
Hobbies: Writing and rhetoric, herpetology, and playing the ukulele

Chelsea Mapp
Education: B.S. Psychology, UCF, 2016
Growing Up: Bronx, New York
Research/Professional Interests and Goals: I completed the Burnett Honors College thesis program during my undergraduate career where I researched the implications of cerebrovascular burden and sleep impairment in aging. I am interested in researching how stem cells and artificial intelligence can aid in finding treatment/therapy for diseases and disorders of the brain and their role in cognitive functioning. I plan on obtaining my Ph.D in neuroscience, continuing research in the lab, and being an educator.
Hobbies: I enjoy weightlifting and fitness, watching horror movies, reading sci-fi books, and being in the water.

Freya Mehta
Education: B.S Biomedical Sciences, UCF, 2019
Growing Up: Rockledge, FL
Research/Professional Interests and Goals: I currently research under a NanoScience faculty Dr. Debashis Chanda. My current research is focused on detecting biomarkers like Homovanillic acid (dopamine metabolite) prominent in diseases such as Parkinson’s disease directly from blood via a microfluidics-based platform in real time. My goal is to devise a concrete protocol in providing selectivity towards dopamine while detecting it. My personal goal involves pursuing a career in medicine.
Hobbies: I am a big fan of salsa dancing and am currently in the process of learning the basics. Besides dancing, I love hiking and biking trips.
Maria Moreno  
*Education:* B.S Biomedical Sciences, UCF, 2018  
*Growing Up:* I was born and raised in Peru, then moved to Miami in 2005. I have now moved to Orlando to start my UCF journey.  
*Research/Professional Interests and Goals:* I am interested in Crohn’s and IBD related research. My goal is to become a doctor.  
*Hobbies:* Volleyball, working out, dancing

Morgan Rook  
*Education:* B.S. Biology, minors in chemistry and psychology, Florida State University, 2018  
*Growing Up:* Orlando, FL  
*Research/Professional Interests and Goals:* I previously completed research in a yeast genetics lab, studying protein quality control and laminopathies. I have an interest in understanding the polygenic inheritance patterns and mechanisms of genetic epilepsies. Upon finishing my master’s, I plan to further my education, pursuing a medical degree and a career in pediatric neurology.  
*Hobbies:* Hiking with my Australian Shepherd, playing/coaching volleyball, photography, snorkeling and reading.

Joseph Schulz  
*Education:* B.A. Chemistry, Rollins College, 2019  
*Growing Up:* Orlando, Florida  
*Research/Professional Interests and Goals:* In my undergraduate studies, I was able to develop a concise and effective synthesis for a natural product that was shown to be a good inhibitor of the enzyme tyrosinase. In my graduate studies, I am interested in learning the molecular mechanisms that govern synapse interactions in the nervous system, as well as learning about the mechanisms that lead to neurodegenerative diseases. I plan on supplementing my synthetic chemistry experience in my undergraduate with the knowledge of the molecular mechanisms to hopefully develop an effective treatment for Alzheimer’s Disease.  
*Hobbies:* Running, reading, cooking, and playing with my dog Jerri
Reetish Singla
Education: B.S Biomedical Sciences, UCF, 2019
Growing Up: Born Winnipeg, Manitoba, Canada. I now call Oviedo, Florida home.
Research/Professional Interests and Goals: I aspire to go to medical school and pursue my dream to become a doctor. I hope to shadow more physicians and other specialists over the next few years in order to open my eyes to the possibilities of what I would like to practice. I am considering a career in research, medicine, or pursuing an MD-PhD.
Hobbies: Travel, reading, and spending time with friends

Emeil Stewart
Education: B.S. Biomedical Sciences - Neuroscience Track, UCF, 2019
Growing Up: I moved around quite a bit living primarily in Boca Raton and the Fort Myers area.
Research/Professional Interests and Goals: My professional interest is to further advance the clinical and biological sciences through research in neuroscience and other relevant subjects in conjunction with becoming a physician that promotes the betterment of people’s lives.
Hobbies: When I have free time I enjoy watching movies, playing basketball with friends, competitive gaming, and some adventurous outdoor activities.
Angela Bautista

**Education:** B.S. Biomedical Sciences, UCF, 2019

**Growing Up:** I was born and raised in Orlando, Florida alongside my two younger sisters. My parents and older sister were born in the Dominican Republic. I am a Turkey baby, meaning I was born on Thanksgiving.

**Research/Professional Interests and Goals:** I am interested in learning more about the subject of human metabolism and its impact on overall health. My goal is to begin osteopathic medical school within the next year or two, and I hope that the knowledge I gain through this program can be carried into practice with my future patients.

**Hobbies:** Baking, cooking, music

Olivia Colbert

**Education:** B.S Health Sciences, University of Central Florida

**Growing Up:** Port St. Lucie, Florida

**Professional Interests and Goals:** Once I have acquired my Master’s in Biomedical Sciences, I plan on attending Physician Assistant school, specializing in either cardiology or obstetrics.

**Hobbies:** Fitness, puzzles, bowling, and anything adrenaline filled

Jason Hendershot

**Education:** B.S. Biology, UCF, 2016

**Growing Up:** Carbondale, CO

**Research/Professional Interests and Goals:** My career interest areas include the gastrointestinal microbiome and how nutrition affects it as well as cardiovascular conditioning and rehabilitation. As an undergrad, I conducted research in this area in sea turtles. Ultimately, I would like to teach human biological/health sciences.

**Hobbies:** Reading and listening to audiobooks, hiking, and spending time at the beach, and just generally being outdoors, especially with friends. My happy/de-stress place is the gym.
Antje Kiesow
Education: B.S Environmental Engineering, University of Weihenstephan, Germany; MBA, UCF; Ph.D Industrial Engineering, UCF, 2010
Growing Up: Germany and Austria
Research/Professional Interests and Goals: I am interested in neurosciences, but also in metabolic and cardiovascular sciences. My main goal is to do research in a biomedical field.
Hobbies: Woodworking, water sports, martial arts

Luisa Moreno
Education: B.S Biomedical Sciences, 2018, UCF
Growing Up: I was born in Lima, Peru and came to the U.S at the age of 10. I live in Miami, Florida.
Research/Professional Interests and Goals: My goal is to become a pediatrician.
Hobbies: Dancing, cooking, reading.

MS Biomedical Sciences- Infectious Disease

Nannette Reyes Calderon
Education: B.S. Natural Sciences, UPR, 2017
Growing Up: San Juan, Puerto Rico
Research/Professional Interests and Goals: I completed an interdisciplinary research during my undergraduate career, where I was able to integrate and apply biology, chemistry and statistics, and gain knowledge on scientific research. I did shadowing and volunteering at a Pathology lab in Clermont, Florida, where I learned about infectious diseases. More recently I was a volunteer at Orlando Health. I am interested in studying memory T cell populations that mediate protection against viruses. I am also interested in the development of vaccines. Upon obtaining my M.S., I plan to continue my studies in the Biomedical or Medical fields and gain my doctorate degree.
Hobbies: Reading, swimming and theater acting.
Jordan Lass
Education: B.S. Biomedical Sciences, UCF, 2018
Growing Up: West Palm Beach, Florida
Research/Professional Interests and Goals: I am interested in focusing on the immune response towards various infectious diseases. I plan to obtain my M.S. and work towards a Ph.D. so I may work at the CDC or NIH and collect lab specimens in the field. I have also been considering going to PA school to work under an Infectious Disease MD or attend MD school to obtain an ID MD.
Hobbies: Training for triathlons, playing video games and board games, going to Disney

Austin Massolio
Education: B.S. Health Sciences, Biomedical Physics Minor, University of South Florida, 2018
Growing Up: Punta Gorda, Florida
Research/Professional Interests and Goals: As an undergraduate, I studied disease ecology with a focus on the priority effects within a living host. I am interested in continuing studying disease ecology but I would like to focus on diseases that are commonly found in patients such as MRSA or blood borne pathogens. After completing my master’s at UCF, I plan on attending medical school. I would like to pursue a career in emergency medicine or general surgery with a fellowship in wound care.
Hobbies: I enjoy an active life style and spending as much time as I can with my family and friends. I currently work as a Patient Care Technician at a teaching hospital (Northside Hospital) located in St. Pete, Florida.

Annsophia Mompoint
Education: B.S. Biology, UCF, 2018
Growing Up: Miramar, Florida
Research/Professional Interests and Goals: I aspire to become a physician. Upon obtaining my master’s degree, I plan on applying to medical school in pursuit of that goal.
Hobbies: Solo travel, attending concerts, and training for races.
Youyou Cheng
Education: B.A Law Management, East China University of Political Science and Law, 2014; B.S Biochemistry, UCF, 2019
Growing up: Shanghai, China
Research/Professional Interests and Goals: I am interested in cancer drug synthesis and new technology of doing cell imaging. I am planning to find a career at a hospital as a radiologist.
Hobbies: Dog walking, cooking, painting, travelling

Sean Moyer
Education: B.S. Health Sciences, Minor in Chemistry, UCF, 2018
Growing Up: Melbourne, Florida
Research/Professional Interests and Goals: After completing my master’s, I plan on pursuing medicine. I have taken an interest in cancer research and the upcoming capabilities and innovations in the field.
Hobbies: Microbiology TAing, basketball, and riding bikes.

Luis Silva Rodriguez
Education: B.S Biomedical Sciences, UCF, 2019
Growing Up: Cape Coral, Florida
Research/Professional Interests and Goals: My passion is Oncology. I shadowed a medical oncologist, Dr. Melgen, and ever since then I’ve grown to enjoy learning about cancer and possible treatments. I plan on attending the UCF Medical School at some point in my career in my effort to become a medical oncologist and help save lives.
Hobbies: Video games, fishing, camping, and cooking.
Charles St. James

Education: B.S. Biology, University of Central Florida, 2017
Growing Up: Ormond Beach, Florida
Research/Professional Interests and Goals: Upon completion of my bachelor’s degree, I worked as a tutor and science teacher in Volusia county. I’m currently investigating the role of Streptomyces Vitaminophilus’ Biosynthetic Gene Clusters in producing the antibiotic, Pyrrolomycin, under the guidance of Dr. Caranto. Upon completion of my Master’s in Biomedical Sciences, I plan on pursuing a doctorate degree in Molecular Biology with a focus on genetic engineering and biotechnology. Hobbies: Producing music, film, and hiking the outdoors of Central Florida.
Fernando Baena

**Education:** B.S. Health Sciences, Psychology Minor, UCF, 2018

**Growing Up:** Born in Pereira, Colombia. Raised in New York, Pereira, and Orlando.

**Research/Professional Interests and Goals:** Nearing the end of my bachelor’s degree I started gaining a big interest in nutrition with an emphasis in clinical nutrition. I hope to do research in that area after completing this program. I plan to apply to medical school with the goal of becoming a pediatrician and traveling to low income countries to volunteer.

**Hobbies:** Traveling, cooking/mixology, tennis, and my hanging out with my dog.

Sarah Geevarughese

**Education:** B.S. Neuroscience & Behavior, FAU, 2018

**Growing Up:** Miami, Florida

**Research/Professional Interests and Goals:** While pursuing my undergraduate degree, I completed research in a biomedical engineering lab focused on creating affordable devices to aid in Assisted Reproductive Technology. I subsequently joined a visual learning laboratory and focused my research on EEG data and its application to visual stimuli. I am interested in attending medical school in the future and pursuing a career as a PM&R physician with a focus in neuromuscular medicine.

**Hobbies:** Cooking, working out, and photography

Christopher Hawkins

**Education:** Biomedical Sciences B.S, UCF, 2017

**Growing Up:** Orlando, Florida

**Research/Professional Interests and Goals:** After attaining my master’s degree, I plan to attend medical school. Currently I have a strong interest in pursuing a career in emergency medicine.

**Hobbies:** Fishing, hiking, weightlifting, learning ASL
Christopher White

Education: B.S. Biomedical Sciences, UCF, 2017
Growing Up: Pembroke Pines, Florida
Research/Professional Interests and Goals: During my undergraduate career, I did research on the transfer of Lyme disease through Borrelia burgdorferi as well as the effect of Bradykinin on hemodialysis patients. Through this research as well as my upbringing, I realized that I wanted to pursue a career in the medical field. After completing my graduate studies, I hope to attend medical school and become a pediatric oncologist.

Hobbies: Music, lifting, biking, and videogames
ABSTRACTS

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45. The effect of E-cigarette nicotine vape on host-bacterial interactions in the oral cavity
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60. Novel lipid modification systems in Actinobacteria and their role in antibiotic resistance
61. The role of miR-146a in regulation of IRAK1/TRAF6/NF-κB signaling in normal human preadipocytes and endothelial cells.
62. Dynamic transcriptomic analysis reveals ripple effects of maternal binge alcohol consumption in the embryonic mouse heart: Implications for congenital heart defects
1. **Therapeutic Delivery of Interleukin 2 Drives Systemic as Well as Lung Innate Inflammatory Responses**

Mate Nagy, Kai K. McKinstry, Tara M. Strutt

Immunity and Pathogenesis Division, Burnett School of Biomedical Sciences, University of Central Florida, Orlando FL 32826

Interleukin 2 (IL-2) is a cytokine that is being investigated as a therapeutic to enhance immune responses, however, administration is known to have toxic side effects. IL-2 is a growth factor that plays a crucial role in the activation of immune responses. It is produced by activated CD4 T cells and acts in an autocrine manner to promote differentiation and proliferation. The receptor for IL-2 is comprised of an alpha, beta, and gamma subunit. Naïve T cells express the low affinity beta and gamma chains and upon activation the alpha chain is expressed that confers high affinity. As we have previously found a role for T cell-derived IL-2 in promoting inflammatory responses in the lung during respiratory virus infection, here we explore the outcome of systemic IL-2 administration on tissue and systemic inflammatory responses in otherwise unmanipulated animals. Our observations show that influx of innate cells such as natural killer cells and neutrophils measured by flow cytometry and production of inflammatory mediators assessed by multiplex analysis are increased both in the lungs and systemically following intraperitoneal administration of IL-2 or IL-2 complexes. Blockade of the IL-2 receptor with blocking antibody ameliorates the inflammatory responses detected following IL-2 administration. Our findings suggest that the lung is particularly sensitive to IL-2 and they may help explain some of the toxic side effects observed following its systemic administration.
2. Avirulins, a Novel Class of HIV-1 Reverse Transcriptase Inhibitors effective in the Female Reproductive Tract Mucosa

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While extensive research efforts have decreased human immunodeficiency virus (HIV) transmissions and mortalities, new challenges have arisen in the fight to eradicate HIV. Drug resistance to antiretroviral therapy threatens infected individuals, while the prevalence of heterosexual transmission creates an urgent need for therapies effective in the female reproductive tract (FRT) mucosa. We screened a library of 2095 small molecule compounds comprising a unique chemical space, purchased from Asinex Corporation, for antiviral activity against human immunodeficiency virus type 1 (HIV-1) strain BaL and identified several molecular representatives of a unique class of HIV-1 inhibitors, which we termed “Avirulins.” We determined that Avirulins were active against clinical isolates of HIV-1 from genetically variant subtypes, several of which have reduced sensitivity to other antivirals. Avirulins displayed specific dose-dependent inhibition of the HIV-1 drug target, reverse transcriptase (RT). Avirulins were effective against several nucleoside RT-inhibitor resistant strains of HIV-1, as well as one nonnucleoside RT-inhibitor resistant strain containing a 106A mutation, suggesting a noncompetitive mechanism of action. Drugs, which are damaging to the FRT, can increase the risk of HIV-1 transmission. We therefore explored the cytotoxicity of Avirulins against epithelial cells derived from the FRT and found no significant toxicity, even at the highest concentrations tested. Importantly, Avirulin antiviral activity was not diminished in human cervico–vaginal fluid, suggesting retained potency in the milieu of the FRT. Based on these promising results, Avirulins should be valuable chemical scaffolds for development into next-generation treatments and preventatives that target HIV-1.
3. **Prenatal influenza infection-associated low birthweight and short-term morphological developmental delays are prevented by LAIV vaccination**

Ali R. Satchmei and Tara M. Strutt

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Influenza A Virus (IAV) is a global health concern and certain groups are at higher risk for serious complications. One of these groups includes pregnant women, who are subject to disproportional morbidity and mortality when it comes to IAV infection. Many pregnant mothers, out of concern for their infants, avoid receiving the vaccine. IAV infection during pregnancy puts mothers at higher risk for gestational complications, including stillbirths and preterm delivery and unvaccinated mothers infected with influenza during pregnancy consistently give birth to infants with lower birth weights than their vaccinated counterparts. Although research and literature are abundant on the topic of influenza and pregnancy, there remains a gap when it comes to the effects of protective, IAV-specific T cell immunity generated before pregnancy on maternal health and fetal development. This type of immunity has the potential to mediate universal pandemic versus strain-specific protection. Here, we investigated how priming with a live attenuated influenza vaccine (LAIV) before pregnancy and subsequent heterosubtypic viral challenge during pregnancy impacts neonatal development in mice. Pup weight, crown-rump length, and skeletal development were measured over a three-week timespan postpartum. In contrast to the significantly lower weights of pups born to unprimed dams observed out to 21 days postpartum, morphological development, while initially hindered, did not significantly differ between pups born to unprimed and primed dams after the first week after birth. These observations support that IAV-specific T cell immunity generated prior to pregnancy has the potential to protect both maternal and fetal health.
4. Selenium vs. Sulfur: Investigating the Substrate Specificity of a Selenocysteine Lyase

Michael A. Johnstonea, Samantha Nelsona, Christine Van Groesbecka, and William T. Selfa

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Selenium is a vital micronutrient in many organisms. While traces are required for survival, excess amounts are toxic; thus, selenium can be regarded as a biological “double-edged sword”. Selenium is chemically similar to the essential element sulfur, but curiously, evolution has selected the former over the latter for a subset of oxidoreductases. Enzymes involved in sulfur metabolism are less discriminate in terms of preventing selenium incorporation; however, its specific incorporation into selenoproteins reveals a highly discriminate process that is not completely understood. In this work, we add knowledge to the mechanism for selenium-over-sulfur specificity in hopes of further understanding the controlled regulation of selenium trafficking and the prevention of its toxicity. We have identified SclA, a selenocysteine lyase in Enterococcus faecalis, and characterized its enzymatic activity and specificity for L-selenocysteine over L-cysteine. Human selenocysteine lyase contains a residue, D146, which plays a significant role in determining its specificity. A D146K mutation eliminated this trait, allowing non-specific L-cysteine degradation. Using computational biology, we identified an orthologous residue in SclA, H100, and generated mutant enzymes with site-directed mutagenesis. The proteins were overexpressed, purified, and biochemically characterized. All mutants exhibited varying levels of activity towards L-selenocysteine, hinting at a catalytic role for H100. Additionally, L-cysteine acted as a competitive inhibitor towards all enzymes with higher affinity than L-selenocysteine. Finally, SclA was observed to possess extremely poor cysteine desulfurase activity. Our findings offer key insight into the molecular mechanisms behind selenium-over-sulfur specificity and may further elucidate the role of selenocysteine lyases in vivo.
5. Integration of antifouling and nitric oxide releasing-polymer for enhanced biocompatibility of insulin cannula

Manjyot Kaur Chug§, Corbin Feit§, Elizabeth J. Brisbois§

§Department of Materials Science & Engineering, University of Central Florida, Orlando, FL USA

Subcutaneous placement of the insulin infusion cannula triggers rapid protein adsorption and activation of neutrophils and macrophages causing localized inflammation. Additionally, lack of skin disinfection prior to cannula placement can lead to the growth of bacteria on cannula resulting in bacterial infection. ~36% of insulin pump users have infection or inflammation at the infusion site and insulin infusion sets have a 65% failure rate after 7 days of implantation. These complications can lead to inadequate insulin delivery and infection, thereby requiring rotation of cannula site every 2-3 days. To overcome these challenges, we present a new generation of insulin infusion cannula that is developed with bioinspired polymers integrating antifouling slippery, liquid-infused porous surface (SLIPs) technology with active nitric oxide (NO) releasing polymer. Nitric oxide is an endogenous signaling molecule with potent anti-inflammatory and antibacterial properties. The cannulas were developed by impregnating the nitric oxide (NO) donor S-nitroso-acetylpenicillamine (SNAP) and silicone oil in commercial medical grade silicone rubber tubing, via a solvent swelling process. The NO release was characterized using chemiluminescence, leaching of SNAP was quantified using UV/Vis spectroscopy and the efficacy of the cannula for protein adsorption and antibacterial properties was evaluated using in vitro bioassays under physiological conditions. The NO-releasing cannulas release physiological levels of NO for > 7 days and exhibit > 90% reduction of pathogen (Staphylococcus aureus and epidermidis) compared to the uncoated controls. This NO-releasing polymer can enhance the biocompatibility of cannula ultimately improving the efficacy of insulin delivery with better care and quality of life.
6. The Significance of the Hormonal Milieu on the Development and Progression of Non-Alcoholic Fatty Liver Disease

Spencer Lessans\textsuperscript{a}, Michael Rohr\textsuperscript{a}, Deborah A. Altomare\textsuperscript{a}

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Non-Alcoholic Fatty Liver Disease (NAFLD) is the most common liver disease in the Western world and varies in severity from a simple steatosis or “benign” fatty liver to advanced liver fibrosis. It is unclear why some patients remain asymptomatic while others progress until a liver transplant is ultimately required. Development and progression of NAFLD is at least in part dependent on sex, since NAFLD has been found to be more common and develops earlier in men than in women. The incidence of NAFLD in women peaks in their late 50s to early 60s following menopause alongside lower estrogen levels, which suggests that the development of NAFLD is affected by hormone levels. The hypothesis is that estrogen should decrease the severity of NAFLD and the risk of progression to fibrosis. We tested HepG2 hepatocytes exposed to a saturated fatty acid (palmitate), and then either exposed to either estrogen or tamoxifen. HepG2 cells were assessed for accumulation of fat droplets and cytokine expression via RT-PCR and Western Blot. Hepatic stellate cells were then incubated in the conditional medium from the hepatocytes and assessed for induction of myofibroblastic activation and collagen expression via RT-PCR. We expect hepatocytes treated with tamoxifen will have increased intracellular fat droplets and inflammation than hepatocytes treated with estrogen. Stellate cells in the tamoxifen conditional medium are expected to have increased activation compared to those in the estrogen conditional medium. This research will help clarify gender-specific factors that place patients at increased risk for NAFLD for targeted screening.
We discuss applications of SAS®9.4 software to assess sex disparities in three quantitative measures: patient’s age, age of smoking initiation, and age at IBD diagnosis among smokers with Inflammatory Bowel Disease (IBD). The project included the following steps: (1) importing data in SAS®9.4, (2) preparing codes to refine the sample and perform statistical analysis, (3) interpreting outputs and (4) drawing inferences. We used data (n=501) of patients with Crohn’s disease and/or ulcerative colitis who self-identified as smokers in the IBD Partners’ online survey in the period from 2011 to 2014. For each measure we checked validity of the assumption of equal variances using Folded F-test. If the data supported validity of the assumption, we used regular t-test with pooled variance estimator. If the data did not support validity of the assumption, we used Satterthwaite t-test (to adjust for unequal variances). The significance level was fixed at 5% for each measure. The analyses indicated that assumption of equal variances was supported only for the patient’s age and age at IBD diagnosis. On average, men and women differed significantly in terms of the age (Pooled t=2.72, df=499, p=0.02) but did not differ in terms of age of smoking initiation and age at IBD diagnosis. Therefore, the study pointed to existing sex disparities in the mean patient’s age among smokers with IBD.
Rheumatoid arthritis (RA) is an autoimmune disease that results in inflamed joints and osteoporosis. Previous studies have shown that *Mycobacterium avium* subspecies *paratuberculosis* (MAP) has a role in the downregulation of active osteocalcin in Crohn’s disease (CD). Since RA and CD have a similar pathology, we hypothesize that active inflammation following MAP infection will result in downregulation of active osteocalcin in RA patients. In this study, a total of 82 individuals (48 RA and 34 healthy) were examined for MAP status and for active osteocalcin. Out of the 48 RA patients, 18 were MAP positive compared to the 1 out of 34 healthy (P-values < 0.05). Overall, active osteocalcin levels between RA (2.70 ± 6.06 ng/mL) were significantly lower than healthy controls (5.84 ± 8.23 ng/mL, P-values < 0.05). MAP positive RA patients’ active osteocalcin levels were lower (0.599 ± 1.26 ng/mL) than MAP negative RA patients (3.859 ± 7.27 ng/mL). The active osteocalcin level in healthy MAP positive controls were lower (1.647 ng/mL) than healthy MAP negative controls (5.966 ± 8.31 ng/mL). The level of active osteocalcin in all MAP positive patients was significantly lower (0.657 ± 1.27 ng/mL) than all MAP negative patients (4.94 ± 7.83 ng/mL, P-values < 0.05). The correlation between presence of MAP in RA individuals and downregulation of active osteocalcin supports a role of MAP as a pathogenesis element RA.
Exosomes Derived from Embryonic Stem Cells Inhibit Doxorubicin-Induced Pyroptosis in Cardiac Cells

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Doxorubicin (Dox) is a potent chemotherapeutic drug used to treat different tumors. Further administration of Dox is restricted due to cardiotoxicity induction, which is mediated through oxidative stress and inflammation. However, it remains unknown whether Dox induces an inflammasome-mediated cell death, known as “pyroptosis” in the heart. Pyroptosis happens via activation of TLR-4 receptor and formation of NLRP3 inflammasome in response to damage-associated molecular patterns (DAMPs), which stimulates caspase-1 and induces secretion of pro-inflammatory cytokines, IL-1β/IL-18, from the dead cells. This study is developed to understand whether Dox induces pyroptosis in cardiac cells and to elucidate the protective effects of embryonic stem cell-derived exosomes (ES-Exos) on inhibition of pyroptosis. For this purpose, we developed a cell culture model using H9c2 cardiomyoblasts. H9c2 cells were exposed to Dox (2 μM for 24 hrs) to generate pyroptosis and then treated with exosomes (10 μg for additional 24 hrs). Cells were divided into 4 groups: Control, Dox, Dox+ES-Exos, and Dox+MEF-Exos (as negative control). According to western blot analysis, immunostaining and ELISA data, Dox significantly increased (p<0.05) expression of inflammasome markers, TLR-4 and NLRP3, pyroptotic markers, caspase-1, IL1-β, Caspase-11, and gasdermin-D, as well as pro-inflammatory cytokines, TNF-α and IL-6, in H9c2 cells. Importantly, ES-Exos treatment significantly reduced (p<0.05) pyroptosis and inflammation in H9c2 cells exposed to Dox. However, MEF-Exos treatment did not make significant changes vs Dox group (non-significant vs Dox). The significance of this study is a novel mechanistic insight on the pathophysiological role of NLRP3 inflammasome-mediated pyroptosis in Dox-induced cardiotoxicity.
Influenza A virus (IAV) remains a global health concern for susceptible individuals, including pregnant women. Physiological changes during pregnancy are known to alter the immune system, resulting in bias towards humoral responses at the cost of cell-mediated immunity, which is essential for anti-viral responses. While much is known about alterations to primary immunity, how memory or secondary responses are impacted during pregnancy remains unexplored. We examined the responsive capacity of IAV-specific memory CD4 T cells in gravid and non-gravid hosts during recall infection. Timed-pregnant Balb/c mice and non-gravid female controls were adoptive transfer recipients of in vitro-generated IAV-specific memory CD4 T cells challenged with IAV. Naïve IAV-specific CD4 transgenic cells were isolated, polarized and rested in vitro to generate donor Th1 CD4+ memory cells. On day 7 post infection, the number of donor memory cells, surface expression of CD127 (IL-7 receptor), and production of cell-mediated response-associated cytokines IFN-γ, TNF, and IL-2 in the spleen, draining lymph nodes, and lung were determined in recipient hosts by flow cytometry. Fetal outcomes were also monitored. Donor memory CD4 T cell recovery in all organs was similar between gravid and non-gravid female mice on day 7 of sub-lethal IAV infection, as well as functional capacity in terms of multi-cytokine production, associated with cell-mediated immunity. However, CD127 expression, essential for memory CD4 T cell survival, was significantly increased in gravid females. Preliminary observations suggest that the altered physiological environment of pregnancy has minimal impact on responsiveness of in vitro-generated Th1 memory CD4 T cells.
Ghrelin has long been implicated in energy homeostasis and is thought to regulate caloric consumption. Secreted by gastric emptying, circulating ghrelin has been shown to work in diverse physiological pathways including caloric intake, glucose regulation, and lipid storage. Previous data have shown that ghrelin and its receptor are located in the taste system. Mice lacking ghrelin have a decreased caloric intake and altered lipid preferences (Cai et al., PLoS One 8(10), 2013). To date, limited research has explored the role of ghrelin in fat taste. Previous work in our lab demonstrated high levels of co-localization between growth-hormone secretagogue receptor (GHSR), a ghrelin receptor, and markers for Type II cells, the main cells responsible for fat taste transduction. The current study is aimed at evaluating the functional role of ghrelin in fat taste using ghrelin-null mice and WT mice. To date, we found that ghrelin KO mice showed a faster extinction rate of linoleic acid aversion in a conditioned taste aversion assay. Moreover, calcium imaging assays in taste cells from ghrelin-null mice have diminished responses to fatty acids (FA) compared to WT mice. Additionally, WT taste cells showed enhanced responses to linoleic acid when perfused with a ghrelin receptor agonist concurrently. Given that obesity is fueled, in part, by overconsumption in response to altered fat taste, our data suggest changes are potentially guided by the caloric intake associated with increased circulating ghrelin levels.

Supported by NIH DC013194 and DC013318 (tag).
12. Biochemical and Biophysical Analysis of the Critical *Borrelia burgdorferi* Virulence Protein BBK13

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Division of Immunity and Pathogenesis, Burnette School of Biomedical Sciences, University of Central Florida College of Medicine, Orlando, Florida, 32827, USA.

The proteome of the Lyme disease pathogen *Borrelia burgdorferi* contains a large subset of uncharacterized proteins of unknown function. In our previous work, we demonstrated that one such protein BBK13, plays a crucial role in infectivity by contributing to spirochete expansion within the skin. In this study, we performed a biochemical and biophysical characterization of BBK13 as a first step to understanding the role of this critical protein. Using a polyclonal antibody generated against recombinant BBK13, we localized the protein to the *B. burgdorferi* membrane but determined it is not surface exposed. In addition, we demonstrated that BBK13 is immunogenic in both *B. burgdorferi* infected mice and human Lyme disease patients. To better understand the structure of BBK13, size exclusion chromatography was performed with purified recombinant BBK13. The results suggested that the 25 kiloDalton (kDa) recombinant protein formed a large oligomeric structure of greater than 600 kDa. Further, 2-dimensional Blue Native Page demonstrated that endogenous BBK13 formed complexes within the spirochete membrane. Endogenous BBK13 was detected in complexes ranging in molecular mass from 1,000 kDa to 480 kDa with no monomeric BBK13 observed. In addition, 2-dimensional isoelectric focusing studies suggested the possibility that BBK13 may undergo post-translational modification. Together, these data indicate that BBK13 is an immunogenic protein that forms large complexes within the *B. burgdorferi* membrane. This biochemical and biophysical analysis furthers our understanding of the critical *Borrelia burgdorferi* virulence protein BBK13 and lays the foundation for future studies into the function of BBK13.
13. Novel carbapenem derivatives with enhanced antimicrobial activity against
*M. tuberculosis* and *M. abscessus.*

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Carbapenems are promising antibiotics with broad-spectrum activity against Gram-positive and Gram-negative pathogens and mycobacteria. The underexplored chemistry of these compounds, with substitutions only at C2 position of the carbapenem scaffold, leaves ample room for optimization of this drug class through the synthesis of novel analog series. In the present study, we targeted the carbapenem backbone for unusual modifications at non-C2 positions. We designed and synthesized non-C2 carbapenems by employing a stereospecific synthesis approach and evaluated for antimycobacterial activity against two important human pathogens, *Mycobacterium tuberculosis* (Mtb) and *M. abscessus* (Mab). *In vitro* activity evaluation against both pathogens revealed several compounds with MIC better than the commercially available carbapenem, meropenem. Addition of β-lactamase inhibitors lowered MICs by 1-2 fold in case of *M. tuberculosis* but had minimal effect against *M. abscessus.* The two most potent non-C2 carbapenems against both pathogens were further characterized for bactericidal activity and inhibition of clinical strains. Time-kill kinetics assay showed better killing (2-4 log decrease) of *M. tuberculosis* and *M. abscessus* with lower concentrations (1X and 2X MIC) of compound 1 as compared to meropenem. Activity against clinical strains revealed a surprisingly wide range of susceptibilities to meropenem and compound 2. However, compound 1, which displayed superior activity in all other assays (MIC<sub>Mtb</sub>=0.7 µM, MIC<sub>Mab</sub>=2.9 µM), was equally active against all strains. This series of compounds illustrates the potential of novel non-C2 modified carbapenems as promising therapeutic candidates for treatment of *M. tuberculosis* and *M. abscessus* infections.
14. Determining differential effects of Interleukin-2 on Immune cells in lymphoid organs and the Gastrointestinal Tract

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Interleukin-2 (IL-2) is a pleiotropic cytokine demonstrated to be effective in treating cancer. However, clinical use of IL-2 can be associated with severe side effects including gastrointestinal toxicity (GT). Similar GT symptoms are observed in inflammatory diseases such as Crohn’s disease (CD). Interestingly mounting evidence indicates a role for IL-2 in CD, but the underlying mechanisms are unknown. Indeed, studies on the in-vivo activities of IL-2 have mostly focused on secondary lymphoid organs and immune cells associated with them. Very few studies have addressed how IL-2 signals impact populations of immune cells in the gut. Here, we aim to identify and compare the effects of systemic IL-2 administration on six major leukocyte population and their subsets in mice using multicolor flow cytometry. While we confirmed previously observed changes in specific immune cell populations in the spleen, very few changes were seen in the gut and gut associated lymphoid tissues. Unexpectedly, a sharp decline was seen in B cells, most notably in Peyer’s Patches, in mice treated with IL-2. Our data furthermore indicates that B cells in IL-2 treated mice undergo enhanced apoptosis in Peyer’s Patches. Our future goals are to determine if IL-2 acts directly on B cells and if it impacts certain B cell subsets in Peyer’s Patches, as some studies suggest that changes in B cells may contribute to development of CD. Thus, this study may aid in defining ways in which IL-2 can contribute to disease etiology, and lead to novel treatments for CD.
Previous results have shown that infection with the cytoplasmic-replicating parainfluenza virus 5 mutant P/V-CPI- sensitizes cells to DNA damaging agents, resulting in the enhanced killing of airway cancer cells. Here, we have tested the hypothesis that histone deacetylase (HDAC) inhibitors can also act with P/V-CPI- infection to enhance cancer cell killing. Using human small cell lung cancer and laryngeal cancer cell lines, 10 HDAC inhibitors were tested for their effect on viability of P/V-CPI-infected cells. HDAC inhibitors such as scriptaid enhanced caspase-3/7, -8 and -9 activity induced by P/V-CPI- and overall cell toxicity. Scriptaid-mediated enhanced killing was eliminated in lung cancer cells that were engineered to express a protein which sequesters double stranded RNA. Scriptaid also enhanced cancer cell killing by two other negative strand RNA viruses – the La Crosse virus and vesicular stomatitis virus. Scriptaid treatment enhanced the spread of the P/V-CPI- virus through a population of cancer cells, and suppressed interferon-beta induction through blocking phosphorylation and nuclear translocation of Interferon Regulatory Factor 3 (IRF-3). Taken together, these data support a role for combinations of a cytoplasmic-replicating RNA virus such as the P/V-CPI- mutant along with chemotherapeutic agents.
16. The Role of Notch-1, IL-6 and miR-146a Polymorphisms in Inflammation and MAP Infection in Crohn’s Disease

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Treatment of Crohn’s disease (CD) mostly depends on TNF-\(\alpha\) inhibitors where more than 50\% of the patients seem not to respond very well. Earlier studies in our lab showed that TNF-\(\alpha\) inhibitors induce survival of \textit{Mycobacterium avium} subspecies \textit{paratuberculosis} (MAP) and inflammatory response. Interestingly, our previous results showed that the pro-inflammatory cytokine IL-6 has been found to be closely correlated with MAP viability in infected macrophages. Here, we are investigating the in vitro effect of TNF-\(\alpha\) inhibitors on Notch-1 and downstream influence on IL-6, apoptosis and survival of MAP. Similarly, we are evaluating expression of Notch-1, and IL-6 in association with MAP infection and miR-146a mutations in clinical samples. To date, 42 clinical samples (20 CD and 22 healthy controls) were analyzed for Single nucleotide polymorphism (SNP) in miR-146a, presence of MAP, gene expression of Notch-1, IL-6 and more. Preliminary data showed that MAP is present in 62.5\% CD compared to 9\% controls. Notch-1 was upregulated in CD clinical samples compared to healthy controls. Similarly, IL-6 was upregulated in CD samples compared to healthy controls. The highest IL 6 gene expression was in CD samples which positive for MAP. MiR-146a SNP data is pending analysis. Overall, CD patients on anti-TNF-\(\alpha\) treatment appear to suffer from upregulation in Notch-1, which in turns seems to upregulate IL-6 and favors MAP infection.
**17. Cryopreserved Influenza A Virus (IAV)-specific memory CD4+ T cells mediate protection against lethal infection and form secondary lung-resident memory**

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Influenza is a global health concern that causes worldwide morbidity, mortality, and economic loss annually. Currently, preventative and therapeutic options are lacking to prevent and treat serious infection; the efficacy of the best available seasonal flu vaccine ranges from 10-60% and antiviral drug use restricted to the first 48 hours of infection. An ideal anti-viral cell therapy should provide rapid and robust immunity against multiple strains of the influenza virus, and ideally form durable immunologic memory at the primary site of infection, the lung. We investigate here whether adoptive cell therapy with cryopreserved *in vitro*-generated Influenza A virus (IAV)-specific memory CD4+ T cells, which are capable of mediating universal protection against IAV prior to cryopreservation, possesses prophylactic and therapeutic potential. In comparison to IAV infected hosts that did not receive memory cells, recipients of cryopreserved IAV-specific memory CD4+ T cells presented with decreased morbidity and expedited recovery from infection. Additionally, we observed that the cryopreserved memory CD4+ T cells form multi-cytokine producing, secondary memory cells. In contrast to prior observations with non-cryopreserved memory CD4+ T cells that form secondary memory in secondary lymphoid tissues as well as lung, cryopreserved memory CD4+ T cells were found to preferentially form only secondary lung memory cells. These observations, which have been validated in different strains of mice and with different CD4+ T-cell receptor transgenic donor cells, show the promise for cryopreserved, *in vitro*-generated IAV-specific memory CD4+ T cells as a preventative and therapeutic option to prevent serious IAV infection.
18. Functional characterization of miR-299-3p that target Androgen Receptor in Prostate Cancer

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Prostate Cancer (PCa) is the second leading cause of cancer-related deaths in the US. African Americans (AA) are particularly susceptible for PCa compared to Caucasians (CA) because of their predisposition to have a higher expression of androgen receptor (AR). Aberrant expression of microRNAs (miRNAs) is commonly noted in many cancers including PCa and can be a significant contributor of cancer progression. Genome-wide miRNA profiling studies in our lab identified a significant downregulation of miR-299-3p in PCa tissues compared to uninvolved areas, particularly in tumors from AA patients compared to CA patients. Here, we show that miR-299-3p directly targets AR and its loss of expression plays a role in PCa progression. Restored expression of miR-299-3p showed decreased AR expression and activity along with cell cycle arrest, reduced cell proliferation, cell migration, and expression of a mesenchymal marker. These observations suggest that miR-299-3p exerts a tumor suppressor role in PCa. We also noted differential promoter methylation of mir-299-3p gene in PCa cells of AA origin compared to CA origin. Treatment with 5-Aza-2-deoxycytidine reversed the expression of miR-299-3p in PCa cells. Furthermore, PCa cells of AA origin also showed increased DNMT activity compared to CA origin. These results suggest that epigenetic regulation is a causal factor for loss of expression of miR-299-3p, which is possibly responsible for aberrant expression of AR and development of aggressive PCa in racially disparate group of patients. This study will provide insight on the function of miR-299-3p in PCa progression and help developing a diagnostic marker for PCa.
19. Functional characterization of a novel long non-coding RNA PAINT as a promoter of aggressive prostate cancer

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Emerging evidences show that long non-coding RNAs (lncRNA) are frequently dysregulated in cancers including prostate cancer. It has been speculated that a vast population of lncRNAs, mostly with unexplored genetic information, may contain drivers of cancer progression. RNA-seq analysis conducted in our laboratory identified reduced expression of a long non-coding RNA LINC00888; ENSG0000240024 (PAINT) and up regulation of several tumor suppressor genes as co-expressers in a transformed subline of an aggressive prostate cancer cell line that exhibit reduced tumorigenicity and migration. TCGA and GEO profile analysis revealed an upregulation of PAINT in prostate cancer cases with higher Gleason Scores and in metastatic prostate cancer cell lines. In this study, we show the mechanistic function of PAINT in promoting tumor progression in prostate cancer cells. Knockdown of PAINT showed decreased cell proliferation, reduced S-phase progression and induction of apoptosis. Down regulation of PAINT showed decreased cell migration and increased expression of the epithelial marker, E-Cadherin. Ectopic expression of PAINT reversed the effects observed upon PAINT down regulation. Increased cell proliferation, expression of proliferation makers and cell migration were noted in prostate cancer cells expressing PAINT. Additionally, larger colony formation and higher expression of mesenchymal markers were observed in these cells. These observations indicate tumor promoting function of PAINT in prostate cancer cells. Furthermore, inhibition of PAINT expression showed an increased sensitivity of metastatic prostate cancer cells to the chemotherapeutic agent docetaxel. Taken together, our study establishes an oncogenic function of PAINT and its potential as a therapeutic target for prostate cancer.
20. Characterization of Sodium Transport in Mouse Gustatory Epithelium using an Ussing Chamber

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Designed by Hans Ussing to study frog skin, the Ussing chamber is a useful tool for studying electrolyte permeation of transport epithelia [Clarke 2009, Li et al. 2004]. Sodium transport by lingual epithelium has been studied in other species utilizing this technique [Simon et al. 1993, Gilbertson and Zhang 1998]. In this study, the Ussing chamber was used to examine the plasticity of salt taste by studying sodium transport in the mouse lingual epithelium. The measure of short-circuit current (Isc) is an indicator of net ion transport occurring across an epithelium. Sodium chloride (NaCl) solutions were applied to the apical (mucosal) side of the lingual epithelium. The mounted tissue’s resistance was monitored by applying a hyperpolarizing 20 mV pulse. High (500 mM) and low (50 mM) NaCl concentrations were used to explore the transport of Na⁺ and Cl⁻ ions across the lingual epithelium. Two major pathways contribute to sodium transport; paracellular and transcellular inhibitors, lanthanum chloride (LaCl₃) and amiloride, were used to determine contributions from each pathway respectively. Fungiform papillae showed an amiloride sensitivity for low mM NaCl, but not high. Circumvallate appeared to be unaffected by amiloride for low or high NaCl concentrations. Application of LaCl₃ showed little effect on low Na⁺ concentration currents for fungiform papillae. Interestingly, circumvallate papilla had an increased current for low concentrations following application of LaCl₃. The data suggests the papilla regions favor different transport pathways at low NaCl concentrations and the Isc measured is the net result of the mobilization of several different ions.
Autism spectrum disorder (ASD) is a childhood onset, neurodevelopmental condition characterized by impaired communication and repetitive behaviors. Evidence suggests ASD is a glial cells disorder; however, what causes this gliosis remains unknown. We previously showed that Propionic acid (PPA), a short chain fatty acid, is able to shift human neural stem cell (hNSC) differentiation towards glial phenotype in vitro; however, this PPA-induced mechanism remains unclear. In this follow up study, we propose PPA induced-gliosis is mediated through glial specific GPR41 receptor, which upon binding PPA reduces Phosphatase and tensin homolog (PTEN) expression; therefore, allowing for pro-survival p-Akt to remain active and induce glia over-proliferation. To address this hypothesis, hNSCs were exposed to ascending concentrations of either PPA or Butyric acid. Results demonstrated significantly (p<0.05) increased GFAP expression upon PPA (2mM) treatment, indicative of increased number of glial cells. Meanwhile, Tubulin-β3 (Neurons) significantly decreased. Moreover, inflammatory cytokines; TNF-α, IL-6, and IL-10 increased with PPA, indicating gliosis. Additionally, GPR41 and downstream p-Akt levels increased with 1mM and 2mM of PPA, respectively; whereas PTEN expression decreased (p<0.05) with 0.5mM of PPA. This data is a proof of concept that PPA may induce gliosis through modulation of glial specific GPR41/PTEN pathway, potentially mirroring the autistic brain.
22. Actin Filament Mechanics and Structure in Crowded Cellular Environments

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Actin filament assembly and mechanics play critical roles in various cellular functions including structural support, cell movement, division, and intracellular transport. Intracellular environments are crowded with numerous types of solutes such as ions, compounds, and macromolecules that reduce accessible volume fractions for protein-protein interactions. Reduction in cellular volume gives rise to excluded volume effects along with depletion forces, affecting protein assembly and stability. Although the impacts of molecular crowding on actin polymerization have been shown, how crowded environments affect actin filament conformations, dynamics, and mechanical properties has yet to be established. In this study, we investigate the effects of solution crowding on the modulations of filament mechanics and structure both in vitro and in silico. We perform direct visualization of filaments in the presence of inert crowding agents using fluorescence microscopy imaging, allowing for the quantification of filament thermal bending dynamics and mechanics. Biophysical analysis indicates macromolecular crowding alters filament thermal bending, enhances filament’s effective stiffness, and reduces average filament lengths. Using all-atom molecular dynamics simulations, we demonstrate that macromolecular crowding alters filament conformations by inducing over-twisting of filament structure thereby promoting compaction, which is directly coupled to filament mechanics. Combined experimental and computational results suggest that macromolecular crowding modulates the mechanical and structural properties of filaments, possibly through interplay between excluded volume effects and non-specific interactions. Our study provides a strong foundation for molecular mechanisms by which macromolecular crowding influences actin cytoskeleton mechanics and structure in cellular environments.
Prostate cancer (PCa) is a leading cause of death for men worldwide. Most PCa patients die from metastasis and bone is the most common metastatic site. Three dimensional (3D) porous chitosan-alginate (CA) scaffolds were developed for bone tissue engineering and demonstrated for culture of cancer cells and enrichment of cancer stem cells. However, only a single scaffold composition was studied. Three compositions of 3D porous CA scaffolds (2, 4, and 6 wt%) were used to investigate the effect of scaffold stiffness on PCa cell response with PC-3, C4-2B, and 22Rv1 cell lines. The PC-3 cells formed cell clusters while the C4-2B and 22Rv1 cells formed multicellular spheroids. The three cell lines demonstrated stiffness independent cell growth and expressed phenotypic PCa biomarkers. The osteoblastic PCa lines C4-2B and 22Rv1 mineralized in basal media, while the osteolytic PC-3 line did not, demonstrating that CA scaffold cultures revealed differences in PCa phenotypes. The CA scaffolds are a 3D culture platform that supports PCa growth and phenotypic expression with adjustable scaffold stiffness to mimic stages of metastatic progression. Further investigation of the scaffolds for co-culture of PCa cells with fibroblasts and primary PCa cell culture should be conducted to develop a platform for screening chemotherapies.
Tobacco has a bivalent effect on the two subsets of Inflammatory Bowel Diseases (IBD); Crohn’s Disease (CD) and Ulcerative Colitis (UC). It was shown to flare inflammation in CD patients, while lessen the symptoms in UC. Interestingly, nicotine, the main tobacco compound, is believed to be protective in UC while cause inflammatory outburst in CD; however, how nicotine may play this dual role remains elusive. Unlike UC, CD has been increasingly associated with *Mycobacterium avium* subspecies *paratuberculosis* (MAP). Interestingly, we previously demonstrated that nicotine was bactericidal on MAP. Also, nicotine decreases apoptosis of *Mycobacterium tuberculosis* (MTB) infected macrophages. Therefore, we hypothesized that in MAP-infected macrophages, nicotine impairs anti-MAP defense by decreasing macrophage apoptosis thereof increasing mycobacterial infection burden. To address this hypothesis, we infected THP-1 macrophages with MAP for 24h followed by nicotine treatment at different concentrations (1, 2, 4, and/or 10ug/ml). Next, we evaluated the viability of MAP within macrophages, macrophage apoptosis and inflammatory profile. Results showed that nicotine at 4ug/ml significantly (p<0.05) favored anti-inflammatory profile with increased expression of CD-206 (M2) marker, and IL-10 cytokine in MAP-free macrophages, similar to UC. However, combined MAP and nicotine dramatically shifted macrophage differentiation into a pro-inflammatory M1 profile, with increased iNOS (M1), IL-6, and TNF-α expression. The pro-inflammatory effect of nicotine on MAP-infected monocytes was several folds higher than MAP alone. Additionally, ongoing data seems to indicate that nicotine may decrease macrophage apoptosis while increasing MAP viability which, if confirmed, will further explain the flares seen in CD smokers.
25. Optimal heterosubtypic immunity against Influenza A Virus requires the cytokine IL-21

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The aim of current influenza A virus (IAV) vaccines is to induce neutralizing antibodies against surface proteins of those IAV strains that are predicted to circulate during the ‘flu’ season. This type of protection, known as ‘homotypic’ immunity, fails to protect if the vaccine does not match circulating IAV strains, such as in the case when pandemic IAV strains emerge. However, it is well established that T cells can also protect against IAV and, unlike antibodies, T cells can recognize viral proteins that are shared across diverse IAV subtypes. This form of protection is known as ‘heterosubtypic’ immunity. The cytokine interleukin 21 (IL-21) contributes to immune responses by stimulating maximal antibody responses, and also impacts T cell function and their survival in certain settings. Here, we examine the role of IL-21 in murine models of homotypic and heterosubtypic immunity against IAV. Wild type (WT) or IL-21 receptor knock-out (IL-21r⁻/⁻) mice were primed with a sub-lethal dose of the mouse-adapted IAV strain A/PR8 (H1N1) and challenged 28 days later with a lethal dose of A/PR8, or of the heterosubtypic IAV strain A/Philippines (H3N2). Surprisingly, although IL-21 is best known in supporting the generation of maximal antibody responses, IL-21r⁻/⁻ mice showed no defects in homotypic immunity. Unexpectedly, primed IL-21r⁻/⁻ mice did not survive heterosubtypic IAV challenge. These studies indicate that IL-21 may contribute more to mechanisms associated with heterosubtypic rather than homotypic immunity. Understanding how to best stimulate T cell-dependent heterosubtypic immunity is a promising approach to improve IAV vaccines.
Tumor Necrosis Factor alpha antagonists (anti-TNFα) have been widely used for Crohn’s disease (CD). Although they may control CD symptoms initially, treatment response varies among patients, which seems to depend on single nucleotide polymorphisms (SNPs) in TNFα receptors superfamily 1A and 1B (TNFRSF1A/B). Most importantly, *M. tuberculosis* infection has been strongly associated with these medications, but no studies have elucidated the effects of anti-TNFα on CD associated with MAP (*Mycobacterium avium* subspecies *paratuberculosis*; a possible causative agent of CD). Here, we are investigating the effects of recombinant inflammatory cytokines and anti-TNFα therapeutics on macrophages infected with MAP isolated from CD patient. We also tested the prevalence of MAP and the significance of nine SNPs in *TNFα*, *TNFRSF1A* and *TNFRSF1B* from the blood of 54 CD and 50 healthy subjects. Overall, 31/54 CD patients were infected with MAP compared to only 4/50 controls [OR = 15.5, 95% CI: 4.88-49.22, *P*<0.05]. Both PEGylated and non-PEGylated forms of anti-TNFα increased MAP viability by nearly 1.5 logs, while rTNFα reduced MAP survival in infected macrophages by 2.63 logs. Gene expression of *TNFα*, *IL-6*, and *IL-12* was between 1.5 to 3 folds higher following MAP or *M. tuberculosis* infection compared to other bacterial strains (*P*<0.05). Additionally, Four SNPs (*TNFα*:rs1800629, *TNFRSF1A*:rs767455, *TNFRSF1B*:rs1061624 and *TNFRSF1B*:rs3397) were distributed significantly among CD patients. Both *TNFRSF1A*:rs767455 and *TNFRSF1B*:rs3397 downregulated their corresponding gene expression and induced susceptibility to MAP infection. The study provides data about the safety of using anti-TNFα in CD, and predictions toward treatment response based on patient’s pharmacogenomics.
27. A novel sRNA important for infectivity of the Lyme disease pathogen, *Borrelia burgdorferi*

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Lyme disease, caused by the bacteria *Borrelia burgdorferi*, is the leading vector borne disease in the US. The enzootic lifecycle of the bacteria requires that the spirochete cycles between the tick vector and vertebrate hosts. In doing so, *B. burgdorferi* is required to sense and respond to its environment to survive, yet there is a gap in understanding of the mechanisms that *B. burgdorferi* uses to do this. We and others have recently identified putative non-coding RNA transcripts (sRNAs), throughout the *B. burgdorferi* genome, whose functions in the biology of the pathogen are unknown. However, in other bacterial species, sRNAs have been demonstrated to play important regulatory roles involving stress responses and virulence. SR0819 is a putative sRNA located on virulence-associated linear plasmid 36 (lp36). Moreover, our *B. burgdorferi* in vivo expression technology (BbIVET) screen identified an infection active DNA sequence, *Bbive166*, that maps directly upstream of SR0819, suggesting that *Bbive166* may function as the promoter for SR0819. Using a luciferase reporter system and in vivo live imaging to analyze *Bbive166* promoter activity, we found that *Bbive166* is specifically active during mouse infection. A ΔSR0819 *B. burgdorferi* mutant clone demonstrated no defect in infection of mice. In contrast, *B. burgdorferi* engineered to overexpress SR0819 demonstrated a significant decrease in bacterial load in the blood of mice 6 days post inoculation, suggesting a possible role for this novel transcript in *B. burgdorferi* mouse infectivity. Future studies will combine molecular genetics, biochemistry and tick-mouse infection models to elucidate the function of SR0819 in the biology of *B. burgdorferi*. 
Psychiatric disorders are among the most prevalent and costly conditions in the United States. Despite this health care burden, current pharmaceutical treatments are not consistently effective and target symptoms rather than the biological root\(^1\). In recent years, strong connections have been made between psychiatric disorders and a decrease in adult neurogenesis. Neurogenesis describes the development of new, functional neurons from neural stem cells\(^2\). While neurogenesis was previously thought to halt after embryonic development, there is now evidence to support the persistence of neurogenesis in two regions of the adult human brain: the sub-granular zone (SGZ) and sub-ventricular zone (SVZ)\(^3\). Interruptions in adult neurogenesis are associated with psychiatric disorders, and regulation of neurogenesis is associated with improvements in neural stem cell proliferation, differentiation, and positive cognitive and behavioral effects\(^4\). There is potential for the treatment of psychiatric disorders by utilizing neurogenesis as a target.

Actin is an essential cytoskeletal protein, controlling cell motility, morphology, and wound healing with the help of actin binding proteins such as gelsolin. Gelsolin is a Ca\(^{2+}\)-regulated actin binding protein that severs and caps filaments. The majority of \textit{in vitro} studies of gelsolin and actin have been performed in dilute buffer conditions, which do not properly model the intracellular environment that these proteins are found in. The intracellular space is packed with carbohydrates and macromolecules that crowd the space, causing an excluded volume effect. We hypothesize that gelsolin and actin filaments present in a crowded environment will lead to greater gelsolin severing activity due to the excluded volume effect. To test this hypothesis, we have visualized actin filaments after incubation with gelsolin in crowded solutions. Polyethylene glycol and sucrose served as inert crowders that emulate intracellular crowding. The resulting images were analyzed, and average actin filament length as well as filament length distributions were determined in order to characterize the effect crowding has on gelsolin-mediated actin filament severing. Based on our preliminary results it appears that macromolecular crowding does modulate gelsolin severing activity, causing a shorter average filament length, and increasing the likelihood of short filaments. We have also visualized real-time filament severing using time lapse total internal reflection fluorescence (TIRF) microscopy imaging, which can be used to calculate the filament disassembly rate. Our results show that macromolecular crowding modulates gelsolin-mediated actin filament severing and offers insights into the function of both actin and gelsolin in the intracellular space.
Colorectal cancer (CRC) is both the third most common and second deadliest cancer in the United States. Despite recent advances in treatment, the 5-year survival rate remains poor (14%) due to multiple factors including delayed diagnosis, chemoresistance, and disease heterogeneity. In addition, a relative deficiency of targetable clinical markers prevents development of patient-specific treatment and thus hinders optimal management. Recent evidence has implicated the endocrine Klotho Beta - Fibroblast Growth Factor 19 - Fibroblast Growth Factor Receptor 4 (KLB-FGF19-FGFR4) signaling axis in various cancer settings including CRC. Where FGF19 and FGFR4 have established oncogenic properties, the role of KLB in CRC remains virtually unknown. A wide-ranging analysis of CRC RNA-seq data within The Cancer Genome Atlas (TCGA) database reveals tumor-suppressor-like properties for KLB with expression directly correlating with survival probability. In addition, we also found significantly reduced KLB expression in all CRC subtypes compared to normal tissue. In contrast, FGF19 expression revealed oncogenic properties while FGFR4 showed no clinical correlation, in line with previous studies. In vitro analysis of KLB expression in various CRC cell lines showed global downregulation associating indirectly with FGFR4 expression. Moreover, KLB levels were related to cell line “stemness” and thus may be an important mediator of epithelial-to-mesenchymal transition (EMT), a pre-requisite of metastasis in CRC. Further exploration of signaling pathways and in vivo modeling will be required to delineate KLB’s function in modulating CRC oncogenesis. This study hopes to establish KLB as a clinically relevant and targetable marker for CRC tumorigenesis.
31. Sociodemographic Factors Associated with Smoking Cessation Among Smokers who Attempted to Quit

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Smoking cessation leads to many health benefits including reduced risk for lung cancer, heart disease and stroke. The goal of this study was to identify sociodemographic characteristics that are positively associated with smoking cessation. We used pooled 2010-2011 and 2014-2015 data from the Tobacco Use Supplement to the Current Population Survey (TUS-CPS) administered by the US Census Bureau. The data (n=15,897) were reported by former smokers who quit smoking within the past year and were abstinent from smoking for seven days or longer and current smokers who had a serious quit attempt within the past year. To adjust for the TUS-CPS complex design we used Rao Scott Chi-square tests. Significance level was 5% for each test. The primary measure—smoking cessation—differentiated among successful and unsuccessful quitters. Sociodemographic characteristics included age, sex, race/ethnicity, education, marital status, employment status, and two residence factors (US region, metro/non-metro area). The overall prevalence of smoking cessation was 20.8% (SE=0.36%). Age (p<0.001), race/ethnicity (p<0.001), education (p<0.001), marital status (p<0.001), employment status (p<0.001), region (p=0.013), and metropolitan status (p=0.014) were significantly associated with smoking cessation. The prevalence of smoking cessation ranged drastically among specific populations, e.g., from 10.0% (SE=4.03%) for 65+ year-old people to 42.5% (SE=6.14%) for 25-44 year-old people. Our study suggests that smoker’s demographic characteristics are associated with success (or failure) of a quit attempt. Existing smoking cessation programs should be refined to improve the odds of successful quitting and increase the prevalence of long-term smoking cessation across all populations.
Cancer is the second leading cause of death worldwide and was responsible for the death of one in six people in 2018. Pancreatic ductal adenocarcinoma (PDAC) is the most lethal form of pancreatic cancer and results in a shockingly low 8% five year survival rate. Prior research in our lab has shown that PDAC has increased intracellular polyamine levels. Polyamines play crucial roles in cell growth and interact with DNA, RNA and proteins. Polyamine homeostasis is maintained by a balance of biosynthesis, catabolism and transport. Thus, targeting regulation of these growth factors may serve as an effective cancer therapy. Difluoromethylornithine (DFMO) is a commonly used drug to inhibit polyamine biosynthesis, while polyamine-based polyamine transport inhibitors (PTIs) are used to inhibit polyamine import. Polyamine-based PTIs can be considered as pan inhibitors since they contain a polyamine motif in their structure and compete with native polyamines for cellular entry. It is unclear whether all modes of polyamine uptake are used in the rescue of DFMO treated cells. Since more than one polyamine entry mode is possible, compounds which target specific import modes may have clinical value. Such molecules would be helpful to deconvolute the complex polyamine transport system. To address this possibility, we have identified new small molecules, which inhibit spermidine rescue of DFMO treated PDAC cells and the entry of a cytotoxic probe. These efforts are important because they could lead to a new anti-cancer strategy based on the combination therapy of DFMO + molecules which disrupt intracellular polyamine trafficking.
33. Set Intersection Bar Plots to Visualize Key Reasons of Using E-Cigarettes

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Analyses of multiple sets of attitudinal and behavioral measures, and intersections of these sets are common in public health research. The objective of this project was to construct set intersection bar plots, specifically UpSet plots, to visualize the key reasons for using e-cigarettes among adult (18+) e-cigarette users in the US. We demonstrate that the UpSet plots can serve as an effective visualization tool that helps communicate research findings to the general public. We use the 2010-2011 and 2014-2015 Tobacco Use Supplement to the Current Population Survey data for respondents who self-identified as current e-cigarette users at the assessment. We used the data to explore intersections of multiple sets of reasons for e-cigarette use among diverse age and sex groups. We illustrated the findings using UpSet plots and Venn diagrams. The plots and diagrams were constructed using R Studio with the UpSetR and Venn packages. Our results demonstrated that Venn diagrams provide a more useful technique (relative to UpSet plots) in the cases involving two or three sets. However, for cases involving four or more sets, Venn diagrams become increasingly more difficult to understand. In these instances, the UpSet plots provide a more valuable technique because their complexity remains consistent.
Genetic Mechanisms in the Pathogenesis of Tetralogy of Fallot

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Tetralogy of Fallot is a cyanotic congenital heart defect affecting 1 in every 10,000 live births, making it the most common defect of its type. It is characterized by four abnormalities, including a high ventricular septal defect, an overriding aorta, pulmonary stenosis, and right ventricular hypertrophy. While neural crest cell disorders like DiGeorge’s syndrome are known to be associated with a high incidence of Tetralogy of Fallot the pathogenesis of non-syndromic Tetralogy of Fallot still remains unclear to researchers. Understanding this process could help lower the incidence of Tetralogy of Fallot, and provide treatment options for long term complications. Therefore, the aim of this paper is to understand the genetic mechanisms involved in the pathogenesis of the defect. This is accomplished by reviewing journal articles related to congenital heart defects for relevant information about growth factors, environmental factors, and developmental genes responsible for the formation the defects present in Tetralogy of Fallot.
Vector mosquitoes such as *Aedes (Ae.) aegypti* and *Ae. albopictus* pose a major threat to public health due to their ability to transmit diseases such as dengue, chikungunya and Zika virus. This threat is increased due to the development of resistance to existing chemical pesticides. Thus, novel pesticides that do not spur resistance in the target insect need to be developed. For this purpose, nanoceria (CNP) and silver-doped nanoceria (AgCNP) were chemically synthesized, characterized by HR-TEM, XPS, XRD, ICP-MS and solution spectroscopy and tested for their larvicidal and adulticidal properties in *Ae. aegypti* mosquitoes. Uptake and biodistribution of nanoparticles within larvae and adult mosquitoes was confirmed by fluorescence microscopy and nanoCT imaging. Under starved conditions, AgCNPs showed 100% and approximately 90% lethality in 1st and 3rd instar larvae, respectively. Further, fecundity studies on adults emerged from larvae surviving nanoceria treatment showed reduced body and wing size. Conversely, both non-doped and doped nanoceria failed to show any lethal effects on adult *Ae. aegypti* mosquitoes. Though no mosquitocidal activity in adults was observed, the amount of eggs generated (i.e., oviposition) per mosquito fed AgCNPs in blood was significantly reduced, although the body and wing size of the resultant progeny was unchanged. Through these observations, we conclude that AgCNPs show promising mosquitocidal properties that could impact global health through decreasing vector disease transmission.
T-bet deficiency impairs CD4 T cell effector trafficking to Influenza A Virus infected lungs but not their anti-viral functions.

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CD4 T cell responses against IAV are predominantly classified as Th1, characterized by strong IFN-γ production. However, protection by IFN-γ is unclear, suggesting that full Th1 polarization maybe dispensable for effective CD4 immunity. We test this hypothesis by analyzing transgenic CD4 T-cells recognizing IAV that are deficient in the transcription factor T-bet (Tbx21−/−), the ‘master regulator’ of Th1 differentiation. 3x10⁶ Wild-type (WT) or Tbx21−/− effectors, primed under Th1 conditions, were transferred to naïve congenic WT mice then infected with lethal IAV. Tbx21−/− effectors protected mice, promoting similar reductions in viral titer and weight loss recovery as WT cells. However, T-bet deficiency impacted production of several cytokines by the effectors resulting in less IFN-γ and GM-CSF but more IL-2, IL-4 and IL-17. Although the peak magnitude of WT and Tbx21−/− effector responses were similar in secondary lymphoid organs, significantly fewer Tbx21−/− cells were observed in the lungs. We thus titrated effectors to 1x10⁶ cells, to expose any potential differences in protection arising from differential lung homing. Lower effector transfer resulted in higher viral titer and delayed weight loss recovery in mice given Tbx21−/− versus WT cells. Mechanistically, Tbx21−/− cells expressed lower levels of the chemokine receptor CXCR3. CXCR3 blockade in mice given WT effectors reduced their numbers in the lungs and delayed weight loss recovery matching patterns seen in mice given Tbx21−/− effectors. Thus, while prototypical Th1 functions are dispensable for protection against IAV, T-bet-dependent chemokine responses are required to maximize anti-viral CD4 responses at the site of infection.
mTOR inhibitor rapamycin can reduce the antiretroviral therapy induced cardiotoxicity

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Accumulating evidences indicate that the combined antiretroviral therapy (cART) has dramatically increased the life expectancy of HIV infected individuals. However, existing HIV population on cART show several complications including liver or pancreas damage, central nervous system disorders and cardiovascular disease (CVD). Unexpectedly the prevalence of HIV+ associated cardiovascular disease is on the rise. Therefore, we need a better understanding of the therapeutic targets and mechanism to improve the heart function of HIV population. In the current study, we tested the effect of antiretroviral drugs on the cardiomyocytes function. Neonatal rat cardiomyocytes were treated with Ritonavir, Atazanavir, Abacavir, and Lamivudine individually, and combined, respectively. After 24 hours treatment, immunoblot analysis shows that there is upregulation of autophagy marker proteins LC3 II, P62 levels in cART treated cardiomyocytes compared to control cells. Similarly, cART treated cells shows upregulation of endoplasmic reticulum (ER) resident proteins expression with mitochondrial dysfunction. These results are suggesting that cardiomyocytes treated with cART have dysregulated autophagy, higher ER stress and mitochondrial dysfunction. The beneficial cardiovascular effects of rapamycin are well described; though, it is unknown whether rapamycin ameliorates the harmful effects of cART-induced toxicity in cardiomyocytes. Hence, in this study, we intend to investigate the effects of rapamycin on rat cardiomyocytes treated with cART. Our results indicate that rapamycin treatment ameliorates the autophagy dysfunction as well as, it normalizes the mitochondrial function and reduces the ER stress. In conclusion, we show that pharmacological drug rapamycin may help to identify therapeutic target for treatment of HIV+ associated cardiovascular disease.
In our pursuit to discover the next generation of antimalarials from novel areas of chemical space we are screening a large library of diverse fungi to identify secondary metabolites with activity against the malarial parasite. Filamentous fungi are rich source of bioactive compound and are capable of synthesizing a wide range of novel pharmacophores. The Cichewicz fungal collection from the University of Oklahoma contains tens of thousands of novel fungal isolates secured from diverse habitats and ecological niches across the United States. We hypothesize that these fungal secondary metabolites, which are underexplored for antimalarial drug discovery, will provide us with a unique opportunity to explore medicinally relevant, but untapped chemical space for the discovery of essential malarial therapeutics. In this effort, we have screened libraries of 750 pure compounds, and 4,500 extracts obtained from diverse fungal sources for their ability to inhibit intraerythrocytic growth of \textit{P. falciparum} \textit{Dd2} strain using a SYBR Green I-based phenotypic fluorescence assay. As a counter screen, we evaluated the cytotoxicity of the top hits in HepG2 cells using the MTS ((3-(4,5 dimethylthiazol-2-yl)-5-(3-carboxymethoxyphenyl)-2-(4-sulfophenyl)-2H-tetrazolium, inner salt) cell proliferation assay. Our hit rate, with an initial screening at concentrations of 1 µM for pure compounds or 2.5 µg/mL for crude extracts is approximately 10%. This screening has identified selective antiplasmodial compounds with picomolar EC_{50} values, including histone deacetylase inhibitors. These unique pharmacophores from wide areas of chemical space can provide chemical starting points to develop lead compounds against diverse cellular targets.
Freeze-FRESH: A 3D printing technique to produce biomaterial scaffolds with hierarchical porosity

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Tissues are comprised of nanoscale, microscale, and macroscale features organized in hierarchical structures. Incorporating hierarchical structures into biomaterial scaffolds may enable better resemblance of native tissue structures and improve cell interaction, but it is challenging to produce such scaffolds using conventional scaffold production techniques. We developed Freeze-FRESH (FF), a technique that combines 3D printing (3DP) and freeze-casting, to produce 3D printed scaffolds with microscale pores in the struts. FF scaffolds are produced by extrusion 3DP using a support bath, followed by freezing and lyophilization. The combination of microscale pores in the scaffold struts and macroscale pores from the printed design created a hierarchical pore structure for the FF scaffolds, while control scaffolds had only macroscale pores. FF scaffolds frozen at -20 °C and -80 °C had similar pore sizes, due to freezing in the support bath. The -20 °C and -80 °C FF scaffolds had porous struts with 63.55 ± 2.59% and 56.72 ± 13.17% strut porosity, respectively, while control scaffolds had a strut porosity of 3.15 ± 2.20%. The -20 °C and -80 °C FF scaffolds had a pore wall stiffness of 41.85 ± 14.41 kPa and 56.63 ± 13.20 kPa, respectively, and demonstrated resilience during handling. FF scaffolds supported MDA-MB-231 cell growth and had significantly higher cell numbers than control scaffolds. Cells aggregated and formed clusters on the porous struts of FF scaffolds with similar cell morphologies observed on freeze cast scaffolds. The FF method successfully introduced microscale porosity into the 3DP scaffold struts to produce hierarchical pore structures that enhanced MDA-MB-231 growth.
The Effect of a High Saturated Fat Diet and Polyunsaturated Fat Diet on Osteogenesis, Bone Microarchitecture, and Structural Strength

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Age-related bone loss is inevitable in both men and women and we soon will have a greater number of people at extreme old age than ever before. Diet is a variable, constantly modified entity that plays a significant role in bone regeneration and repair and the Western Diet is characterized by its unhealthy components, specifically the excess amount of saturated fat intake. A highly saturated fat diet is associated with a number of diseases due to chronic activation of pro-inflammatory pathways, adipogenesis, osteoclastogenesis and subsequent bone-resorption. Research suggests that polyunsaturated fats (PUFA) have many potential health benefits and this study investigated the hypothesis that polyunsaturated fatty acids interact with both hematopoietic and stromal derived bone cells leading to inhibition of osteoclastogenesis and increased osteoblastogenesis thereby suppressing bone resorption and loss. A review of the literature was carried out and results showed that the molecular and cellular processes involved in bone formation are heavily influenced by diet. Consumption of omega-3 fatty acids such as docosahexaenoic acid and eicosapentaenoic acid were significantly associated with increased bone regeneration through mechanisms including reduced inflammatory cytokine release (Il-1, Il- 6, TNFα), prostaglandin E2 production and enhanced calcium transport and retention. However, omega-6 fatty acids were typically pro-inflammatory. This study suggests a potential role for PUFA as a non-pharmacological method of reducing bone loss in our aging population.
Pancreatic cancer is the fourth leading cause of cancer death in the United States, with a five-year survival rate of less than 8%. Pancreatic ductal adenocarcinoma (PDAC) is extremely chemo-resistant and has poor patient prognosis. Since PDAC cells exhibit increased expression of polyamines, which play a role in transcription, translation and promote cell growth, therapeutics which exploit the altered polyamine metabolism in pancreatic cancer cells may overcome this challenge. Inhibiting ornithine decarboxylase (ODC) with difluoromethylornithine (DFMO) blocks polyamine biosynthesis, however, tumors can circumvent blockade of polyamine biosynthesis by upregulating polyamine import. Therefore, to prevent tumor escape via alternative polyamine transport, we evaluated the effects of DFMO in combination with a polyamine transport inhibitor (PTI), Trimer44NMe. Promising studies from our group showed intraperitoneal injections of Trimer44NMe PTI in combination with oral DFMO increased survival of mice with pancreatic tumors. We sought to test higher concentrations of the PTI using immune competent mice with pancreatic cancer to determine drug effects on the expression of the proteins associated with polyamine pathways, and the tumor microenvironment. Using a higher dose of the PTI in combination with DFMO, we will also test effectiveness on overall animal survival. Future studies will analyze the response of stromal cells and immune cells in the tumor microenvironment to drug treatments using genetic mouse models to recapitulate PDAC development. Study impact is expected to be multifactorial in advancing our understanding of how targeting polyamine signaling and molecular interactions between tumor, stroma and immune cells improve patient survival.
Chemotherapeutic drug, Doxorubicin (Dox) is an effective agent used to treat various cancers. Unfortunately, further use of Dox has been restricted due to serious side effects including skeletal muscle toxicity. Dox-induced muscle toxicity (DIMT) is mediated through enhanced oxidative stress, inflammation, and unwanted cell death, apoptosis and necrosis. However, it is still not known whether an inflammation-mediated cell death known as “pyroptosis” plays a role in DIMT. Therefore, DIMT model was generated using C57BL6/J mice (10±2 weeks, male and female), which were injected with either saline (control) or Dox (12mg/kg bodyweight (BW) cumulative dosage through 3 intraperitoneal (i.p.) injections in alternative days). 14 days following the last injection, mice were sacrificed and soleus muscle (SM) was harvested for further analysis for inflammasome formation (TLR4-NLRP3), pyroptotic markers (TLR4, NLRP3, caspase-1, IL-1β, and IL-18), and pro-inflammatory cytokines (TNF-α and IL-6) using immunostaining and western blot techniques. Moreover, muscle function analysis was performed prior to sacrifice. Our immunostaining and western blot analysis revealed a significant increase (p<0.05) in expression of inflammasome markers, pyroptotic markers, as well as pro-inflammatory cytokines following Dox administration in comparison to control. Furthermore, muscle function data shows significant reduction (p<0.05) of muscle strength in Dox-treated mice vs. control. In conclusion, our data indicates that Dox induces NLRP3 inflammasome-mediated pyroptosis, increases inflammation, and causes muscle weakness. The significance of this study is to bring a novel mechanistic view on the pathophysiological role of pyroptosis in DIMT.
Diabetic cardiomyopathy and associated muscle toxicity is a major concern, therefore it is essential to understand the mechanism and pathophysiology of diabetic skeletal muscle which remains unknown. The current study is undertaken to investigate whether there is an upregulation of High Mobility Group-B1 (HMGB1) that initiates inflammation induced pyroptosis and involves adverse muscle remodeling. C57BL/6J mice (10±2 weeks) were divided into 2-groups (n=16/group; 8males and 8females); control (saline) and STZ treated (200mg/kg body weight) via i.p. injection. Diabetes was established by determining the increased levels of glucose in STZ animals. At Day-42, muscle function was determined using Grip-strength meter and Rotarod. Mice were euthanized and gastrocnemius (GM) muscle was harvested. Pyroptotic initiator HMGB1, inflammasome marker (NLRP3), and pyroptotic markers (caspase1, IL1β, IL-18 and GSDMD) were examined using immunohistochemistry, western blotting and RT-PCR. Muscle atrophy and fibrosis was examined histologically. We observed a significant (p<0.05) increase in glucose levels, pyroptotic initiator HMGB1, inflammasome and pyroptotic markers in GM of STZ administered mice as compared to control. A significant (p<0.05) decrease in myofibrillar area was observed in diabetic mice. Atrophy was also confirmed by significant increase (p<0.05) in MuRF1 gene expression. Interstitial and vascular fibrosis was significantly (p<0.05) increased in STZ group versus control. Moreover, a significant decrease (p<0.05) in muscle functions were observed in diabetic animals compared to control. In conclusion, our data suggest that diabetes involves a novel form of inflammation induced pyroptosis that leads to muscle dysfunction. Further therapeutic approaches need to be developed to target pyroptosis in diabetes.
Diabetic cardiomyopathy is a common complication of diabetes and can cause heart failure, arrhythmia and sudden death. The current study was designed to understand whether TLR4 initiates NLRP3 mediated pyroptosis that contributes in adverse cardiac remodeling and heart dysfunction in streptozotocin (STZ)-induced cardiomyopathy. C57BL/6J mice (n=32, 16 mice/group) were divided into control and STZ (diabetic) groups. STZ groups were administered streptozotocin (200 mg/kg, IP injection, Type-I diabetic dose), whereas control animals received 0.9% saline. After 6 weeks, random blood glucose and echocardiography was determined. H&E, Masson`s Trichrome staining, western blots and immunostaining assay were performed in heart tissues. STZ administration significantly increased inflammation driven pyroptotic markers such as TLR4, NLRP3, caspase-1, IL-1β and IL-18 compared to controls (P<0.05). Further, TLR4, NLRP3 and caspase-1 overexpression (P<0.001) were confirmed by western blots and densitometric analysis. STZ administration increased adverse cardiac remodeling in mice by increasing cardiomyocyte diameter area, interstitial fibrosis and vascular collagen deposition versus controls (P<0.001). Moreover, a significant deterioration in heart function was achieved after STZ administration, as indicated by decreased left ventricular (LV) heart function (EF% and FS%) and increased LV dilatation (LVIDd, LVIDs, EDV and ESV) parameters relative to controls (P <0.05). Our data suggested that hyperglycemia induced TLR4 that initiates NLRP3 inflammasome-mediated pyroptosis signaling cascades leading to cardiac fibrosis; deteriorate myocardial remodeling and LV dysfunction in diabetic cardiomyopathy.
The effect of E-cigarette nicotine vape on host-bacterial interactions in the oral cavity

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Oral squamous cell carcinoma (OSCC) is the sixth most common form of cancer, comprising over 5% of all cancer cases worldwide, characterized by poor prognosis and a low 5-year survival rate. Cigarette smoke is a well-established risk factor for OSCC. While it is known that cigarette smoking contributes to pathogenesis through alterations of the oral microbiome, the effect of the recently popularized E-cigarette (E-cig) vape on the resident microbial communities and the epithelium has not been well studied yet. We hypothesize that E-cig vape modulates host-microbe interactions by inducing bacterial stress responses and consequently increases epithelial inflammation and proliferation. We exposed normal oral and esophageal cells to E-cig vape with and without nicotine (3 mg) and measured pro-inflammatory cytokine expression through qRT-PCR. We measured bacterial adhesion to epithelial cells and pro-inflammatory cytokine expression. Our data showed that E-cig vape with 3 mg nicotine elicited an inflammatory response, measured by an increased expression of COX2 and the cytokines TNF-α, IL-8 and IL-1β. We also showed that exposure of epithelial cells to Staphylococcus aureus, collected from smokers and non-smokers elicits an inflammatory response. However, the response is significantly greater in the presence of S. aureus from smokers. Functionally, E-cig vape exposure reduces cell proliferation. Next, we will expose S. aureus to E-cig and investigate the bacterial response and the resulting host-bacterial interaction changes. We aim to understand how E-cig vape may be causing the initiation of the tumorigenesis process.
Involvement of RIP2 in the Production of Specialized Pro-resolution Lipid Mediators

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Nucleotide Oligomerization Domain 2 (NOD2) is a cytosolic receptor which recognizes muramyl dipeptide (MDP), a breakdown product of bacterial peptidoglycan. Receptor Interacting Serine/Threonine Kinase 2 (RIP2) mediates many of the downstream signals resulting from NOD2 engagement including activation of NF-κB, MAPK, IRF, and autophagy pathways which are important in mediating host defense. Dysregulation of NOD2:RIP2 signaling (decreased and increased) has been associated with the development of various inflammatory disorders, and genetic loss or pharmacologic inhibition of RIP2 has been demonstrated to alleviate disease in murine models of inflammatory bowel disease, multiple sclerosis, and allergic airway inflammation. Conversely, multiple studies also demonstrate the efficacy of prophylactically promoting NOD2:RIP2 signaling to reduce experimental colitis, bacterial, and viral infections. These paradoxical findings may be reconciled if NOD2/RIP2 engagement induces both a host defense and an immunoregulatory response. In support of this dual role of NOD2, we demonstrate the involvement of NOD2/RIP2 in the production of Specialized pro-resolving lipid mediators (SPMs). These are lipid mediators shown to have protective effects in various inflammatory diseases in both dampening inflammation and promoting resolution or wound healing. We show that RIP2 promotes activation of key enzymes (5 Lo and 15 Lo) involved in production of SPMs. Moreover, RIP2 promotes production of SPMs during the course of an MDP-induced peritonitis model. These data suggest that RIP2 activity is important both in the production of, and in the regulation of SPMs. This finding will have important implications for future therapies designed to target this kinase.
Macrophages have a duality of function; initially, they can defend tissues from invading pathogens and promote wound healing after injury, but prolonged inflammation can cause damage to native tissue and initiate tumorigenesis. Pro-inflammatory cytokines like IL-1β are important modulators of both acute and chronic inflammation. The polarization status of the macrophage is both a product of and a contributor to the cytokines of the microenvironment. Of particular interest is Activin A, a signaling molecule that may act as a pro-inflammatory immune modulator. Activation of the Activin A signaling pathway can have an enduring pro-inflammatory effect on the polarization of macrophages. Upregulation of Activin A has been reported for inflammatory gastrointestinal diseases including pancreatitis and inflammatory bowel disease. While nothing is known about its role in esophageal disease, we have recently shown that the levels of Activin A in human tissues increase in a stepwise fashion from normal tissue to gastroesophageal reflux disease (GERD) to Barrett’s esophagus (BE), a pre-cancerous lesion. We test the hypothesis that the increased level of Activin A secreted by bile acid-tolerant esophageal cells may perpetuate a chronic inflammatory condition by reducing the plasticity of tissue resident pro-inflammatory macrophages, contributing to Th1 inflammation and an aberrant wound healing response. Using the monocytic cell lines, THP-1 and SC, we show that priming the cells with Activin A leads to an increased expression of pro-inflammatory IL-1β, pSTAT1, and CD 80. This chronic inflammation leads to BE, and if left unchecked, esophageal adenocarcinoma (EAC).
Migraine is a unilateral throbbing head pain that may initiate by various stimuli such as light, sound, and even head movements. The pain may last for 4-72 hours and patients may experience nausea and vomiting, photophobia, or phonophobia. Two main types of migraine are frequently discussed, migraine with aura also called classic migraine and migraine without aura referred to as common migraine (Samsam M, Cent Nerv Syst Agents Med Chem. 2012, 12:158-72).

Given that migraine is of an unknown pathophysiology treatment of this condition has proven difficult. Traditionally it was believed to be primarily a vascular condition, but recent evidence suggests migraine is a brain disorder that contains components of sensory disfunction and abnormal sensory processing in pathways of the brainstem which are responsible for regulating vascular tone and pain. Many patients do not respond to drug treatments or have contraindications to pharmacological approach. To mitigate these issues migraine therapy is also moving towards preventative treatments focusing on improvement in quality of life. Such rising treatments include neurostimulation (Samsam M, Neuro Open J. 2016; 3: e5-e10) of certain nerve pathways as well as yoga, acupuncture, mild physical therapy (Amin, FM, J. Headache & Pain 2018; 19:83) and even cannabinoid use (Leimuranta P, Front Pharmacol. 2018, 24;9:420). In this capstone project, we review the literature on alternative therapies of migraine.
49. Effects of Nicotine in Diet and Smoking on Parkinson’s Disease

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Nicotine intake from smoking and certain food products have been found to be effective in patients with Parkinson’s disease (Ma C, et al., Transl Neurodegener. 2017 Jul 2;6:18). Multiple studies show that nicotine in various products affects the mortality rate of Parkinson’s disease (PD). A recent study has evaluated the relationship between different forms of nicotine exposure such as cigarettes, smokeless tobacco, and environmental tobacco exposure in PD (Rodriquez, T, The Troubling Link between Parkinson’s and Smoking, 2015, www.neurologyadvisor.com). These studies showed the length of tobacco exposure, rather than the amount of tobacco exposure, had an inverse relationship with the onset and development of Parkinson’s disease. Other studies show that transdermal nicotine improves motor scores and slows the degeneration of dopaminergic nerve terminals (Rausch, T. et al., Neurogastroenterol Motil. 1998 Jun;10(3):263-70). In this capstone project, we looked at the current opinion about the possible effects of nicotine on PD and its mechanism of action. In addition, we used the ADME WorkBench from AEgis, a pharmacokinetic predictive simulation software to evaluate the relationship between nicotine and the administration of certain drugs in humans. We show the concentrations of nicotine, L-Dopa (major drug in the treatment of PD), and metoprolol (a co-administered drug to control the blood pressure) in the brain, gut and the lung using the simulation software and predict their possible effects.
Prostate cancer (PC) has become one of the most common types of cancer in men, second only to lung cancer. PC is treatable if caught early in older men despite of its increasing prevalence (Vane S. Urologic Nursing, 2019, 39(3): 133-138). Increased serum testosterone is a major cause of PC since it actively feeds the tumor while localized, and possibly later, when it metastasizes to other tissues. However, some argue that testosterone may not be as important as other factors or that it does not affect the cancer at all (Michaud J.E, Therapeutic Advances in Urology, 2015, 7.:378-387). Androgen receptor, might have larger role in PC progression. Androgen receptor is more associated with castrate-resistant prostate cancer, where its increase leads to prevalence of the tumor even after androgen deprivation therapy (Tan E, Acta Pharmacologica Sinica, 2015, 36:3-23). PC can occur due to the use of drugs like steroids and hormone therapy or testicular tumors. Although it is very rare for the occurrence of a testicular tumor with prostate cancer, increased testosterone levels may be crucial as it could be involved in the progression of the cancer. In this capstone project, we look at the PC and its relation with testosterone.
Nanotechnology is a multidisciplinary field that investigates the development of nanomaterials across multiple disciplines such as engineering, chemistry, physics, medicine, and agriculture. Nanomaterials are part or all man-made substances with a dimension size in the 1-100 nm range. Nanomaterials and devices development have been widely studied for drug delivery, anticancer treatment, agriculture biocide, antimicrobial agents, and imaging contrast agents. In agriculture, nanomaterials are being widely investigated for the development of pesticides, bactericides, and fertilizers. For instance, nanomaterials are being used to develop treatments and solutions to crop loss due to bacterial pathogens. Therefore, Zinkicide is a nanomaterial-based biocide that was developed to treat citrus greening disease also called Huanglongbing (HLB). Zinkicide is a zinc-based nanomaterial. Zinkicide exhibits systemic antimicrobial abilities due to its small size (<10nm), which is a key quality for the treatments of HLB infected plants. Furthermore, Zinc Oxide (800nm-bulk) is widely used in many applications including ceramics, pharmaceutical, personal care (sunscreen), and animal feed. Zinc Oxide material are recognized by the U.S. Food and Drug Administration as “generally recognized as safe” (1). Zinc Oxide usage in agriculture was primarily as a fertilizer, but Zinc oxide also has antimicrobial properties. In this study, a comparison of an equal concentration of Zinc in Zinkicide and Zinc Oxide will be completed to investigate the antimicrobial activities including membrane damage capabilities of both Zinkicide and Zinc Oxide on Pseudomonas syringae, a gram-negative plant pathogen, and Clavibacter michiganensis, a gram-positive plant pathogen.

References

52. Elucidating the molecular mechanisms driving the environmental persistence of *Vibrio cholerae*

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*Vibrio cholerae*, the etiological agent of the severe diarrheal disease Cholera, is a natural inhabitant of estuarine and brackish waters. Often found associated with other marine organisms, this bacterium is frequently impacted by the fluctuating nature of its environment. In order to withstand conditions that are not favorable for its growth or survival, *V. cholerae* has evolved to employ strategies that allow it to endure adverse conditions by entering an environmentally persistent state (PS). The individual role of some abiotic factors such as nutrient limitation or pH have been studied in association with entry into PS. However, in the natural environment of *V. cholerae*, conditions are dynamic as changes to these abiotic factors occurs simultaneously. To date, the complex interplay between abiotic variables and the regulatory mechanisms driving the entry into the PS remains mostly enigmatic. Using a microcosm composed of artificial sea water (ASW), we identified a synergistic effect between the different abiotic variables such as salinity, temperature, pH, and oxygen availability, indicating their interdependency. Additionally, proteolysis of ToxR, a major virulence regulator that regulates over 150 genes in *V. cholerae*, has been previously associated with entry of the bacterium into the PS. In this study, we examined the synergistic relationship between abiotic environmental conditions and the ToxR-regulatory cascade. We identified a number of virulence-associated genes that were differentially regulated in a ToxR-dependent manner. This project will further elucidate the role of these genes in the entry of *V. cholerae* into a PS.
Methionine Depletion Agents for the Treatment of Pancreatic Ductal Adenocarcinoma

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Pancreatic cancer is expected to be the second leading cause of cancer-related deaths by 2030. The most common form of pancreatic cancer is pancreatic ductal adenocarcinoma (PDAC). The native polyamines (putrescine, spermidine, and spermine) are low molecular weight amines that exist as polycations at physiological pH. These natural polycations interact with DNA, RNA, and influence many cellular processes. Intracellular polyamine levels are maintained via a balance of polyamine biosynthesis, catabolism, and transport. Ornithine, an amino acid obtained from L-arginine, is decarboxylated by ornithine decarboxylase (ODC) to form putrescine. The biosynthesis of the higher polyamines, spermidine and spermine, requires the addition of an amino-propyl group donated by decarboxylated S-adenosylmethionine (dc-SAM), which itself is derived from the amino acid L-methionine. Therefore, one way to affect the growth of PDAC cells is to deplete their intracellular polyamine pools by inhibiting methionine import. LAT1 is the principal transporter of methionine into human cells and we have identified a new LAT1 inhibitor. As expected, this inhibitor reduced intracellular methionine and polyamines levels along with inhibited cell growth. This compound contains two chiral centers and the question remains which diastereomer is the most potent LAT1 inhibitor design. This report will describe our efforts to optimize the LAT1 inhibitor design. Utilizing organic synthesis, we synthesized the four isomers of the LAT1 inhibitor. We then evaluated these compounds for their ability to inhibit L3.6pl cell growth, deplete intracellular methionine levels, and reduce intracellular polyamine pools. A success here could provide new drugs which target pancreatic cancers via methionine starvation.
54. Ecological drivers and molecular mechanisms driving the environmental persistence of Vibrio cholerae

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Vibrio cholerae, the etiological agent of the severe diarrheal disease cholera, is a natural inhabitant of estuarine and brackish waters. The bacterium is often found associated with other marine organisms and is regularly impacted by the fluctuating nature of its environment. In order to withstand conditions that are not favorable for its growth or survival V. cholerae enters an environmentally persistent state (PS). The individual role of some abiotic factors such as nutrient limitation or pH have been studied in association with entry into PS. However, in the natural environment of V. cholerae, conditions are dynamic as changes to these abiotic factors occurs simultaneously. To date, the complex interplay between abiotic variables and the regulatory mechanisms driving the entry into the PS remains mostly enigmatic. Using a microcosm composed of artificial sea water (ASW), we identified a synergistic effect between the different abiotic variables such as salinity, temperature, pH, and oxygen availability, indicating their interdependency. Additionally, proteolysis of ToxR, a major virulence regulator that regulates over 150 genes in V. cholerae, has been previously associated with entry of the bacterium into the PS. In this study, we examined the synergistic relationship between abiotic environmental conditions and the ToxR-regulatory cascade. We identified a number of virulence-associated genes that were differentially regulated in a ToxR-dependent manner. This project will further elucidate the role of these genes in the entry of V. cholerae into a PS.

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Vibrio cholerae, the etiological agent of the severe diarrheal disease cholera, is a natural inhabitant of estuarine and brackish waters. Interestingly, within the species only a small subset of strains, the pandemic group (PG), have emerged as pathogens and can cause the disease, while the majority cannot. Previous studies show that PG strains have a genomic background containing virulence adaptive polymorphisms (VAPs). VAPs are allelic variations of core genes that confer pre-adaptations to virulence and naturally occur in environmental populations of V. cholerae. To date, the conditions that drive their transmission in a population leading to their accumulation in a given genome remain enigmatic. Given that the emergence of non-choleragenic strains of V. cholerae as pathogens are not dependent on the acquisition of virulence factors or niche location, we hypothesized that prevalence of these VAPs may be driven by abiotic and biotic selective pressures. Using the Indian River Lagoon as a model ecosystem, samples were collected from two environmentally contrasting sites with drastically different anthropogenic influences. Genetic analysis of these isolates revealed that a proportion contained PG alleles of VAPs, suggesting that the prevailing environmental conditions foster the selection of virulence-associated genes. Furthermore, water quality data collected from each site will be used to determine the dependency of these VAPs on changes in abiotic factors such as pH, temperature, turbidity or chlorophyll. Overall, this project highlights how environmental factors drive the spatiotemporal dynamics of VAPs in V. cholerae and could be applied to other bacterial pathogens with environmental reservoirs.
The obesity epidemic has been recognized as one of the main preventable causes of an array of health hazards including heart disease, type 2 diabetes, certain types of cancer, and other chronic illnesses. Without lifestyle modifications, obesity and its co-morbidities can work parallel to promote a condition known as Non-Alcoholic Fatty Liver Disease (NAFLD). Predicted to affect 34% of the US adult population, NAFLD is characterized by excessive fat accumulation in the liver, which if left untreated can lead to inflammation, hepatocyte damage, and fibrosis. Studies of the disease progression often rely on diets that are predominantly high in fat to model the pathological and metabolic changes, overlooking the significant role played by cholesterol in most NAFLD patients. A high fat high cholesterol diet with fructose water (FFD) was fed to mice for 24 weeks to study the molecular changes that act as pivotal players in the advancement from NAFLD to the more dangerous stage, called NASH (Non-Alcoholic Steatohepatitis). To uncover the extent of cholesterol’s influence on the severity and manifestation of NAFLD, a high fat high cholesterol diet supplemented with sodium cholate (HFHCC) was also fed to mice. After 24 weeks of feeding, RNA was extracted from the mouse livers and sequenced. Transcriptome data has revealed potential compensatory mechanisms in the HFHCC fed mice related to cholesterol usage that allowed them not only to limit fibrosis but maintain insulin sensitivity along with relative metabolic stability. Analysis of mRNA expression changes in the liver will be applied to future studies aimed at understanding the contribution of diet towards malignancies.
57. *Drosophila* ovarian follicle cells as an *in vivo* epithelial model to study T3SS-delivered early effectors of *Chlamydia trachomatis*.

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*Chlamydia trachomatis* infection is the most frequently reported sexually transmitted disease in the United States, with approximately 2.9 million new infections annually. *C. trachomatis* is a gram-negative, obligate intracellular bacteria that specializes in rapidly invading host cells. Delivery of multiple *C. trachomatis* early effectors into the host cell via a Type 3 secretion system (T3SS) is thought to drive cytological changes important for bacterial invasion. One such effector, Tarp, is delivered into the host cell cytosol within seconds after pathogen attachment. We have shown through in vitro studies that Tarp has both actin polymerization and unique F-actin bundling properties but the biological relevance of these findings have not been fully explored *in vivo*. To address this, we developed a cell biological model in *Drosophila* to study the effect of Tarp on F-actin dynamics *in vivo*. The follicle cell layer is a monolayer of polarized epithelium that surrounds the developing egg. Tarp expression in follicle cells leads to the overgrowth of apical microvilli, which are F-actin-rich membrane protrusions on the cell surface. Microvilli length is determined by the interplay of F-actin polymerization, bundling, and depolymerization—a useful framework for understanding how Tarp alters F-actin dynamics *in vivo*. Furthermore, this cell biological platform will also allow us to: 1) dissect the contributions of Tarp protein domains by expressing different domain deletion mutants; and 2) test for functional interactions between Tarp and other *C. trachomatis* early effectors by simultaneous transgenic expression of different effectors in the follicle cells. Moreover, this work exemplifies the utility of experimentally tractable model organisms such as *Drosophila* in providing new avenues to address existing research questions.
58. Ovarian aging in growth hormone receptor knockout mice supplemented with 17α-Estradiol

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Extending reproductive life can have many implications for women's health-span and lifespan. Decades of studying reproductive function and gonadal manipulations in different model organisms established strong links between reproduction and longevity. The postulated Reproductive-Cell Cycle Theory of Aging states that the factors that regulate reproduction acts in an antagonistic pleiotrophic manner to control aging via cell cycle signaling, suggesting the existence of a tradeoff between growth rate, sexual maturation and longevity. Dwarf mice with growth hormone receptor gene disruption (GHRKO) are characterized by significantly extended lifespan, healthspan and female reproductive function. Recent studies showed that treating mice with 17α-estradiol (17α-E2), an epimer of the primary female sex hormone estradiol extends lifespan in males mice, but surprisingly there was no longevity benefit in female mice. The detailed mechanism of 17α-E2 action on aging is not determined yet, and observed sex specific action of this hormone makes it important to determine its role in females reproductive aging. To decipher the role of 17α-E2 on female health, the aim of this study was to evaluate the ovarian reserve and physiology in normal and GHRKO mice supplemented with 17α-E2. Our study showed that 17α-E2 treatment did not affect ovarian reserve in N mice. However, as we expected long-living GHRKO mice were characterized by increased number of primordial follicles, when comparing with N controls (P=0.011). Surprisingly, the treatment with 17α-E2 depleted the number of primordial follicles in GHRKO ovaries when comparing with non-treated GHRKO mice (P=0.013), and normalizing it with the levels observed in N controls. Importantly, according to our other studies with life extending interventions, such as calorie restriction (CR) and rapamycin, we showed increased ovarian reserve, we speculate the lack of similar alterations due to 17α-E2 might explain lack of lifespan extension in N female mice.

Surprisingly, the observed interaction between genotype and treatment can suggest that 17α-E2 could have negative effects on reproductive health and lifespan in long-living GHRKO female animals. Due to increasing anti-aging potentials and growing participation in hormonal therapies by menopausal women there has to be more extensive studies in the future to better understand the role of sex hormones on women health, longevity and potential complications due to hormonal supplementation during aging.

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Ceria nanoparticles have wide uses within the field of science. From use as sensors to optimization within the industrial field, ceria nanoparticles (CNP) is revolutionizing the field of nanotechnology. With its ability to be structured in multiple shapes, this particle is very versatile. There are many structures of nanoceria including hollow ceria which has the ability to utilize its high surface area. This nanoparticle structure can be created using several methods but a hard template of a silica nanoparticle was utilized to not only control size of the hollow ceria but by principle, its surface area as well. However, ceria nanoparticle’s most promising use is with its biomedical applications. From an absorber of free radicals to its antimicrobial properties, ceria nanoparticles have the potential to revolutionize the way we treat diseases. However, little study has been done utilizing hollow ceria to measure its catalytic and oxidative properties. The high surface area could potentially maximize the effect of its catalytic and oxidative properties compared to regular ceria particles. This paper will not only pursue the catalytic and oxidative properties of hollow ceria, but also delve into its biocompatibility with cells in suspension. The importance of surface area within the ceria nanoparticle will be observed with the use of different sized hollow ceria structures.
Aminoacyl-phosphatidylglycerol synthases are a family of proteins that modify membrane lipids with amino acids using aminoacylated tRNAs, normally used in protein synthesis, as amino acid donor molecules. Modification of membrane lipids with amino acids alters the electrostatic properties of the cytoplasmic membrane and increases bacterial resistance against antimicrobials targeting the membrane (i.e., cationic antimicrobial peptides, glycopeptides, and lipopeptides). Our bioinformatics analysis showed that Actinobacterial pathogens encode more than one aaPGS homolog (in some instances up to six) suggesting that these enzymes carry out distinct biological functions, potentially conferring resistance to a wider spectrum of environmental stressors and antimicrobials. This hypothesis is supported by our preliminary investigations involving two aaPGS homologs in *Corynebacterium pseudotuberculosis* that use diacylglycerol (DAG) as a lipid substrate to generate two novel lipid modifications: alanyl-DAG and lysyl-DAG. We showed that lysyl-DAG is an important determinant for resistance of this species against polymyxin B and for virulence in an insect infection model.

Our long-term goal is to define the complete repertoire of lipid modifications across bacterial species and to characterize their role in cellular physiology, antimicrobial resistance, and pathogenesis. The aims of our current work, which will lay the groundwork for future studies, are to i) explore aaPGS homolog diversity in Actinobacteria and characterize the biochemical functions of representative enzymes from important human pathogens in the genera Mycobacterium, Nocardia, Rhodococcus, and Streptomyces; and ii) determine the role of these proteins in resistance to CAMPs of the human immune system and other antimicrobial agents.
MicroRNAs have been shown to be potent regulators of various biological processes. MicroRNA 146a-5p (miR-146a) is a typical multifunctional miRNA expressed in a wide variety of tissues and cells, and plays several biological processes including an important role in the function of the innate immune system through regulation of inflammation. Previous studies have indicated that miR-146a inhibits IRAK1 and TRAF6 genes that are crucial for pro-inflammatory signaling and regulation of NF-κB activation, which is an important inflammatory regulator responsible for activation of downstream pro-inflammatory cytokines. The aim of this study was to investigate the effect of overexpression and inhibition of miR-146a in IRAK1/TRAF6/NF-κB pathway in normal Human Endothelial Cells (HUVEC) and Human Preadipocytes (HPAD). The effects of miR-146a were assessed by transfecting the cells with miR-146a mimic and inhibitor for 72 hours and analysis of the miRNA level and its target genes using RT-PCR. The transfection of HPAD and HUVEC cells with miR-146a showed increased expression levels of this miRNA in the cells as well as it increased the secretion if this miRNA to the culture media. The overexpression of miR-146a decreased the levels of IRAK1 and TRAF6 in HUVEC cells, while there was no effect of miR-146a on its target genes in HPDA cells. Overall, these data show that the mechanism involved in miR-146a modulation of IRAK1, TRAF6 and NF-κB signaling is different in endothelial cells and preadipocytes in normal conditions, which might be due to different levels or regulation of miR-146a in these two types of cells.
Dynamic transcriptomic analysis reveals ripple effects of maternal binge alcohol consumption in the embryonic mouse heart: Implications for congenital heart defects

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Excessive ethanol (alcohol) consumption during critical early periods of pregnancy is associated with increased risk of congenital heart defects, but the molecular and biochemical mechanisms underlying this phenomenon are not well-understood. To evaluate the impact of a single binge-drinking episode, we administered alcohol (0.25 ml of 30% ethanol via oral gavage) to timed-pregnant C57BL/6J mice (Jackson Laboratories, Bar Harbor, ME) at embryonic day 9.5 (E9.5). This resulted in maternal blood alcohol concentrations of ~0.3% and embryonic concentrations of ~0.16% by 1-3h. Hearts were isolated for RNA extraction at 24h, 48h, and 72h post-treatment for subsequent whole-genome RNA-sequencing (RNA-Seq) analysis. During the first 24-48h, significant (p<0.001; n=5/group) decreases ranging from ~16-fold to >106-fold in magnitude of transcript expression for several key regulators of cardiovascular development, including Wnt7a, Foxb1, and Nkx2.1, was observed in hearts from alcohol-treated dams when compared to saline-treated controls. These differences were no longer evident by 72h post-treatment, but we surprisingly found that maternal binge alcohol induced strong (>300-fold) and significant (p<0.001, n=5/group) increases in several X-linked transcripts including Tsix and Xist, long non-coding RNA transcripts involved in X chromosome inactivation that did not appear until the 72h timepoint. These results imply that maternal binge alcohol consumption has “ripple” effects on embryonic heart gene expression patterns that initially manifest as repression of a few key cardiac target genes known to be important for heart development, followed by a delayed (2-3 days later) activation of major non-coding X-linked transcriptional regulators of X-chromosome inactivation. These ripple-effect transcriptomic changes provide novel insights that may help to explain the underlying biochemical and molecular mechanisms mediating alcohol-induced congenital heart defects.
Student Affairs Committee Mission

The mission of the Student Affairs Committee is to discuss issues pertaining to Masters and PhD students and to serve as the liaison committee between faculty and the Biomedical Sciences Graduate Student Association. The committee is also responsible for the planning of events like the Annual Graduate Student Colloquium.

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