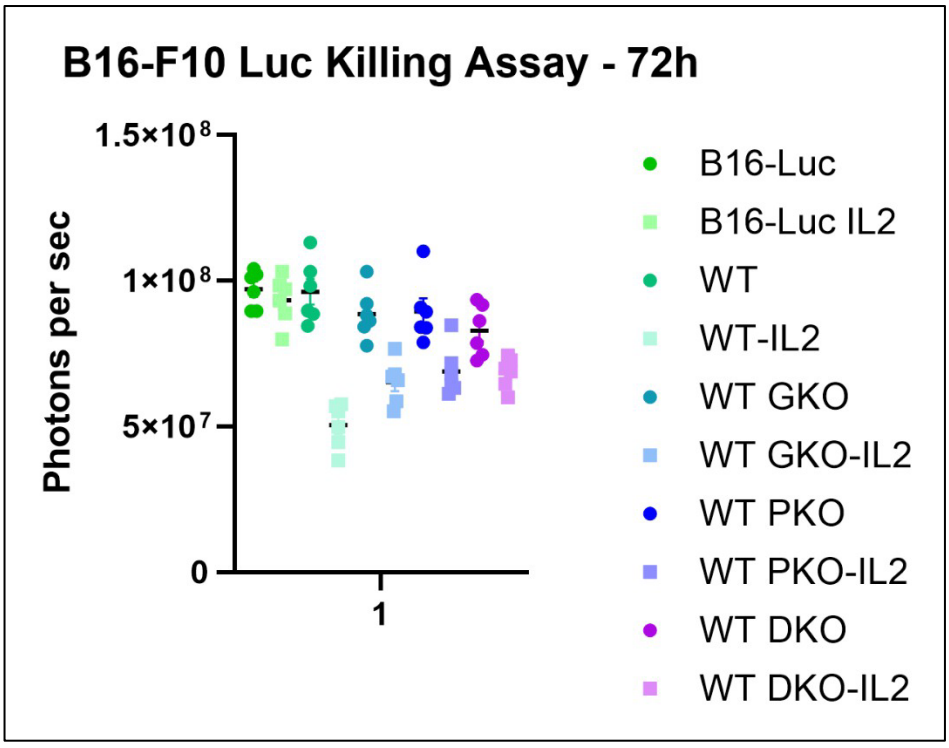
UNIVERSITY of MARYLAND
SCHOOL OF MEDICINE

SUMMER RESEARCH PROGRAMS
STUDENT RESEARCH FORUM

Hosted by
Office of Student Research

Thursday, July 24 & Friday, July 25, 2025
Southern Management Corporation (SMC)
Campus Center 621 West Lombard Street
Baltimore, MD 21201



Mikayla Wills, P1.07
Graph of the photons emitted from the tumor cells remaining in each vitro co-culture, including wild-type, gzmB knockout, perforin knockout, and gzmB-perforin double knockout. Attributed to the Cao lab

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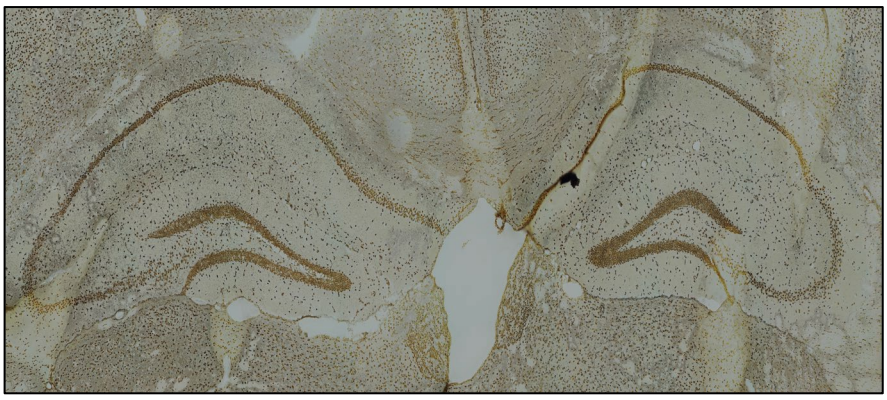

Tyler Wishard, O4.03
Bilateral hippocampal formations in a coronal section of mouse brain stained with NeuroSilver(TM) acquired using high-resolution microscopy at 20x magnification.

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Office of Student Research

Kathryn S. Robinett, M.D. Professor of Medicine
Associate Professor of Medicine
Associate Dean for Student Research and Education
Assistant Dean for Admissions

Gregory B. Carey, Ph.D.
Associate Professor of Microbiology and Immunology
Associate Dean for Biomedical and Health Profession Pathways and Workforce Development
Assistant Dean for Student Research and Education

Donald R. Matteson, Ph.D.
Associate Professor of Physiology
Assistant Dean for Student Research and Education

Fiama Romero, M.S.
Program Manager

Deja Williams
Program Coordinator

Acknowledgements

We would like to express our appreciation for all the summer research faculty mentors and staff who supported and guided the students in their research.

Our special thanks go to the Pass and Susel family for their continued support of student programs and enrichment.

We are grateful to all the graduate students, undergraduate students, postdoctoral fellows, faculty, and staff who generously gave of their time to assist during this event.

Event Volunteers

Olohitare Abaku, Undergraduate Student
Ariel Abraham, Undergraduate Student
Eleni Alvarez, Staff
Rosita Asawa, Graduate Student
Aditi Banerjee, Faculty
Cassidy Beck, Staff
Sasha Cardozo, Graduate Student
Laura Carreto-Binaghi, Postdoctoral Fellow
Aamna Cheema, Medical Student
Sid Dante, Faculty
Heather Ezelle, Faculty
Martin Flajnik, Faculty
Leah Han, Undergraduate Student
Andrew Hummer, Undergraduate Student
Margaret Kato, Undergraduate Student
Jaylyn King, Graduate Student
Alexander Laurenson, Graduate Student
Christina Manoto, Staff
Sherry McCammon, Staff
Amanda Nsubuga, Graduate Student
Neeta Rajbanshi, Faculty
Priyanka Ravi, Medical Student
Karen Scanlon, Faculty
Ciaran Skerry, Faculty
Luisa Valencia, Graduate Student
Michael Wagner, Graduate Student

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UM SOM Office of Student Research
Summer Research Programs
Student Research Forum

Thursday, July 24 and Friday, July 25, 2025

Southern Management Corporation (SMC) Campus Center

Attire: Business Casual

Day 1: Thursday, July 24

8:00 a.m. – 8:50 a.m.	***Breakfast and registration (2nd Floor Lounge, Elm Room A)***
9:00 a.m. – 9:30 a.m.	Opening Remarks **Gregory B. Carey, PhD** Associate Professor of Microbiology and Immunology Associate Dean for Biomedical and Health Profession Pathways and Workforce Development Assistant Dean for Student Research and Education Executive Director of Student Research and Community Outreach Remarks by Program Leaders **Bret A. Hassel, PhD** Professor of Microbiology and Immunology Assistant Director Training and Education Marlene and Stewart Greenebaum Comprehensive Cancer Center Director Nathan Schnaper Intern Program **Tonya J. Webb, PhD** Associate Professor of Microbiology and Immunology Assistant Director for Diversity, Equity and Inclusion Director, American Cancer Society (ACS) Internship for Mentored Projects Advancing Cancer Translation (IMPACT) Brief Procedures **Fiama Romero, MS** Program Manager, OSR **Deja Williams** Program Coordinator, OSR
9:30 a.m. – 9:35 a.m.	*Break*

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Day 1: Thursday, July 24 *(continued)*

9:35 a.m. – 11:45 a.m.	***Presentations***
9:35 a.m. – 10:35 a.m.	Oral Presentation Session 1 (Elm Room A) Oral Presentation Session 2 (Elm Room B) Oral Presentation Session 3 (Room 223)
10:35 a.m. – 10:45 a.m.	*Break*
10:45 a.m. – 11:45 a.m.	Poster Presentation Session 1 (Room 349)
11:45 a.m. – 1:20 p.m.	***Lunch and Keynote***
11:45 a.m. – 1:20 p.m.	Lunch (Elm Room A)
12:10 p.m. – 12:50 p.m.	Keynote Address (Elm Room A) **William Checkley, MD, PhD, ScM** Professor of Medicine, Pulmonary and Critical Care Joint Appointment in International Health, Bloomberg School of Public Health Founding Director, Center for Global Non-Communicable Disease Research and Training Division of Pulmonary and Critical Care Johns Hopkins University, Baltimore, MD **(click here to see bio)**
1:25 p.m. – 3:35 p.m.	***Presentations***
1:25 p.m. – 2:25 p.m.	Poster Presentation Session 2 (Room 349)
2:25 p.m. – 2:35 p.m.	*Break*
2:35 p.m. – 3:35 p.m.	Oral Presentation Session 4 (Elm Room A) Oral Presentation Session 5 (Elm Room B) Oral Presentation Session 6 (Room 223)

PHOTOGRAPHY AND VIDEOGRAPHY AT THIS EVENT

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Attire: Business Casual

Day 2: Friday, July 25

8:00 a.m. – 8:50 a.m.	***Breakfast and registration (2nd Floor Lounge, Elm Room A)***
9:00 a.m. – 12:20 p.m.	***Presentations***
9:00 a.m. – 10:00 a.m.	Poster Presentation Session 3 (Room 349)
10:00 a.m. – 10:10 a.m.	*Break*
10:10 a.m. – 11:10 a.m.	Oral Presentation Session 7 (Elm Room A) Oral Presentation Session 8 (Elm Room B) Oral Presentation Session 9 (Room 223)
11:10 a.m. – 11:20 a.m.	*Break*
11:20 a.m. – 12:20 p.m.	Poster Presentation Session 4 (Room 349)
12:25 p.m. – 12:30 p.m.	Closing Remarks (Room 349) **Gregory B. Carey, PhD and Fiama Romero, MS**

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Presentation Schedule (Ordered by Session and Presentation ID)

Session	ID	Last Name	First Name	Program	Title	Date/Time	Room	Mentor(s)
Oral 1	O1.01	Lawrence	Audrey	PRISM	Evaluation of Akt Signaling as a Critical Effector in Anti-N-Methyl-D-Aspartate Receptor Encephalitis	Thursday, 07/24 9:35-10:35 AM	Elm Rm. A	David Benavides
Oral 1	O1.02	Nguyen	Thach-Vu	PRISM	Low-Intensity Focused Ultrasound Attenuates Local Field Potential Ictal Activity and Spontaneous Seizures in a Rat Model of Temporal Lobe Epilepsy	Thursday, 07/24 9:35-10:35 AM	Elm Rm. A	Whitney Parker
Oral 1	O1.03	Nusraty	Sabrina	PRISM	Polysomnographic Predictors of Prefrontal Cortex Function in Children with Sleep Disordered Breathing	Thursday, 07/24 9:35-10:35 AM	Elm Rm. A	Amal Isaiah
Oral 1	O1.04	Yang	Sarah	PRISM	Predictors of Pulmonary Hypertension in Children with Obstructive Sleep Apnea and Down Syndrome	Thursday, 07/24 9:35-10:35 AM	Elm Rm. A	Amal Isaiah
Oral 1	O1.05	Pyo	Daniel	Radiation Oncology	Clinical Validation of the Role of Caveolin-1 in Radiation and Chemotherapy Resistance in Lung Cancer	Thursday, 07/24 9:35-10:35 AM	Elm Rm. A	Hem Shukla
Oral 1	O1.06	Jensen	Peter	PRISM	Social Determinants of Health in Pediatric Respiratory Disease	Thursday, 07/24 9:35-10:35 AM	Elm Rm. A	Siddhartha Dante
Oral 2	O2.02	Myers	Andrew	UM Scholars at SOM	Evaluating the role of macrophages in a Matched Model of Graft vs Host Disease (GVHD) - ocular and systemic GVHD.	Thursday, 07/24 9:35-10:35 AM	Elm Rm. B	Sarah Sunshine
Oral 2	O2.03	Addepalli	Anirudh	UM Scholars at SOM	Computer Vision Tool for Diagnosing Ocular Graft vs Host Disease	Thursday, 07/24 9:35-10:35 AM	Elm Rm. B	Sarah Sunshine
Oral 2	O2.04	Jamka	Emma	UM Scholars at SOM	Utilizing Tear Cytokine Changes to Determine the effect of Systemic Therapy on the eyes in patients with ocular GVHD: The Impact of JAK Inhibition	Thursday, 07/24 9:35-10:35 AM	Elm Rm. B	Sarah Sunshine
Oral 2	O2.05	Yang	Jack	PRISM	Evaluating Serum Tears as a Treatment for Ocular Graft vs. Host Disease after Hematopoietic Stem Cell Transplantation	Thursday, 07/24 9:35-10:35 AM	Elm Rm. B	Sarah Sunshine
Oral 2	O2.06	Kang	Andrew	PRISM	Measuring T-cell Expression in Ocular and Systemic Tissue in Response to Systemic and Topical JAK 1/2 Inhibitors in a Murine Model of Ocular Graft Versus Host Disease	Thursday, 07/24 9:35-10:35 AM	Elm Rm. B	Sarah Sunshine
Oral 3	O3.01	Abaku	Olohitare	UM Scholars at SOM	Mapping Health Inequities: Leveraging Geospatial Analysis to Refine Food Desert Identification and its Impact on Diabetes Outcomes	Thursday, 07/24 9:35-10:35 AM	Rm. 223	Rozalina McCoy
Oral 3	O3.02	Ge	Aaron	PRISM	Comparing Semantic and Numerical Representations in Clinical Risk Prediction	Thursday, 07/24 9:35-10:35 AM	Rm. 223	Bradley Maron
Oral 3	O3.03	Singh	Aditi	PRISM	Trends in Weight Management Therapies Among Patients with Obesity and Type 2 Diabetes	Thursday, 07/24 9:35-10:35 AM	Rm. 223	Rozalina McCoy
Oral 3	O3.04	Linus	Olivia	UM Scholars at SOM	Investigating the Impact of GLP-1 Receptor Agonists on Epigenetic Changes and Inflammatory Responses in Human Ileum and Gallbladder Mucosa	Thursday, 07/24 9:35-10:35 AM	Rm. 223	Rosangela Mezghanni
Oral 3	O3.05	Camara	Habib	MPower/MDH	Assessing, Scanning, and Curating: Enhancing Local Health Officer Orientation Through a National Environmental Scan	Thursday, 07/24 9:35-10:35 AM	Rm. 223	Vanessa Lamers
Oral 3	O3.06	Panshin	Maclean	MPower/MDH	Strengthening Maryland's Healthcare Workforce: A Comparative Analysis of Maryland Loan Repayment Program's Applicants and Awardees	Thursday, 07/24 9:35-10:35 AM	Rm. 223	Sara Seitz
Oral 4	O4.01	Chaudhuri	Swapno	UM Scholars at SOM	Cognitive Function and Cerebrovascular Reactivity in Persons with HIV	Thursday, 07/24 2:35-3:35 PM	Elm Rm. A	Linda Chang
Oral 4	O4.02	Snyder	Kirsten	PRISM	Characterizing Naive Expression of Lactate Dehydrogenase Isoforms A and B in a Cell-Type Specific Manner	Thursday, 07/24 2:35-3:35 PM	Elm Rm. A	Alexander Ksendzovsky
Oral 4	O4.03	Wishard	Tyler	PRISM	Investigation of Molecular and System-Level Brain Changes Following Temporal Lobe Contusion in the Development of Post-Traumatic Epilepsy	Thursday, 07/24 2:35-3:35 PM	Elm Rm. A	Alexander Ksendzovsky; Volodymyr Gerzanich; J. Marc Simard
Oral 4	O4.04	Cheema	Aamna	Guest	The ZIP Code Effect: ?Neighborhood Socioeconomics and Pre-Hospital Response in Status Epilepticus	Thursday, 07/24 2:35-3:35 PM	Elm Rm. A	Matthew Woodward
Oral 4	O4.05	Hummer	Andrew	UM Scholars at SOM	iPSC and iPSC-EVs Therapies for Peripheral Nerve Regeneration Following Sciatic Nerve Crush in Rats.	Thursday, 07/24 2:35-3:35 PM	Elm Rm. A	Xiaofeng Jia
Oral 4	O4.06	Han	Leah	UM Scholars at SOM	Brain Recovery after Cardiac Arrest treated with Metabolic Glycoengineered Stem Cell Therapy	Thursday, 07/24 2:35-3:35 PM	Elm Rm. A	Xiaofeng Jia
Oral 5	O5.01	Cheraghi	Nora	PRISM	Evaluating the Efficacy of T-Regulatory Cell Specific Interleukin-2 Nanoparticles for the Treatment of Ocular Graft versus Host Disease in a Mouse Model	Thursday, 07/24 2:35-3:35 PM	Elm Rm. B	Sarah Sunshine
Oral 5	O5.02	Menon	Riva	PRISM	Predicting visual acuity and glaucoma 10 years after unilateral congenital cataract surgery: Results from a Randomized, Multicenter Study	Thursday, 07/24 2:35-3:35 PM	Elm Rm. B	Janet Alexander
Oral 5	O5.03	Zhang	Justin	PRISM	Epidemiology and Risk Factors of Ocular Involvement in Graft-Versus-Host Disease After Allogeneic Hematopoietic Stem Cell Transplant	Thursday, 07/24 2:35-3:35 PM	Elm Rm. B	Sarah Sunshine
Oral 5	O5.04	Cabrera	Gabriela	M4I	The association between maternal mental health and child executive function among children who are HIV exposed and uninfected	Thursday, 07/24 2:35-3:35 PM	Elm Rm. B	Andrea Buchwald
Oral 5	O5.05	Meyer	Jonathon	UM Scholars at SOM	Evolution of the MHC: Characterization of Nonclassical MHC Class I Gene Emergence and Evolution Using a Shark Model	Thursday, 07/24 2:35-3:35 PM	Elm Rm. B	Yuko Ota

Presentation Schedule (Ordered by Session and Presentation ID)

Session	ID	Last Name	First Name	Program	Title	Date/Time	Room	Mentor(s)
Oral 5	O5.06	McLuckey	Robert	M4I	Testing AI-Powered Camera Effectiveness to Study Red Zone Precautions in Preventing the Spread of Antibiotic-Resistant Bacteria	Thursday, 07/24 2:35-3:35 PM	Elm Rm. B	Anthony Harris
Oral 6	O6.01	Noyes	Charlotte	UM Scholars at SOM	CAR-Tregs: Could they be the key to solving xenotransplant rejection?	Thursday, 07/24 2:35-3:35 PM	Rm. 223	Nevil Singh
Oral 6	O6.02	Siddiqui	Mustafa	PRISM	Evaluation of Platelet Adhesion and Hemodynamic Influences on ECMO Thrombosis: A Comparative Study of Biomaterial Surfaces	Thursday, 07/24 2:35-3:35 PM	Rm. 223	Zhongjun Wu
Oral 6	O6.03	Oluwafemi	AyoOluwakiitan	PRISM	The effect of Mechanical Circulatory Devices on Blood Components	Thursday, 07/24 2:35-3:35 PM	Rm. 223	Zhongjun Jon Wu
Oral 6	O6.04	Nur	Mubassira	MPower/MDH	Examining Sociodemographic Characteristics by Place of Death for Marylanders Who Died of Heart Disease or COVID-19 From 2020 to 2023	Thursday, 07/24 2:35-3:35 PM	Rm. 223	Monique Wilson
Oral 6	O6.05	Whiteleather	Julia	PRISM	Factors Influencing Clinical Decision-Making for Pulmonary Vasodilation Therapies in Premature Neonates with Chronic Lung Disease	Thursday, 07/24 2:35-3:35 PM	Rm. 223	Alicia Chaves
Oral 6	O6.06	Mansoor	Shaiza	PRISM	Deep Vein Thrombosis and Thrombus Resolution	Thursday, 07/24 2:35-3:35 PM	Rm. 223	Brajesh Lal
Oral 7	O7.01	Lindley	Abel	PRISM	SARM1 Drives Schwann Cell Dysfunction and Axonal Degeneration following Peripheral Nerve Injury	Friday, 07/25 10:10-11:10 AM	Elm Rm. A	Xiaofeng Jia
Oral 7	O7.02	Rinaldi	Annabella	UM Scholars at SOM	Characterizing Symptoms of Trichomoniasis	Friday, 07/25 10:10-11:10 AM	Elm Rm. A	Gentry Wilkerson
Oral 7	O7.03	Tieu	Kenneth	PRISM	Early Preeclampsia Risk Assessment: A Window into Congenital Heart Defect Development	Friday, 07/25 10:10-11:10 AM	Elm Rm. A	Shifa Turan
Oral 7	O7.04	Jacobs	Lauren	PRISM	Patient Satisfaction with IUD Insertion with IUD Pain Management Option Checklist	Friday, 07/25 10:10-11:10 AM	Elm Rm. A	Jessica Lee
Oral 7	O7.05	Pugazhendhi	Ashwini	PRISM	Determination of the Rate and Accuracy of Hypertensive Disorder Diagnosis in Pregnant People with Opioid Use Disorder	Friday, 07/25 10:10-11:10 AM	Elm Rm. A	Katrina Mark
Oral 8	O8.01	Berhaneyessus	Lina	UM Scholars at SOM	Investigating Immune Markers Correlated with Intestinal Permeability	Friday, 07/25 10:10-11:10 AM	Elm Rm. B	Bing Ma
Oral 8	O8.02	Kato	Margaret	UM Scholars at SOM	Genomic Specialization of Bifidobacterium for Human Milk Oligosaccharide Utilization in Preterm Infant Gut Maturation	Friday, 07/25 10:10-11:10 AM	Elm Rm. B	Bing Ma
Oral 8	O8.03	Yang	Angela	M4I	Using STING agonists to reshape tumor environment in hepatocellular carcinoma, to improve response of anti-PD1 immunotherapy	Friday, 07/25 10:10-11:10 AM	Elm Rm. B	Daniel Shu
Oral 8	O8.04	Przygocki	Tyler	PRISM	Primary and Revision Hip Arthroscopy Patients Report Similar Outcomes 2-years After Surgery	Friday, 07/25 10:10-11:10 AM	Elm Rm. B	Sean Meredith
Oral 8	O8.05	Diep	David	PRISM	Mobile is the Goal: Validating Functional Outcomes and Return to Baseline Activity With Apple Health Metrics in Patients Undergoing ACL Reconstruction	Friday, 07/25 10:10-11:10 AM	Elm Rm. B	Jonathan Packer
Oral 8	O8.06	Polsky	Daniel	PRISM	Private Vehicle vs. Ambulance for Pediatric Orthopaedic Transfers	Friday, 07/25 10:10-11:10 AM	Elm Rm. B	Joshua Abzug
Oral 9	O9.01	Wilmer	Joshua	PRISM	Investigating the Functional Role of WNT5A in Prurigo Nodularis	Friday, 07/25 10:10-11:10 AM	Rm. 223	Shawn Kwatra
Oral 9	O9.02	Forsberg	Alisa	PRISM	Expression of Growth Differentiation Factor 6 (GDF6) in Acral Melanoma.	Friday, 07/25 10:10-11:10 AM	Rm. 223	Thomas Hornyak
Oral 9	O9.03	Huang	Christopher	PRISM	Comparison of Outcomes in Transperineal vs. Transrectal Prostate Biopsy in the VA Patient Population	Friday, 07/25 10:10-11:10 AM	Rm. 223	Mohummad Siddiqui
Oral 9	O9.04	Ravi	Priyanka	PRISM	An Analysis of Socioeconomic Deprivation and Cancer Trial Enrollment at the University of Maryland's Greenebaum Comprehensive Cancer Center: A Retrospective Cohort Study	Friday, 07/25 10:10-11:10 AM	Rm. 223	Benjamin Powers
Oral 9	O9.05	Jaranson	Renee	UM Scholars at SOM	Ultrasound-mediated Drug Activation for the Treatment of Infiltrating Gliomas	Friday, 07/25 10:10-11:10 AM	Rm. 223	Pavlos Anastasiadis
Oral 9	O9.06	Rajabi Abhari	Delara	PRISM/Rad-Onc	Practical Clinical Interventions to Help Improve Cancer Care for Patients Who Experience Food Insecurity	Friday, 07/25 10:10-11:10 AM	Rm. 223	Melissa Vyfhuis
Poster 1	P1.01	Olibris	Gabrielle	ACS-IMPACT	Targeting Medulloblastoma Cells with Novel Therapeutic Compound VNPP-433.3β.	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Aditi Banerjee
Poster 1	P1.02	Mohammed	Maawiah	NSIP	Hijacking the Endogenous Repair Mechanisms of DNA to Cure Genetic Disorders	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Alexandros Poulopoulos
Poster 1	P1.03	Chukwura	Chukuamaka	ACS-IMPACT	Investigating the antileukemic mechanism of action of ART838 in acute myeloid leukemia (AML)	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Curt Civin
Poster 1	P1.04	Sandagorda	Karem	ACS-IMPACT	Investigating Cytokine Influence on Tumor Dynamics in Ovarian Cancer Cell Models	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Tonya Webb
Poster 1	P1.05	Brown	Nicole	ACS-IMPACT	Rewiring T Cell Signaling To Make a Better CAR-T Cell	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Nevil Singh

Presentation Schedule (Ordered by Session and Presentation ID)

Session	ID	Last Name	First Name	Program	Title	Date/Time	Room	Mentor(s)
Poster 1	P1.06	Hountangni	Grace	ACS-IMPACT	The Cellular Response of Bladder Cancer Cell Lines to Gemcitabine	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Minhaj Siddiqui
Poster 1	P1.07	Shah	Ronit	NSIP	Transcriptional Profiling of Cancer-Related Fatigue	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Amber Kleckner
Poster 1	P1.08	Wills	Mikayla	ACS-IMPACT	In Vitro Study of Granzyme B-dependent Antitumor Immune Response	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Xuefang Cao
Poster 1	P1.09	Reid	Tayah	ACS-IMPACT	The Impact of Financial stress on overall Survival in Acute Myeloid Leukemia	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Sandrine Niyongere
Poster 1	P1.10	Stratton	Elliot	NSIP	Building Molecular Tools to Identify Protein Binding Partners of AUF1 Involved in Post-Transcriptional Regulation	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Gerald Wilson
Poster 1	P1.11	Shetty	Amogh	NSIP	Photodegradation Shifts Indocyanine Green (ICG) Aggregation, Modulating Fluorescence and Thermal Response	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Vikas Kundra
Poster 1	P1.12	Radov	Michelle	NSIP	Identifying Antigen-Driven Tolerance Signatures in Tumor-Reactive T cells	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Nevil Singh
Poster 1	P1.13	Chicas	Kristal	NSIP	Investigating the Impact of JAK-1 Inhibitors on Tumorigenesis Through Regulation of JAK-1 Suppressed Genes	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Darren Perkins
Poster 1	P1.14	Savage	Lauren	NSIP	Impact of Methotrexate on Motor Learning and Motor Function	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Victoria Marchese
Poster 1	P1.15	Li	Anna	M4I	A Comparison of Various MRI Acquisition Methods for Visualization of MRI Findings Critical to the New Multiple Sclerosis Diagnostic Criteria	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Daniel Harrison
Poster 1	P1.16	Idsassi	Jehan	ACS-IMPACT	Characterizing the Immunosuppressive Effect of Mesenchymal Stromal Cells on Peripheral Blood Mononuclear Cells	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Magali Fontaine
Poster 1	P1.17	Tobin-Xet	Gabriel	M4I	The Impact of Structured Provider Education on HIV Pre-Exposure Prophylaxis Delivery in a Pediatric Primary Care Office	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Matthew Grant
Poster 1	P1.18	Tan	Angela	NSIP	SIX Family Proteins Drive Human Erythropoiesis	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Curt Civin
Poster 1	P1.19	Wang	Phoenix	NSIP	Physiologically Relevant Ca2+ and Mg2+ Concentrations to Regulate the BP2 Region of the STRA6-Calmodulin Complex	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Kristen Varney
Poster 1	P1.20	Abraham	Ariel	UM Scholars at SOM	Low-Intensity Focused Ultrasound: A Non-Invasive Treatment Alternative for Opioid Use Disorder	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Linda Chang
Poster 1	P1.21	Frederick	Kirie	ACS-IMPACT	Quality assessment in Orthopedic randomized controlled trials: Retracted vs non-retracted studies	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Michelle Ghert
Poster 1	P1.22	Mbaya	Benick	NSIP	Assessing the Effects of Exercise During Chemotherapy on Physical Function Among Women with Breast Cancer	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Ian Kleckner
Poster 1	P1.23	Peng	Xiwei	PRISM	Impact of Intraoperative Autologous Blood Donation in Heart Transplantation on Hemodynamics and Transfusion Requirements: Interim Analysis	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Brittney Williams
Poster 1	P1.24	Bardi	Georgia	UM Scholars at SON	The New Imperative: Measuring Primary Palliative Care Education for Undergraduate Nursing Students	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Hannah Murphy-Buc
Poster 1	P1.25	Mata	Alejandra	UM Scholars at SON	From Conference Presentation to Journal Publication: Restorative Practices to Strengthen Student-Faculty Relationships	Thursday, 07/24 10:45-11:45 AM	Rm. 349	Hannah Murpyh Buc
Poster 2	P2.01	Fenn	Jeffrey	M4I	Emerging Clinical Patterns Among Young Adults Living With HIV	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Patrick Ryscavage
Poster 2	P2.02	Sarpong	Maame	SUMMIR	Bordetella pertussis infection downregulates the angiotensin system in the murine right ventricle	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Karen Scanlon
Poster 2	P2.03	Bonnet	Paige	NSIP	Role of OAS1 rs1131454 SNP in Modulating Enterovirus Replication	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Saumen Sarkar
Poster 2	P2.04	Baffoe-Bonnie	Janice	SUMMIR	Does Integrin β4 Function as a Host Receptor for Fungi	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Vincent Bruno
Poster 2	P2.05	Murr	Lucy	SUMMIR	The Role of Integrin b4 in Candida albicans Adhesion to Human Cells	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Vincent Bruno
Poster 2	P2.06	Chopra	Pratham	UM Scholars at SOM	MMP-9 Enhanced Mesenchymal Stem Cells Improve Islet Xenograft Survival in Diabetic Mice	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Raphael Meier
Poster 2	P2.07	Johnson	Najah	SUMMIR	Identifying Immune Cells in Nurse Shark Tissues.	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Helen Dooley
Poster 2	P2.08	Naranjo	Joshua	Bridges to Doctorate	Developing an agent based model of the pancreatic ductal adenocarcinoma microenvironment	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Daniel Bergman; Elana Fertig; Jeanette Johnson
Poster 2	P2.09	Van't Hof	Vivien	NSIP	Effects of ERK1/2 Modulation on Tristetraprolin Expression in A375 Melanoma Cells	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Paul Shapiro

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Poster 2	P2.10	Berroya	Arryn Joeina	NSIP	The role of nuclear receptor NR1D1 in Tumor Response to Anti-cancer Therapeutics	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Min Yu
Poster 2	P2.11	Parakhoodi	Andrew	NSIP	Investigating the Role of Drug Inhibition of hnRNP A18 In Suppressing Hypoxia-Induced Glioblastoma Proliferation	Thursday, 07/24 1:25-2:25 PM	Rm. 349	France Carrier
Poster 2	P2.12	Acle	Grace	NSIP	Fluid Shear Stress-Mediated Activation of Ras in Primary and Metastatic Brain Cancer	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Pavlos Anastasiadis
Poster 2	P2.13	Ravipati	Laasya	NSIP	A Novel Construct that Enables Simultaneous Tracking of Cell Cycle Progression and Calcium in Glioblastoma	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Yajie Liang
Poster 2	P2.14	Russell	Lynijah	NSIP	Modeling Pancreatic Cancer and Precancer Microenvironments Using PhysiCell Agent-Based Simulations	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Daniel Bergman
Poster 2	P2.15	Obi	Justin	ACS-IMPACT	A Novel PD-1 and LAG-3 targeting bispecific molecule in a murine glioblastoma model	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Pavlos Anastasiadis
Poster 2	P2.16	Kerner	Jackson	UM Scholars at SON	Qualitative Analysis of Cancer Survivors' Experience in a Nutritional Clinical Trial	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Amber Kleckner
Poster 2	P2.17	Seluchins	Chloe	NSIP	Evaluating the in vitro Radiosensitizing Effects of Oxaliplatin in Comparison to the Current Standard of Care Treatment: Temozolomide	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Ananya Elati
Poster 2	P2.18	Kolodgie	Nina	Radiation Oncology	Pulmonary Complications Following Lateral Beam Total Body Irradiation: A Retrospective Analysis of Dose-Dependent Outcomes	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Zaker Rana
Poster 2	P2.19	Bredar	Sophia	NSIP	ROR1 CAR T-Cells As a Novel Treatment for Prostate Cancer	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Djordje Atanackovic
Poster 2	P2.20	Gelin	Ashley	Bridges to Doctorate	Engineered CAR T-cells targeting ROR1 for the treatment of lung cancer	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Djorde Atanackovic
Poster 2	P2.21	Terrillion Arroyave Ward	Keira Davis Imani	Summer RISE Mont. County Public Schools); Goucher College	Exploring the Role of Race in Adolescent Dietary Choices: United States High School Students	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Diane Marie St. George
Poster 2	P2.22	Westlake	Cameron	NSIP	Investigating the Functional Role of UBASH3B Isoforms in HPV-Negative Head and Neck Squamous Cell Carcinoma	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Daria Gaykalova
Poster 2	P2.23	Qiao	Eva	NSIP	Liposomal HDAC8 inhibitor formulation for neuroblastoma treatment	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Ryan Pearson
Poster 2	P2.24	Trageser	Karl	NSIP	The Effect of Combining MEK Inhibitors with Commonly Used Chemotherapies in KRAS Mutated Pancreas Cancer	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Rena Lapidus
Poster 2	P2.25	Rawlett	Grace	NSIP	Caveolin-1 Upregulation Fuels EGFR-Mediated Resistance to Radiation and Osimertinib in Lung Cancer	Thursday, 07/24 1:25-2:25 PM	Rm. 349	Hem Shukla
Poster 3	P3.01	Gadagkar	Ruchika	PRISM	What Happens to Pain?: An Observational, Longitudinal Study in Patients with Aneurysmal Subarachnoid Hemorrhage Treated in Neurocritical Care Units (WHOL-PAIN)	Friday, 07/25 9:00-10:00 AM	Rm. 349	Nicholas Morris
Poster 3	P3.02	Agoh	Ozioma	UM Scholars at SON	Investigating the Role of Expectations in a Virtual Reality Based Intervention in people with Temporomandibular Disorder	Friday, 07/25 9:00-10:00 AM	Rm. 349	Luana Colloca
Poster 3	P3.03	Ni	Rachel	UM Scholars at SON	Efficacy of Exercise on Improving Cardiovascular Function in Breast Cancer Patients Undergoing Chemotherapy	Friday, 07/25 9:00-10:00 AM	Rm. 349	Ian Kleckner
Poster 3	P3.04	Waller	Daphney	UM Scholars at SON	Ongoing Study Investigating Virtual Reality as an At-Home Intervention for Temporomandibular Disorder	Friday, 07/25 9:00-10:00 AM	Rm. 349	Nandini Raghuraman
Poster 3	P3.05	Morris	Kayla	UM Scholars at SON	Ongoing Investigation of The Impact of Virtual Reality on Orofacial Pain Intensity and Interference among Participants with Temporomandibular Disorder	Friday, 07/25 9:00-10:00 AM	Rm. 349	Luana Colloca
Poster 3	P3.06	Parrish	Madison	SUMMIR	Troubleshooting Alginate Microcapsules for Oral Mice Trial	Friday, 07/25 9:00-10:00 AM	Rm. 349	Scott Baliban
Poster 3	P3.07	Kumar	Rishi	SUMMIR	mRNA and miRNA Dual Omics Profiling of Ischemia-Reperfusion Injury in Liver Allografts	Friday, 07/25 9:00-10:00 AM	Rm. 349	Valeria Mas
Poster 3	P3.08	Hassan	Mariam	SUMMIR	Investigating the Regulation and Expression of Colonization Factor CS1 in Enterotoxigenic E. coli (ETEC) Clinical Isolates	Friday, 07/25 9:00-10:00 AM	Rm. 349	Elieen Barry
Poster 3	P3.09	Ford	Savannah	SUMMIR	Regulation of the Ferritin-like Protein PA4880 in Pseudomonas fluorescens Under Oxidative Stress	Friday, 07/25 9:00-10:00 AM	Rm. 349	Amanda Oglesby
Poster 3	P3.10	Bazzi	Kinan	SUMMIR	A Study of Rickettsial Lipase (R Lip.) Effector Protein in Host Pathogenesis	Friday, 07/25 9:00-10:00 AM	Rm. 349	Oliver Voss
Poster 3	P3.11	Yussuf	Samiatu	SUMMIR	Epitranscriptomics: Developing a Rapid Diagnostic to Identify Single-Based CRISPR-Induced Deletions in an Epitranscriptomic Writer in Drosophila melanogaster	Friday, 07/25 9:00-10:00 AM	Rm. 349	Robin Bromley
Poster 3	P3.12	Davis	Alisa	SUMMIR	High-Dimensional Flow Cytometry Analysis on CAR-T Cells for Cancer Patients	Friday, 07/25 9:00-10:00 AM	Rm. 349	Xiaoxuan Fan

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Poster 3	P3.13	Aime	Nimah	SUMMIR	Peri-hippocampal Mast Cells Influence Oligodendrocyte Development During the Early Postnatal Period	Friday, 07/25 9:00-10:00 AM	Rm. 349	Matthew Bruce
Poster 3	P3.14	Gonzalez	Rafael	SUMMIR	Identification of Genomic Mechanisms of Multi-Drug Resistance in the Fungal Pathogen Candida Auris	Friday, 07/25 9:00-10:00 AM	Rm. 349	Mary Ann Jabra-Rizk
Poster 3	P3.15	Carlos-Figueroa	Jeremi	SUMMIR	Dynamics of "Double Duty" Dendritic Cells (XL Cells) during a Humoral Immune Response in the Amphibian Xenopus	Friday, 07/25 9:00-10:00 AM	Rm. 349	Martin Flajnik
Poster 3	P3.16	Quezada	Yaneysi	UM Scholars at SOM	Gene Expression Differences in Cerebral vs. Asymptomatic Malaria in Malawian Children	Friday, 07/25 9:00-10:00 AM	Rm. 349	Mark Travassos
Poster 3	P3.17	sanders	Princaya	SUMMIR	Assessing EV-D entry following ATG-14 knockdown	Friday, 07/25 9:00-10:00 AM	Rm. 349	William Jackson
Poster 3	P3.18	Weir	Sydney	SUMMIR	Evaluation and selection of optimal antigen expression cassettes to maximize the immunogenicity of non-replicating adenoviral vectored vaccines.	Friday, 07/25 9:00-10:00 AM	Rm. 349	Aisha Souquette
Poster 3	P3.19	Huserova	Zuzana	UM Scholars at SON	Impact of Social Discrepancy on Placebo Hypoalgesia: Preliminary Analyses	Friday, 07/25 9:00-10:00 AM	Rm. 349	Luana Colloca
Poster 3	P3.20	Jordan	Olivia	PRISM/Rad-Onc	A Comparative Analysis of Decipher Genomic Classifier and Artera Multi-Modal AI for Risk Stratification and Treatment Optimization in Non-Metastatic Prostate Cancer	Friday, 07/25 9:00-10:00 AM	Rm. 349	Phuoc Tran
Poster 3	P3.21	White	Cynthia	CATALYST	Continuing the CATALYST Program: Teacher Researchers Connecting Cancer Research to the Classroom	Friday, 07/25 9:00-10:00 AM	Rm. 349	Bret Hassel
Poster 3	P3.22	Shaw Ahenda	Brittney	CATALYST	Bringing Cancer Research to the Classroom: a novel immunotherapeutic approach for Ovarian Cancer as foundational material for curriculum development	Friday, 07/25 9:00-10:00 AM	Rm. 349	Tonya Webb
Poster 3	P3.23	Umer	Hooria	UM Scholars at SON	Randomized Control Trial of Flange Fitting for NICU Pumping Parents to Explore Improved Milk Production and Satisfaction	Friday, 07/25 9:00-10:00 AM	Rm. 349	Rachel Breman
Poster 3	P3.24	Chander	Megha	UM Scholars at SON	Ongoing Study; Virtual Reality as a Home-Based Pain and Wellness Intervention in Cancer Survivors	Friday, 07/25 9:00-10:00 AM	Rm. 349	Luana Colloca
Poster 3	P3.25	Arroyo	Ayanna	UM Scholars at SON	Effects of Immersive Virtual Reality on Experimental and Clinical Pain in Patients with Temporomandibular Disorder (TMD)	Friday, 07/25 9:00-10:00 AM	Rm. 349	Luana Colloca
Poster 4	P4.01	Kim	Haley	UM Scholars at SOM	Investigating Differential Gene Expression in Two Severe Malaria Syndromes in Mali	Friday, 07/25 11:20-12:20 PM	Rm. 349	Mark Travassos
Poster 4	P4.02	Bristow	Paige	M4I	Time to Patent Bloodstream Infection for Males Versus Females in Controlled Human Malaria Infection at the University of Maryland	Friday, 07/25 11:20-12:20 PM	Rm. 349	Matthew Laurens
Poster 4	P4.03	Mason	Jack	M4I	Identifying Parasite Targets of Whole-Organism Vaccination through Whole Genome Sieve Analysis, Using Plasmodium falciparum Isolates from Trials of Sporozoite-based Vaccines	Friday, 07/25 11:20-12:20 PM	Rm. 349	Joana Carneiro da Silva
Poster 4	P4.04	Robinson	Mary Mae	M4I	Cross-Reactivity among Plasmodium falciparum Strains	Friday, 07/25 11:20-12:20 PM	Rm. 349	Andrea Berry
Poster 4	P4.05	Heath	Maxwell	SUMMIR	Contribution of Antigen-Specific and Bystander T Cell Responses to Disease Tolerance After a Malaria Infection	Friday, 07/25 11:20-12:20 PM	Rm. 349	Nevil Singh
Poster 4	P4.06	Fitzgerald	Allison	SUMMIR	Shifting immune profile of clinical pertussis isolates	Friday, 07/25 11:20-12:20 PM	Rm. 349	Ciaran Skerry
Poster 4	P4.07	Reddy	Alyssa	SUMMIR	Impact Of mtDNA Thresholds on Single Cell RNA-seq Analysis Outcomes in Bordatella Pertussis	Friday, 07/25 11:20-12:20 PM	Rm. 349	Ciaran Skerry
Poster 4	P4.08	Lee	Christina	UM Scholars at SON	Mapping Mindfulness to Reduce Psycho-social Suffering for Adolescents and Young Adults with Cancer	Friday, 07/25 11:20-12:20 PM	Rm. 349	Kim Mooney-Doyle
Poster 4	P4.09	Li	Abigail	M4I	Estrogen Modulation of Inflammatory Cytokine Responses in 3D Human Trophoblast Organoids	Friday, 07/25 11:20-12:20 PM	Rm. 349	Jun Lei
Poster 4	P4.10	Wegner	Claire	PRISM	Understanding Disparities in Prenatal Diagnosis of Congenital Heart Disease	Friday, 07/25 11:20-12:20 PM	Rm. 349	Alicia Chaves
Poster 4	P4.11	Emamian	Nikki	PRISM	Retrospective Chart Review of Infant & Mother Dyads seen in Special Parent and Infant Care and Enrichment (SPICE) Clinic	Friday, 07/25 11:20-12:20 PM	Rm. 349	Matthew Grant
Poster 4	P4.12	Yang	Ethan	PRISM	Evaluating Post-operative Pain Management with NSAIDs in Pediatric Orthopedic Patients	Friday, 07/25 11:20-12:20 PM	Rm. 349	Joshua Abzug
Poster 4	P4.13	Li	Rebecca	PRISM	Investigating Use of IV Lidocaine in Perioperative Pain Control following Colorectal Surgery	Friday, 07/25 11:20-12:20 PM	Rm. 349	Megan Anders
Poster 4	P4.14	Colliver	Lauren	PRISM	An Observational Study of Physiologic Changes During Prolonged Breath Holds in Bronchoscopic Lung Biopsies	Friday, 07/25 11:20-12:20 PM	Rm. 349	Megan Anders
Poster 4	P4.15	Bosworth	Eugene	PRISM	Investigating the Relationship Between Cortical Inhibition and Cognitive Control in Schizophrenia Using TMS-Measured SICI and the Stop-Signal Task	Friday, 07/25 11:20-12:20 PM	Rm. 349	Stephanie Hare
Poster 4	P4.16	Kim	Yehyun (Abby)	PRISM	Evaluating the Role of Conserved miRNAs Across Species in the Optic Nerve Laminar Region (ONLR) that Promote Retinal Ganglion Cell Survival and Regrowth	Friday, 07/25 11:20-12:20 PM	Rm. 349	Steven Bernstein

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Poster 4	P4.17	Pandey	Meghna	PRISM	Cortical interneuron development in Polyhydramnios, Megalencephaly, and Symptomatic Epilepsy	Friday, 07/25 11:20-12:20 PM	Rm. 349	Whitney Parker
Poster 4	P4.18	Fortune Hernandez	Nicole	UM Scholars at UMCP	The Impact of Depression on Antiretroviral Therapy Adherence in People Living with HIV: The Moderating Role of PTSD	Friday, 07/25 11:20-12:20 PM	Rm. 349	Abigail Hines
Poster 4	P4.19	Veloso	Isabel	M4I	The Continuum of Care in Hospitalized Patients with Opioid or Stimulant Use Disorder and Infectious Complications of Drug Use – Treatment as Usual, Addiction/ID Integrated Clinic (CHOICE-STAR Study) & Effects on Patient Antibiotic Completion and Infection Resolution	Friday, 07/25 11:20-12:20 PM	Rm. 349	Sarah Kattakuzhy
Poster 4	P4.20	Chowdhury	Urvi	MPower/MDH	Impact of Health Equity Notes on Maryland Department of Health's Policy Positions in the 2025 Maryland General Assembly Session	Friday, 07/25 11:20-12:20 PM	Rm. 349	Meghan Lynch
Poster 4	P4.21	Large	Madeline	CATALYST	The CATALYST Program: Teacher Researchers Bringing Biomedical and Cancer Research to the Classroom	Friday, 07/25 11:20-12:20 PM	Rm. 349	Saumen Sarkar
Poster 4	P4.22	Johnson	Lakesha	Guest	Tracking the Pulse of Survivorship: HRV as a Public Health Indicator of Physiological Stress	Friday, 07/25 11:20-12:20 PM	Rm. 349	Amber Kleckner
Poster 4	P4.23	Meshesha	Gabriella	Guest	Congenital CMV infection among infants born to mothers with and without HIV infection in Malawi.	Friday, 07/25 11:20-12:20 PM	Rm. 349	Miriam Laufer
Poster 4	P4.24	Meher	Zumar	Guest	Gene Set Analysis Identifies Immune Dysregulation Signatures of Prostate Cancer Recurrence	Friday, 07/25 11:20-12:20 PM	Rm. 349	Arif Hussain
Poster 4	P4.25	Lowe	Katie	Guest	The Localization of Granzyme B in Corneal Tissue of Ocular Graft versus Host Disease Murine Models and its Significance on the Pathological Pathway	Friday, 07/25 11:20-12:20 PM	Rm. 349	Sarah Sunshine
Poster 4	P4.26	Mileto Vize	Martina	CATALYST	Bringing Research to the Classroom: Targeting EGFR-Mediated Resistance to Treatment in Non-Small Cell Lung Cancer	Friday, 07/25 11:20-12:20 PM	Rm. 349	Hem Shukla

O1.01 Evaluation of Akt signaling as a critical effector in anti-N-methyl-D-aspartate receptor encephalitis

Presenter: Audrey Lawrence[1]
Mentor: David R. Benavides, MD, PhD[1]
[1]Department of Neurology, University of Maryland School of Medicine, Baltimore, MD

Autoimmune encephalitis (AIE), a rapidly progressive and potentially fatal neurologic condition, remains poorly understood at a molecular level. The development of novel therapeutics necessitates a deeper understanding of disease pathogenesis. The current prevailing theory of the pathogenesis of anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis, the most common subtype of AIE, supports antibody-mediated NMDAR crosslinking and downregulation. However, this theory fails to explain the full spectrum of clinical phenotypes observed in patients with pathogenic anti-NMDAR antibodies. To explore further, our lab is interrogating a novel molecular mechanism of disease that hypothesizes that synaptic deficits in the disease are due to altered activity of critical protein kinases within dendritic spines. Our preliminary data suggest that protein kinases are altered in primary neurons following anti-NMDAR antibody exposure, particularly Akt signaling. Here, we sought to further elucidate the effect of pathogenic anti-NMDAR encephalitis antibodies in primary neurons on Akt signaling, a serine/threonine protein kinase pathway that promotes survival and growth. Using subcellular fractionation and Western blotting, we assayed Akt abundance in cytosolic fractions of primary neurons following anti-NMDAR and control antibody exposure, as well as the level of regulatory phosphorylation sites at pThr308 and pSer473. We found dynamic, time-dependent, regulation of Akt phosphorylation across treatment conditions. Together, these data support the interpretation that anti-NMDAR antibody exposure regulates Akt signaling in primary neurons. Future studies will explore the dependence of Akt signaling pathway as a critical effector of downstream effects of anti-NMDAR antibodies. These studies will provide insights into the advent of future novel therapeutics in AIE.

This work was supported by NIH K08NS114039-01 (D.R.B.) and the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O1.02 Low-Intensity Focused Ultrasound Attenuates Local Field Potential Ictal Activity and Spontaneous Seizures in a Rat Model of Temporal Lobe Epilepsy

Presenter: Thach-Vu Nguyen[1]
Mentor: Whitney Parker MD, PhD[1]
Other Co-Authors: Sandesh Kamdi, PhD[1,2]; Reana Young-Morrison, BS[1,3]; Alexandra Seas, BS[1]; David Kolb, BS[1,2]; Adarsha Malla, PhD[1]; Iness Gildish, MS[3]; Tina Wang, MS[1]; Marianna Baybis, MS[1,2]; Joseph Cheer, PhD[3]; Pavlos Anastasiadis PhD[1]
[1]Department of Neurosurgery, University of Maryland School of Medicine, Baltimore, MD
[2]Department of Neurology, University of Maryland School of Medicine, Baltimore, MD
[3]Department of Neurobiology, University of Maryland School of Medicine, Baltimore, MD

Temporal lobe epilepsy (TLE) is a post-traumatic epilepsy affecting 50 million people globally, and 20-30% of patients suffer from treatment refractory seizures. Understanding the mechanism of epileptogenesis, the development of a seizure network following traumatic brain injury, remains an important research goal. The latent period following a traumatic brain insult may be a critical window for intervention to disrupt the development of an epileptic network and prevent spontaneous seizures from developing. Low intensity focused ultrasound (LOFU), a minimally invasive alternative, has shown promise in reducing spontaneous seizures when delivered in the post-latent period of epileptogenesis. However, the therapeutic potential of LOFU in disrupting epileptogenesis when delivered during the latent period has yet to be investigated. In the present study we utilized a well-established rat model of TLE using a unilateral intrahippocampal injection of the excitotoxin kainic acid. After a period of status epilepticus, a subset of animals received LOFU treatment to the site of insult. We observed that animals receiving LOFU intervention demonstrated reduced stereotyped seizure behavior, including forelimb clonus and rearing. Animals were also implanted with a microwire array after kainic acid injection and LOFU intervention to enable live local field potential (LFP) recordings. Preliminarily, LOFU treatment attenuated abnormal electrophysiological features of epilepsy including ictal micro-discharges at the LFP level. Immunostaining of hippocampal subregions has shown a decrease of excitatory glutamatergic neurotransmission as well as mossy fiber sprouting in animals receiving LOFU intervention. These results suggest that LOFU has potential in therapeutic neuromodulation for the disruption of epileptogenesis.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O1.03 Polysomnographic Predictors of Prefrontal Cortex Function in Children with Sleep Disordered Breathing

Presenter: Sabrina Nusraty[1]
Mentor: Amal Isaiah, MD, PhD[1]
[1]Department of Otorhinolaryngology-Head & Neck Surgery, University of Maryland School of Medicine, Baltimore, MD

Purpose: Sleep disordered breathing (SDB), characterized by snoring and sleep disruption, affects one in ten children. Adverse outcomes of SDB such as poor classroom performance, are mediated by structural and functional alterations within the prefrontal cortex (PFC). Although polysomnography and parent reports are used for stratification of SDB severity, the extent to which these parameters are related to executive dysfunction is unknown.

Methods: We recruited patients aged 5-11 years presenting for SDB management from University of Maryland (Baltimore, MD) and University of Texas Southwestern (Dallas, TX) Medical Centers. Their attention and executive function were assessed using a computer-generated Go/No-Go task, a psychometrically validated measure of response inhibition. The primary outcome was d-prime (d'), representing the ability to separate a signal from competing noise. Secondary outcomes included performance on a flanker task (cognition) using the NIH Toolbox and parent-reported executive function assessed via the Behavior Rating Inventory of Executive Function (BRIEF-2). Polysomnographic parameters include those representing obstruction, hypoxia, and sleep disruption. A correlation-adjusted regression analysis was used to rank polysomnographic predictors of primary and secondary outcomes while adjusting for collinearity among them.

Results: 77 patients with SDB were included in the study (mean age=7.8 [7.4-8.2]; 51.9% male; 55.8% Black). The mean apnea hypopnea index (AHI) was 12.4 [9.0-15.8] and mean oximetry nadir was 86.8% [85.1-88.5]. The mean BRIEF-2 t-scores representing Behavioral Regulation, Emotional Regulation, and Cognitive Regulation indices were 52.6 [49.9-55.3], 54.27 [51.5-57.1], and 51.6 [49.2-54.1], respectively. Results from the analysis of PFC function from the Go/No-Go task and related insights are forthcoming.

Conclusions: TBD

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O1.04 Predictors of Pulmonary Hypertension in Children with Obstructive Sleep Apnea and Down Syndrome

Presenter: Sarah Yang[1]
Mentor: Amal Isaiah, MD, PhD[1,2,3,4]
[1]Department of Otorhinolaryngology, University of Maryland School of Medicine, Baltimore, MD
[2]University of Maryland Institute for Health Computing, Bethesda, MD
[3]Department of Pediatrics, University of Maryland School of Medicine, Baltimore, MD
[4]Department of Diagnostic Radiology and Nuclear Medicine, University of Maryland School of Medicine, Baltimore, MD

Background and Aims: Down Syndrome (DS) is the most common chromosomal disorder. Patients with DS are at high risk of sleep-disordered breathing (SDB), which increases right heart strain and pulmonary hypertension (PH). However, the relationship between OSA severity and right heart dysfunction in DS remains poorly characterized. We hypothesized that greater severity of OSA, measured by apnea hypopnea index (AHI), is associated with increased right ventricular systolic pressure, representing right heart function. We also aimed to define DS-OSA phenotypes based on OSA and PH severity metrics.

Methods: To address this knowledge gap, we retrospectively analyzed medical records of patients aged 0-18 years diagnosed with DS and OSA within the University of Maryland Medical System (UMMS). We collected transthoracic echocardiograms and polysomnographic records manually. To define symptom burden and extract DS dysmorphology features related to upper airway obstruction, we fine-tuned an OpenAI O-1 based large language model (LLM) to analyze relevant encounter-based consult notes, extracting pertinent terms associated with SDB symptoms and anatomical features. We will assess the relationships between quantitative and qualitative variables representing DS features, OSA severity, and right heart function.

Results: Our cohort comprised 243 patients with DS and OSA (mean age=11.1 [9.2-13.0]; 55.6% male; 51% white). Of the 162 with sleep study data, the mean AHI was 30.9 events/hr [25.0-36.9] and mean oximetry nadir was 80.8% [78.9-82.6]. Congenital heart disease was present in 110 patients (67.9%), while 33 patients (20.4%) had pulmonary hypertension. Results from the LLM-based symptom burden extraction and related insights are forthcoming.

Conclusions: TBD

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O1.05 Clinical Validation of the Role of Caveolin-1 in Radiation and Chemotherapy Resistance in Lung Cancer

Presenter: Daniel Pyo[1]
Mentors: Hem Shukla, PhD[1]; Sanjit Roy, PhD[1]; Dan Kunaprayoon, MD[1], Zach Keepers[1]
[1]Department of Radiation Oncology, University of Maryland School of Medicine, Baltimore, MD

Non-small cell lung cancer (NSCLC) remains a leading cause of cancer-related death, with limited therapeutic options for patients who develop resistance to chemotherapy and radiation therapy (CRT). Emerging evidence suggests that Caveolin-1 (CAV1), a membrane protein, plays a critical role in tumor progression and therapeutic resistance. Preliminary data from Dr. Shukla's laboratory indicate that CAV1 upregulation is associated with CRT resistance and increased tumor aggression in NSCLC models. We also believe this protein interacts with another important receptor called EGFR which is known to help lung cancer cells grow and spread, highlighting its potential as a therapeutic target. This study seeks to elucidate the molecular mechanisms by which CAV1 contributes to CRT resistance through its interaction with EGFR signaling, with the goal being to one day turn these findings into identifying novel strategies to overcome resistance and improve outcomes for NSCLC patients. In this proposed research project, we hypothesize that aberrantly activated Caveolin-1 and its interactions with EGFR, a receptor involved in modulating cell replication and growth, will be co-upregulated with each increase in tumor stage (1-3), conferring to increased tumor aggression and CRT resistance in lung cancer. For this study, I will start with the acquisition of NSCLC tissue samples, followed by IHC staining, Caveolin-1 analysis, and EGFR analysis. After completing this study, we hope to have a better understanding of the mechanism of Caveolin-1 in NSCLC and its interactions with EGFR and essentially determine whether upregulation of Caveolin-1 leads to increased or decreased EGFR protein levels across the three different stages of lung cancer.

This research was supported in part by the Radiation Oncology Summer Fellowship Program, University of Maryland School of Medicine Office of Student Research.

O1.06 Social Determinants of Health in Pediatric Respiratory Disease

Presenter: Peter Jensen, MS[1]
Mentor: Siddhartha Dante, MD[1]
Other Co-Authors: Hannah Goodwin, MD[1]; Adrian Holloway, MD[1]; Alexander Dulla, MHA[1]
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Social determinants of health (SDoH) have been shown to have significant effect on pediatric health outcomes in respiratory disease from an early age. Recent advancements in respiratory support and pharmacology have shown to reduce intubation and hospital admissions for children with pulmonary diseases. However, there has been an increase in respiratory support patients admitted to the pediatric intensive care unit (PICU). This inconsistency requires an analysis of the sociodemographic background relation to the increase in burden on critical care resources. Our goals are to (1) identify significant univariant sociodemographic factors in respiratory disease severity, (2) analyze referring hospital capabilities, and (3) conduct a multivariant comparison between significant univariant variables. We hypothesize that children who face higher SDoH will have a significant difference in respiratory disease severity from those who face lower SDoH. A single center retrospective chart review was conducted from 2018-2023 of patients aged 0-5 admitted to the University of Maryland Medical Center (UMMC) PICU for a primary respiratory admission. Included in the data is the child opportunity index, using 44 variables to determine the quality of resources and health of neighborhoods. Additionally, length of stay and change in level of respiratory support will be used to determine severity of illness.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O2.02 Evaluating the role of macrophages in a Matched Model of Graft vs Host Disease (GVHD) – ocular and systemic GVHD.

Presenter: Andrew Myers[1]
Mentor(s): Sarah Sunshine, MD[1,2]
Other Co-Author(s): Cassidy Beck[1]; Andrew Kang[1]; Justin Zhang[1]
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Patients with hematologic malignancies can receive an allogeneic hematopoietic stem cell transplant (allo-HSCT), unfortunately, this life-saving therapy can have life-long and devastating consequences, most notably Graft vs Host Disease (GVHD). GVHD can affect multiple organ systems including the eyes. Ocular Graft vs Host Disease (oGVHD) occurs when donor T-cells attack the recipient's ocular surface tissue. oGVHD causes a severe inflammatory dry eye disease resulting in dryness, blurred vision, increased risk of infection, pain, and perforation of the cornea. The aim of this project is to evaluate a mouse model of oGVHD to better characterize the role of macrophages and apoptosis in the pathogenesis of oGVHD. Key proteins - Colony Stimulating Factor 1 Receptor (CSF1R - macrophage), Granzyme B (GZMB - apoptosis), and Caspase 3 (apoptosis), were examined to better understand their role in ocular and systemic GVHD development. Recently, the FDA approved a monoclonal antibody, Axatilimab (CSF1R inhibitor), to treat chronic systemic GVHD. Axatilimab is a CSF1R inhibitor which prevents macrophage growth and survival, leading to lower absolute macrophage numbers throughout tissues. The efficacy of Axatilimab highlights the importance of understanding macrophage biology in specific tissue, specifically CSF1R in oGVHD. In the mouse model of oGVHD, the genes of interest were studied using RT-qPCR to detect and quantify the expression of specific genes that relate to macrophage and apoptosis to better understand the pathophysiology of oGVHD. We hypothesize the expression of CSF1R will be notably elevated in the ocular and systemic tissue, reflecting increased macrophage activity. The expression of GZMB and Caspase 3 will be amplified, correlating to a direct increase in apoptotic activity as well.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State, the Cigarette Restitution Fund (SBS), and the CIBMTR KL2 (SBS).

O2.03 Computer Vision Tool for Diagnosing Ocular Graft vs Host Disease

Presenter: Anirudh Addepalli[1]
Mentor(s): Sarah Sunshine MD[1]
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Ocular Graft-versus-Host Disease (oGVHD) is a significant morbidity that occurs in more than 50% of patients following a life-saving allogeneic hematopoietic stem cell transplantation (HSCT), with substantial impacts on patients' visual function and quality of life. It is characterized by a T cell-mediated immune response that leads to inflammation of ocular structures, including the lacrimal glands, eyelids, cornea, and conjunctiva. The diagnosis of oGVHD can be challenging due to its varied clinical presentations, requiring objective and reliable diagnostic methods by an ophthalmologist, ideally a cornea specialist. The goal of this project is to develop an artificial intelligence (AI)-based diagnostic tool capable of detecting oGVHD in its early stages using clinical data and ocular imaging. A convolutional neural network (CNN) was trained on 252 labeled green fluorescein images. The model achieved 95.92% accuracy, 0.239 loss, precision of 0.91, recall of 1.00, F1-score of 0.95, AUC-ROC of 0.97, and specificity of 0.93. The model was optimized by adjusting parameters such as network size, image resolution, and epochs to reach these metrics and weights were added to address negative class bias. The final model was 6 convolutional layers, images were resized to 1024x768 pixels, and the model was trained over 22 epochs. Next steps include building a model for conjunctival injection and combining multiple diagnostic strategies into a unified tool to further improve diagnostic accuracy and enable earlier intervention for oGVHD. This project aims to enhance clinical workflows by providing an automated, user-friendly system for diagnosing oGVHD, ultimately improving patient care and outcomes.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State, University of Maryland School of Medicine Office of Student Research, the Cigarette Restitution Fund (SBS), and the CIBMTR KL2 (SBS).

O2.04 Utilizing Tear Cytokine Changes to Determine the effect of Systemic Therapy on the eyes in patients with ocular GVHD: The Impact of JAK Inhibition

Presenter: Emma Jamka[1]
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Graft-versus-host disease (GVHD) is a severe complication following hematopoietic stem cell transplantation (HSCT). Among patients who develop GVHD, up to 60% also suffer from ocular GVHD (oGVHD). This is a form of chronic GVHD that occurs when the HSCT donor's cells attack the recipient's eye tissues, leading to debilitating ocular surface disease from inflammation and resulting in corneal damage. While several therapies have recently received FDA approval for the treatment of chronic GVHD, their ocular impact remains unclear. This study explores whether Janus kinase (JAK) inhibitors like Ruxolitinib can effectively target oGVHD by modulating cytokine-driven inflammation. Tear samples (n=40) were collected from 28 patients classified into four groups: [1] HSCT recipients with oGVHD who were on JAK inhibitors (n=9), [2] HSCT recipients with oGVHD who were not on JAK inhibitors (n=16), [3] HSCT recipients without oGVHD (n=9), and [4] No HSCT controls (n=6). The proteomic analysis software OLINK was used to measure 45 inflammatory proteins. Statistical comparisons were then conducted using t-tests in R. 12 cytokines exhibited statistically significant differences between patients who received JAK and patients who did not. Cytokine expression was found to increase significantly between the controls and the definite oGVHD groups in KRT18, AREG, FASLG, IL1A, IL1RN, LIF, PDCD1, and IL4R. Conversely, cytokine expression was found to decrease significantly between the controls and the definite oGVHD groups in IL17D, IL19, and IL20. These results support our assertion that patients' tears can be used to show an effect on the ocular surface and a potential future biomarker for therapeutic efficacy both in the eyes and systemically.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State, the University of Maryland School of Medicine Office of Student Research, the Cigarette Restitution Fund (SBS), and the CIBMTR KL2 (SBS).

O2.05 Evaluating Serum Tears as a Treatment for Ocular Graft vs. Host Disease after Hematopoietic Stem Cell Transplantation

Presenter: Jack Yang[1]
Mentor(s): Sarah Sunshine, MD[1]
[1]Department of Ophthalmology, University of Maryland School of Medicine, Baltimore, MD

Ocular graft vs. host disease (oGVHD) is a common and debilitating complication characterized by severe inflammatory dry eye disease arising in 40-60% patients who have undergone hematopoietic stem cell transplantation (HSCT). Autologous serum eye drops, also referred to as serum tears, are eye drops prepared from a patient's own blood. Serum tears act as a lacrimal substitute and contain essential proteins and growth factors that facilitate corneal healing and help maintain a healthy ocular surface. While serum tears have shown therapeutic potential in other severe dry eye conditions, their efficacy in treating oGVHD remains understudied. We conducted a retrospective cohort study analyzing patient data from the Stoler Eye Clinic from 2020-2025. We compared oGVHD severity, measured on an 11-point scale by the International oGVHD Consensus Group, between post-HSCT patients with definite oGVHD who were treated with serum tears (n=10) and those who were recommended but never started treatment (n=7). Comparing the change in oGVHD severity from baseline between the two groups at 1, 6, and 12 months after treatment initiation/recommendation, we found that there was a significant decrease in oGVHD severity in the serum tears patients. When breaking down the oGVHD severity score into its parameters, the severity of corneal fluorescein staining and conjunctival injection was reduced the most in patients on serum tears. These findings support the potential effectiveness of serum tears in treating oGVHD and highlight the need for further investigation in larger, controlled studies.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research, the Cigarette Restitution Fund (SBS), and the CIBMTR KL2 (SBS).

O2.06 Measuring T-cell Expression in Ocular and Systemic Tissue in Response to Systemic and Topical JAK 1/2 Inhibitors in a Murine Model of Ocular Graft Versus Host Disease

Presenter: Andrew Kang[1]
Mentor: Sarah B. Sunshine, MD[1]
Other Co-Authors: Justin Zhang[1]; Andrew Myers[1]
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Ocular graft versus host disease (oGVHD) arises as a common complication of allogeneic hematopoietic stem cell transplantation (allo-HSCT) affecting roughly 40 to 60% of allo-HSCT recipients. oGVHD causes inflammation, scarring, and dryness of the ocular surface with limited therapies currently available. Ruxolitinib, recently approved for the treatment of chronic GVHD, is a JAK 1/2 inhibitor that downregulates inflammatory cytokines and modulates T-cell activity both of which are thought to be important in ocular damage in oGVHD; however, the precise mechanism in the eyes is poorly understood. Our project aims to characterize T-cell expression in ocular and systemic tissue in an MHC-matched murine model of oGVHD in response to treatment with systemic or topical JAK 1/2 inhibition. All mice received T and B-cell depleted bone marrow transplants, with the disease group receiving donor splenocytes to induce GVHD development. Within this group, mice received JAK 1/2 inhibitor administered via chow or eyedrops, or no treatment. We measured the expression of CD3, CD4, and CD8 in spleen, conjunctiva, and lacrimal tissue from the mice using RT-qPCR. The anticipated results include significantly elevated T-cell markers in mice with oGVHD compared to the negative controls, with an expected decline in T-cell markers in all tissues in mice that had systemic JAK treatment and a decline within only ocular tissue in mice receiving topical JAK eyedrops. These results suggest the importance of T-cell activity in the development of oGVHD and the potential efficacy of JAK 1/2 medications in oGVHD management.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research, the Cigarette Restitution Fund (SBS), the CIBMTR KL2 (SBS), Incyte Corporation, and Novartis Pharmaceuticals Corporation.

O3.01 Mapping Health Inequities: Leveraging Geospatial Analysis to Refine Food Desert Identification and Its Impact on Diabetes Outcomes

Presenter: Olohitare Abaku[1,2]
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Increasing Type 2 diabetes (T2D) prevalence in low-income and minority communities underscores the urgent need to address structural drivers of health disparities, such as food deserts. Food deserts are geographic areas with limited access to nutritious and affordable food and are closely linked to poor diabetes outcomes. Current food desert identification relies on area-level measures that can exclude households with reduced food access. Recent advances in individual-level data enable more precise identification of food deserts that can clarify impacts on diabetes etiology. We aim to leverage individual-level geospatial and electronic health record data to more precisely measure food deserts and assess their impact on diabetes outcomes in Maryland. We match the GPS-Health dataset with address-level economic data and exact food retailer locations to patient-level diabetes outcomes from an electronic health record diabetes registry. We perform logistic regressions to test the association of food desert exposure in Maryland with T2D diagnosis prevalence (ICD-10 codes, medication use, or A1C ≥6.5%) and poor glycemic management amongst T2D patients (A1C ≥ 9%). The registry includes n=92,338 adult T2D patients. Mean A1C is 8.14 (standard deviation 1.66). We will report the geographic extent of food desert exposure in Maryland, odds ratio of diabetes diagnosis by food desert exposure, and odds ratio of A1C ≥ 9% by food desert exposure. Precise food desert extent and its association with diabetes outcomes provide necessary information to community advocates, policymakers, and clinicians to prioritize and advance public health equity through data-driven, place-based solutions.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State and the MD Institute for Health Computing (UM-IHC).

O3.02 Comparing Semantic and Numerical Representations in Clinical Risk Prediction

Presenter: Aaron Ge[1]
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While large language models (LLMs) excel at natural language understanding, their effectiveness for clinical risk prediction compared to traditional numerical approaches remains unclear. We investigated whether semantic representations of clinical data offer complementary or superior predictive information to numerical features for in-hospital mortality prediction.

Using MIMIC-III data from 22,591 ICU patients, we compared semantic text embeddings against a strong XGBoost numerical baseline. We systematically evaluated three data serialization formats: uninterpreted clinical values (F1), interpreted clinical values (F2), and narrative clinical summaries (F3), each tested with six prompting strategies (P0-P5) across three Google text embedding models. The numerical baseline used 458 engineered features from the first 24 hours of ICU stay.

The XGBoost numerical baseline achieved superior performance (AUROC: 0.9080, 95% CI: 0.8968-0.9193; AUPRC: 0.6193). Among semantic approaches, narrative summaries consistently outperformed structured formats, with the best embedding model (F3+P2, text-embedding-004) achieving AUROC: 0.8384 (95% CI: 0.8217-0.8541), representing a performance gap of 0.07 AUROC points. Prompt engineering showed modest but consistent effects, with persona-driven prompts (P2) generally optimal. Performance varied significantly across embedding models: text-embedding-004 > embedding-001 > text-embedding-005.

Despite sophisticated prompt engineering and multiple representation formats, semantic embeddings underperformed traditional numerical features for clinical risk prediction. However, the systematic evaluation reveals that data serialization format significantly impacts embedding quality, with narrative representations showing the most promise. These findings suggest complementary rather than replacement roles for semantic approaches in clinical prediction systems.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O3.03 Trends in Weight Management Therapies Among Patients with Obesity and Type 2 Diabetes

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Obesity and type 2 diabetes (T2D) are associated with increased risks of cardiovascular disease, kidney complications, and premature mortality. Clinical guidelines recommend the use of glucagon-like peptide-1 receptor agonists (GLP-1RAs) for individuals with comorbid T2D and obesity due to their benefits in promoting glycemic management, weight loss, and cardiovascular and kidney protection. However, their utilization is limited by high out-of-pocket costs, insurance restrictions, and drug shortages. Older anti-obesity medications (AOMs) remain more accessible and affordable but are less effective and historically underutilized. There is limited contemporary data evaluating real-world use and costs of weight-reducing medications (GLP-1RAs and AOMs) in this high-risk population. In this study, we aim to investigate the natural history of obesity pharmacotherapy among patients with T2D and obesity. A retrospective cohort study was performed using de-identified administrative claims data from the OptumLabs Data Warehouse to evaluate trends in the utilization and costs of GLP-1RAs and older AOMs in 1.2 million adults from 2010 to 2024. Ongoing analyses will evaluate quarterly prescription trends, treatment trajectories, and costs. Specifically, we will calculate the total, health plan, and out-of-pocket costs for each medication's 28-day supply over the study period. We anticipate GLP-1RA utilization will increase significantly over time, while use of AOMs will remain low. We further anticipate that GLP-1RA use will be lower among individuals with lower income levels. This study will be used to inform future health policy and clinical efforts to optimize obesity pharmacotherapy in patients with T2D.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O3.04 Investigating the Impact of GLP-1 Receptor Agonists on Epigenetic Changes and Inflammatory Responses in Human Ileum and Gallbladder Mucosa

Presenter: Olivia Linus [1]
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Glucagon-like peptide-1 Receptor Agonists (GLP-1 RAs), such as semaglutide, lower blood sugar levels, promote weight loss, and exert anti-inflammatory effects. However, the mechanisms behind their dose-dependent actions, particularly their association with gallbladder inflammation, remain unclear. We hypothesize that GLP-1 RAs engage in distinct biological pathways in a dose-dependent manner. Characterizing these pathways may help optimize therapeutic strategies and reduce side effects, such as acute cholecystitis. To test this hypothesis, we utilize polarized human stem-cell-derived mucosa from the ileum (HIM) and gallbladder (HGM). Varying concentrations of semaglutide are applied basolaterally to mimic subcutaneous delivery both under homeostatic conditions and under an inflammatory response induced using *Salmonella* Typhi flagellin. Epigenetic gene expression signatures and pro-inflammatory cytokine production are assessed across a dosage range. Our data shows that cytokine secretion and the expression of epigenetic markers vary with semaglutide dosage, tissue type, and inflammatory status. These findings support a model in which low versus high concentrations of GLP-1RAs activate distinct pathways, helping to explain the clinical observation that weight-loss efficacy decreases over time while insulin production remains stable. Characterizing these dose-dependent mechanisms may support personalized GLP-1 RA treatments that retain metabolic benefits while reducing gallbladder-related side effects.

This research was supported in part by NIAID, NIH, DHHS federal research grant U19-AI181108 (RM, RP#3). The content is solely the responsibility of the authors and does not necessarily represent the official views of the NIAID or NIH. Additionally, this research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

O3.05 Assessing, Scanning, and Curating: Enhancing Local Health Officer Orientation Through a National Environmental Scan

Presenter: Habib Camara[1]
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Local Health Officers (LHOs) are critical community health leaders, but often face significant challenges navigating their complex roles, especially early in their service. This project aims to enhance LHO orientation in Maryland through a systematic review of existing nationally available orientation materials and guidance documents.

The systematic review was a multi-phase approach. Phase 1 identified State Associations of County and City Health Officials (SACCHOs), Phase 1 focused on identifying State Associations of County and City Health Officials (SACCHOs) through NACCHO's database, followed by qualitatively assessing their primary websites and categorizing them into statuses such as 'Highly Current,' 'Current & Functional,' 'Needs Attention,' and 'Significantly Outdated' or 'Unavailable'

Phase 2 involved curating a comprehensive dataset of resources, utilizing inductive thematic analysis to derive essential LHO topics, identify best practices, and pinpoint support gaps. Key deliverables include a curated resource guide and actionable recommendations for Maryland's LHO orientation program.

Initial investigation revealed that the existence of State Associations of County and City Health Officials (SACCHOs) and their functional online presence varied more than anticipated. These findings from Phase 1 revealed that 23 (67.6%) of assessed SACCHO websites were categorized as 'Highly Current.' Additionally, findings indicate limited availability of general "Health Officer Resources" (only 20 (58.8%) of SACCHOs providing them). Specific "Health Officer Orientation Materials" were even scarcer, found in only 4 (12.9%) of 31 assessable SACCHOs. This disparity between website currency and targeted orientation content highlights a substantial opportunity to enhance resource provision for incoming LHOs.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

O3.06 Strengthening Maryland's Healthcare Workforce: A Comparative Analysis of Maryland Loan Repayment Program's Applicants and Awardees

Presenter: Maclean Panshin, MPH[1]
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Background: Maryland faces persistent healthcare workforce shortages, especially in rural and medically underserved areas. The Maryland Loan Repayment Programs (MLRP), including the Maryland Loan Assistance Repayment Program (MLARP) and the State Loan Repayment Program (SLRP), aim to address this by offering educational loan repayment to physicians, advanced practice registered nurses, physician assistants, and nursing staff who commit to serving high-need communities.

Objective: To analyze and compare demographic and practice characteristics of MLRP applicants and awardees from the 2025 application cycle, with a focus on identifying disparities between those who applied and those who ultimately received awards. Understanding these differences can inform strategies to strengthen equitable access to loan repayment support and improve workforce distribution.

Methods: Applicant and awardee data, including age, gender, race, ethnicity, specialty, and practice site information, were analyzed using descriptive statistics. Comparisons were made to examine differences in demographic representation between applicants and awardees. Special attention was given to groups underrepresented among awardees despite strong application rates.

Results: Preliminary findings indicate that early-career professionals and those in primary care or emergency medicine specialties were more likely to receive awards. The majority of awardees were female, with a notable increase in representation among Black physicians compared to Maryland's physician workforce overall. However, certain demographic groups and specialist applicants experienced lower award rates, reflecting the program's prioritization of primary care. Geographically, most awardees served in Health Professional Shortage Areas (HPSAs) or Medically Underserved Areas/Populations (MUA/Ps), with a concentration around Baltimore City.

Discussion: This analysis highlights how MLRP selection criteria shape award distributions and impact workforce diversity. Identifying disparities between applicants and awardees supports ongoing efforts to refine program design, strengthen equitable recruitment, and ensure that loan repayment incentives effectively address Maryland's most critical healthcare workforce needs.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

O4.01 Cognitive Function and Cerebrovascular Reactivity in Persons with HIV.

Presenter: Swapno Chaudhuri[1]
Mentor: Linda Chang, MD, MS[1]
Other Team Members: Celine Koko, BS[1]; Jordan Walter, MD, PhD[1]; Huajun Liang, MBBS; PhD[1]; Fariba Badrzadeh, MD[1]; and Peiying Liu, PhD[1]
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Persons with HIV (PWH) are experiencing near-normal life expectancies due to the increasing effectiveness and availability of antiretroviral therapies. In 2022, 54% of PWHs were age 50 years or older in the U.S. and this is projected to increase to 70% by 2030. HIV-associated neurocognitive disorders were found in 30-50% of all PWH, with a significantly higher prevalence in those 50 years of age or older. We hypothesized that impairment of MRI-assessed cerebrovascular reactivity (CVR), the ability of blood vessels in the brain to dilate or constrict in response to oxygenation or pH, in PWHs is associated with cognitive impairment or dysfunction. 19 HIV-seronegative controls and 35 HIV-seropositive participants were administered a battery of neurocognitive assessments, such as the Montreal Cognitive Assessment, standard neuropsychological testing methods, and the NIH Toolbox to assess 7 cognitive domains. These scores were used along with a Cognitive Dementia Rating and a history of symptom onset to diagnose participants with “No Cognitive Impairment”, “Asymptomatic Neurocognitive Impairment”, “Mild Neurocognitive Disorder”, or “HIV-associated Dementia”. In all participants, MRI data were collected using a 3T Siemens Prisma system; the sequences include BOLD fMRI, T1-MPRAGE, T2-FLAIR, and diffusion-weighted-imaging. Significant correlations between neurocognitive diagnoses, CVR, and quantified results from neurocognitive testing were found in our preliminary analyses. These results suggest that CVR dysfunction may contribute to neurocognitive impairment in PWH and may be a biomarker that can be used to diagnose or assess the severity of cognitive impairment in PWH.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

O4.02 Characterizing Naive Expression of Lactate Dehydrogenase Isoforms A and B in a Cell-Type Specific Manner

Presenter: Kirsten Snyder
Mentors: Alexander Ksendzovsy, MD, PhD[1]; Marc Simard, MD, PhD[1];
Co-Authors: Tyler Wishard, PhD[1]; Mitchell Moyer, PhD[1]; Mathew Kreinbrink[1]
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Alterations in energy metabolism in the brain underlie a plethora of neurologic disorders. Epilepsy is a debilitating neurologic condition that can be very challenging to treat due to a limited understanding of the numerous etiologies underlying symptomatology at a cellular level. Current research demonstrates that cells within epileptogenic tissue exhibit elevated rate levels of Lactate Dehydrogenase (LDH) isoform A, but not B. LDHA is responsible for the enzymatic conversion of pyruvate to lactate while LDHB catalyzes the reverse reaction. This variation in LDHA and LDHB expression between seizure and non-seizure states has led to interest in the differential expression of both isoforms in naive tissue, and where expression is localized throughout a non-epileptic brain. To characterize expression we used immunofluorescent labeling on naive brains assessing for both LDH isoforms and colabeling with cell markers for neurons and astrocytes. This revealed that cells labeled for LDHA co-expressed neuronal marker NeuN, but not astrocytic marker GFAP, whereas cells labeled for LDHB co-expressed both neuronal and astrocytic markers. This suggests that LDHA is expressed primarily in neurons, while LDHB expression was observed in both neurons and glia. Additionally, LDHA was expressed throughout the cortex, hippocampus, hypothalamus, and brainstem in our naive model, while LDHB was primarily expressed in the cortex and brainstem. This finding likely has implications for the role of LDHA versus LDHB in seizure onset. We anticipate that our findings will help further our understanding of the biochemical changes occurring in the epileptic brain, paving the way for more effective therapeutics.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O4.03 Investigation of Molecular and System-Level Brain Changes Following Temporal Lobe Contusion in the Development of Post-Traumatic Epilepsy

Presenter: Tyler James Wishard, PhD[1]
Mentors: Alexander Ksendzovsky, MD, PhD[1]; Vladimir Gerzanich, PhD[1]; and J Marc Simard, MD, PhD[1]
Co-Authors: Kirsten Snyder[1]; Mitchell Moyer, PhD[1]; Kaspar Keledjian, PhD[1]; Orest Tsymbalyuk, PhD[1]; Cigdem Tosun, PhD[1]
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Traumatic Brain Injury (TBI) is a leading global cause of morbidity and mortality, affecting 69 million people annually. In Maryland, the lifetime prevalence of TBI-related hospitalizations is 30%, with Baltimore City having the highest rate. While TBI is clinically heterogeneous, certain injury patterns, such as temporal lobe contusion (tlCont), increase risk for post-traumatic epilepsy (PTE). However, the mechanisms linking tlCont to PTE remain unclear. We developed a mouse model of tlCont targeting the entorhinal cortex with traumatic interstitial hemorrhage. Previous work has demonstrated that this injury results in electrographic seizures and hippocampal-dependent behavioral deficits. Here, we extended continuous video-EEG monitoring and identified a peak in seizure frequency between 4–6 weeks post-injury. To investigate structural changes underlying this epileptogenic window, we performed serial MRI to evaluate the entorhinal-hippocampal circuit—specifically the perforant path, which connects the entorhinal cortex to the hippocampus via the subiculum and is critical for memory and spatial navigation. Baseline MRI showed no statistical differences between sham and injured mice. We hypothesize that volumes of the hippocampus, entorhinal cortex, and subiculum ipsilateral to injury will be reduced at 1 and 4 weeks post-injury. Preliminary histological analysis using NeuroSilver™ staining suggests increased hippocampal degeneration in tlCont brains compared to controls. Ongoing work will validate these findings and examine microstructural changes. These results support the hypothesis that altered entorhinal-hippocampal connectivity contributes to epileptogenesis after TBI. Future studies will use a within-subject design to correlate seizure burden with structural connectivity, behavioral outcomes, and perforant path integrity.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research. We acknowledge the support of the University of Maryland, Baltimore, Institute for Clinical & Translational Research (ICTR) and the National Center for Advancing Translational Sciences (NCATS) Clinical Translational Science Award (CTSA), UM1TR004926.

O4.04 Association between Neighborhood Socioeconomic Status and Pre-Hospital Management of Status Epilepticus

Presenter: Aamna Cheema
Mentor: Matthew R. Woodward, DO, MS
Other Co-Authors: Ashwin Reddi, MD; Maeve Murphy, BS; Sneha Kuppireddy; Matthew Williams; Eric Yang; Timothy Chizmar, MD; Nicholas A. Morris, MD; Gunjan Y. Parikh, MD; Neeraj Badjatia, MD

Background and Purpose:
Pre-hospital status epilepticus (SE) management includes benzodiazepine administration and consideration of intubation. The effect of neighborhood socioeconomic status (nSES) on pre-hospital SE management is not known. We aim to evaluate the association between neighborhood SES and outcomes in patients receiving pre-hospital SE care.

Methods:
We identified patients with SE, defined as EMS primary or secondary impression of 'seizure' and receipt of benzodiazepine, within a cohort of EMS transports for neurological problems within Maryland from 1/1/2016 to 6/30/2020. We excluded age<18, age>100, post-cardiac arrest, and intubation prior to benzodiazepine administration. Exposure was national Area Deprivation Index (ADI) at EMS pickup (1-100, higher indicates more neighborhood deprivation). We performed a mixed-effects multivariate linear regression for our primary outcome of time to benzodiazepine administration, controlling for EMS agency as a random effect, and demographics, vital signs, substance use indicators, and Glasgow coma scale (GCS) as fixed effects. We performed a mixed-effects logistic regression to evaluate odds of pre-hospital intubation and included time to benzodiazepine and benzodiazepine dose.

Results:
Of 124,667 patients, 1578 met inclusion criteria. Median (inter-quartile range (IQR) age, national ADI, and time to benzodiazepine administration were 48 years (32-63), 39 (IQR: 25-51), and 12.1min (7-20) respectively. Longer time to benzodiazepine administration was associated with higher GCS (β: 0.70min, 95% confidence interval (CI): 0.57-0.83min, p<0.00001), but not national ADI (β: 0.007min, 95%CI: -0.02-0.04min, p=0.66). Sixty-six (4.2%) patients were intubated. Higher ADI (Odds ratio (OR); 0.97, 95% CI: 0.95-0.99) and oxygen saturation (OR: 0.93, 95% CI: 0.89-0.98) were associated with lower odds of intubation.

Conclusion:
Lower nSES (higher ADI) was associated with lower odds of pre-hospital intubation, but nSES was not associated with time to benzodiazepine administration. These findings suggest that nSES may influence subjective assessments, such as airway protection assessments more than protocolized actions like benzodiazepine administration in SE. Further studies are needed.

O4.05 iPSC and iPSC-EVs Therapies for Peripheral Nerve Regeneration Following Sciatic Nerve Crush in Rats.

Presenter: Andrew Hummer[1,2]
Mentor(s): Xiaofeng Jia, MD, PhD, FCCM[1,2]
Other Co-Author(s): Anam Anjum, PhD[1]; Abel Lindley, BS[1]; Joy Odigbo, BS[1,2]
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Peripheral nerve injuries (PNI) impair sensory and locomotor functions and are accompanied by permanent injury and lifelong complications. Effective recovery in animal models depends on restoration of sensorimotor function, prevention of muscle atrophy, particularly in the gastrocnemius muscle, and regeneration of injured demyelinated nerve fibers. Current treatment approaches are often limited in efficacy, invasive in nature, and associated with risks including immune reactions and potential damage to surrounding healthy nerve tissue. Previous research has demonstrated that the transplantation of induced pluripotent stem cells (iPSCs) restored sensorimotor function and promotes neuronal repair and regeneration. However, iPSCs' clinical utility is restricted by challenges such as immune rejection, low in vivo stability, and tumorigenic potential. In contrast, extracellular vesicle therapy offers a promising, less invasive alternative in comparison to stem cell therapy. In this study, the therapeutic efficacy of iPSCs versus induced pluripotent stem cell-derived EVs (iPSC-EVs) following a sciatic nerve crush injury in rats was examined. Immediately after a sciatic nerve crush injury, 2×10^5 iPSC cells and 20 ng/mL iPSC-EVs were administered via intraneural injection. Rats were assigned to either the iPSC or iPSC-EV treatment group (n = 8) and monitored over 3 weeks. Functional recovery was assessed using the CatWalk gait analysis to evaluate locomotor coordination. Masson's trichrome staining was used to assess fibrosis in gastrocnemius muscle tissue. Immunohistochemical analyses using NF200 and MBP markers were used to assess axonal regeneration and remyelination in the sciatic nerve. Rats treated with iPSC-EV had significantly higher max contact area (cm^2) ($p<0.05$) and print area (cm^2) ($p<0.05$) in Catwalk than rats treated with iPSC, indicating a better locomotor recovery. Masson's trichrome staining indicated significantly greater muscle fiber positive area ($p<0.05$) in rats treated with iPSC-EV compared to rats treated with iPSC. Immunohistochemical analyses showed significantly greater neurofilament mean fluorescent intensity ($p<0.05$) and positive area ($p<0.01$), as well as significantly greater myelin mean fluorescent intensity ($p<0.0001$) and positive area ($p<0.01$) in iPSC-EV treated rats compared to iPSC treated rats. Our study showed that treatment of iPSC-EVs led to significant improvement in CatWalk gait analysis, preservation of gastrocnemius muscle mass atrophy, and more robust nerve tissue regeneration compared with iPSCs treatment in current setting, suggesting that iPSC-EVs offer a safer and effective therapeutic approach for peripheral nerve repair.

This research was supported in part by the Maryland Stem Cell Research Fund (2020-MSCRFD-5384 to X. J); the National Institutes of Health (NS117102 to X. J.). Andrew Hummer was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

O4.06 Brain Recovery after Cardiac Arrest treated with Metabolic Glycoengineered Stem Cell Therapy

Presenter: Leah Han[1,2]
Mentor: Xiaofeng Jia, MD, PhD[1,2]
Other Co-Authors: Songah Chae, MS, PhD[1]; Zhulin Wang, MD, MS[1]
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Cardiac arrest (CA) is a leading cause of global morbidity and mortality, often resulting in severe hypoxic-ischemic brain injury. Recent advancements in regenerative medicine have introduced neural stem cell (NSC) therapy as a promising strategy to restore neural function after brain injury. However, major challenges remain, including NSC survival, proper differentiation, and functional integration into injured brain tissue post-transplantation. To address these limitations, our research focuses on the emerging approach of metabolic glycoengineering (MGE). By chemical modification of NSC surface glycans, MGE enhances cell-cell interactions, survival signals, and immune evasion for effective brain repair. We use the non-natural, thiol-modified ManNAc analog Ac_5ManNTProp ("TProp") to evaluate its effect on brain recovery and explore the effects of Wnt/β-catenin pathway modulation. This underlying signaling pathway is hypothesized to play a pivotal role in MGE-mediated neuroregeneration, regulating cell survival and differentiation. To evaluate the role of its downstream signaling β-catenin-GSK3β in mediating the therapeutic effects of glycoengineered NSCs in vitro, we quantified β-catenin and p-GSK3β protein expression levels in control, TProp-treated, and TProp + IWR-1 (Wnt signaling inhibitor) treated NSCs via Western blot (WB). This analysis was completed with three-day analog treatment NSC samples. NSCs were then transplanted into rat CA models for in vivo evaluation. Following CA and return of spontaneous circulation (ROSC), animals were randomized to receive TProp-modified NSCs or TProp-NSC + IWR-1 (n = 6, 4×10^5 cells in 10 µL PBS) delivered via intracerebroventricular (ICV) administration 3 hours post-ROSC. CA rat brain sections were stained using Fluoro-Jade C (FJC) to assess degenerating neuron levels in TProp-NSC vs. TProp-NSC + IWR-1 treated rats. WB band intensity ratios indicate that three-day TProp treatment significantly increased β-catenin and p-GSK3β levels, supporting MGE-mediated enhancement of Wnt signaling. ICV transplantation of TProp-NSCs following CA significantly improved neurological scores. Hippocampal FJC positive area percentage of the TProp-NSC group exhibited significantly reduced neuronal degeneration compared to the TProp-NSC + IWR group. This further signifies Wnt pathway activation in neuroprotection enabled by MGE. Our findings suggest that MGE using TProp enhances neuroprotective effects via Wnt/β-catenin signaling, revealing a promising strategy to improve stem cell-based therapies for ischemic brain injury after CA.

This research was supported in part by the R01NS125232 and R01NS110387 from the National Institute of Neurological Disorders and Stroke and 2024-MSCRFD-6401 from Maryland Stem Cell Research Fund (all to Xiaofeng Jia). LH was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

O5.01 Evaluating the Efficacy of T-Regulatory Cell Specific Interleukin-2 Nanoparticles for the Treatment of Ocular Graft versus Host Disease in a Mouse Model

Presenter: Nora Cheraghi, BA[1]
Mentor: Sarah B Sunshine, MD[1,2]
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Ocular graft-versus-host disease (oGvHD) affects ~50% of allogeneic stem cell transplant recipients when the donor immune cells attack the host's eye tissue, resulting in debilitating chronic complications. In recent years, Regulatory T cells (Tregs) have been widely investigated for their role in suppressing autoimmune responses. Interleukin-2 (IL-2) is critical for Treg-cell development, expansion, and activity. The goal of this study is to evaluate the efficacy of IL-2 nanoparticles for the treatment of oGvHD in a murine model. All mice underwent total body irradiation followed by repletion with bone marrow and splenocyte transplants, and bilateral subconjunctival injections every two weeks for a total of four treatments. The treatment group (n=5) received IL-2 nanoparticles and rapamycin. Of note, one mouse died before irradiation during the subconjunctival injection. To preferentially activate Treg cells and avoid effector T-cell stimulation, the IL-2 nanoparticles were conjugated with F5111, an antibody targeting CD25+ proteins highly expressed on Tregs. The sham group (n=5) received blank nanoparticles and rapamycin, and the control group (n=10) received sucrose. We hypothesize that subconjunctival IL-2 nanoparticles will upregulate Treg cells and prevent oGvHD development. The mice are monitored for one week post-transplant for ocular and systemic GvHD severity. Within 10-12 days post-transplant, ten mice across all groups died, suggesting an unsuccessful engraftment and reconstitution. To confirm the cause of death was not drug-related, we are conducting a toxicity study of IL-2 Nanoparticles in healthy mice. Upon confirming safety and optimal dosing, we will reassess therapeutic efficacy in an oGvHD murine model.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research, the Cigarette Restitution Fund (SBS), and the CIBMTR KL2 (SBS).

O5.02 Predicting visual acuity and glaucoma 10 years after unilateral congenital cataract surgery: Results from a Randomized, Multicenter Study

Presenter: Riva Menon[1]
Mentor(s): Janet Alexander, MD[1]
Other Co-Author(s): Claudia Wong[1]; Urjita Das[1]; He Forbes[2]; Taylor Kolosky[1]; Euna Cho[1]; Shaiza Mansoor[1]; Sera Chase[1]; Madi Kore[1]; Moran Roni Levin, MD[1]; Larry Magder, PhD[3]; Carolyn Drews-Botsch, PhD, MPH[4]; Scott R Lambert, MD[5]
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Visual outcomes after unilateral congenital cataract surgery often remain poor, despite uncomplicated surgery and adherence to patching. Age at surgery, refractive correction, and patching are the only known, albeit weak, predictors of postoperative visual acuity. Glaucoma is a sight-threatening adverse event after congenital cataract surgery and the most common secondary cause of glaucoma in children. This study evaluates ocular anatomy as a potential predictor of visual acuity and glaucoma risk 10 years after congenital cataract surgery. Improved understanding of these factors may support earlier intervention and guide patient counseling.

This post-hoc secondary analysis of the Infant Aphakia Treatment Study included 114 participants who underwent unilateral congenital cataract surgery between November 2004 and January 2007. Participants were 1-6 months old at surgery. Ocular measurements were obtained from biometry and surgical video footage, and visual acuity was assessed 10 years postoperatively. By study endpoint, 41% of the cohort was diagnosed as glaucoma or glaucoma suspect.

Subjects with glaucoma had an anterior chamber depth (ACD) of 2.76 ± 0.48 mm, compared to 3.08 ± 0.38 mm in those without (mean difference = 0.32 mm, p=0.003). Logistic regression identified shallow ACD as the strongest anatomical predictor of glaucoma (OR 5.8 [1.8, 18.9], p=0.004), outperforming axial length, lens thickness, corneal diameter, and age. In conclusion, shallower ACD at the time of surgery is strongly associated with increased glaucoma risk and may serve as a valuable tool for early risk assessment.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research. Dr. Alexander is supported by the National Institutes of Health (grant no. K23EY03525).

O5.03 Epidemiology and Risk Factors of Ocular Involvement in Graft-Versus-Host Disease After Allogeneic Hematopoietic Stem Cell Transplant

Presenter: Justin Zhang, BS[1]
Mentor: Sarah Sunshine, MD[1]
Co-Mentor: Nancy Hardy, MD[2]
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Graft-versus-host disease (GVHD) is a severe complication after allogeneic hematopoietic stem cell transplant (HSCT) that has chronic and ocular subtypes. Ocular graft-versus-host disease (oGVHD) damages the corneal surface of the eyes through inflammation that reduces tear production and lubrication, resulting in severe dry eye symptoms. Prior reports suggest that the incidence of oGVHD can be as high as 60% after HSCT. This study utilized TriNetX, a global federated research network of de-identified electronic health records, to investigate the incidence rate and risk factors for ocular involvement in patients with GVHD. Patients were queried using International Classification of Diseases, 10th Revision (ICD-10) and Current Procedural Terminology (CPT) codes. The study population consists of patients with GVHD diagnosis (D89.811) following HSCT (38,240) between January 2015 and December 2024. Ocular involvement included dry eye syndrome (H04.12), keratoconjunctivitis sicca (H16.22), and meibomian gland dysfunction (H02.88). Risk factor analyses were conducted within the TriNetX analytics platform. Chi-squared analyses, Cox proportional hazards models, Kaplan-Meier curves, and incidence rates were calculated. Out of 10,073 HSCT patients, 3,488 had chronic GVHD, and 1,273 of these GVHD patients had ocular involvement, resulting in an ocular involvement rate of 36.5%. Age, sex, race, and myeloablative regimens were shown to significantly be associated with ocular involvement. Additionally, chronic GVHD incidence has decreased over time, with 39.7% of HSCT patients from 2015-2018 developing GVHD while 26.2% of HSCT patients from 2022-2024 developed GVHD. The dataset size and characteristics suggest the need for improved and standardized coding of HSCT, GVHD, and oGVHD.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research, the Cigarette Restitution Fund (SBS), and the CIBMTR KL2 (SBS).

O5.04 The association between maternal mental health and child executive function among children who are HIV exposed and uninfected

Presenter: Gabriela, Cabrera
Mentor(s): Andrea Buchwald, PhD[1]; Miriam Laufer, MD[2]
Department of Infectious Diseases, University of Maryland School of Medicine, Baltimore, MD

Mothers living with HIV face high rates of mental health challenges, including depression and anxiety which can worsen health outcomes, including child executive functioning. While it is well established that factors like maternal education, poverty, and maternal mental health play important roles in shaping executive function, teasing apart the unique contribution of HIV exposure from these interconnected influences remains a significant challenge. We aim to examine the association between maternal depression and child executive function among a cohort including children exposed to HIV but uninfected (CHEU). We hypothesized that worsening maternal mental health will be associated with decreasing executive function in children and specifically most apparent among CHEU. We recruited HIV positive and negative pregnant women from 2018-2022 during the second trimester of pregnancy in Malawi and followed their children up to two years of age. Mental health status was measured using a Self-Reporting Questionnaire-20 (SRQ-20) and child executive function was measured using the Behavior Rating Inventory of Executive Function – Preschool Version (BRIEF-P).

HIV-positive mothers had significantly higher SRQ-20 scores than uninfected mothers (3.10 vs. 2.75, p = .015). Higher SRQ-20 scores were also observed among mothers with less than secondary education (p = .003) and those experiencing food insecurity (p = .008). Increasing SRQ-20 scores were associated with poorer executive function, however there was no effect modification by HIV status. Overall, this project highlights the need for continued research to guide targeted interventions to strengthen maternal mental health and promote childhood development.

This research was supported in part by the Maryland Infection, Immunization, Intervention, and Impact Training Program (M4I), University of Maryland School of Medicine Office of Student Research.

O5.05 Evolution of the MHC: Characterization of Nonclassical MHC Class I Gene Emergence and Evolution Using a Shark Model

Presenter: Jonathon Meyer[1]
Mentor(s): Yuko Ota, PhD[2]
Other Co-Author(s): Martin Flajnik, PhD[2] and Erik Cruz, BS[2]
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MHC-based adaptive immunity first appeared in sharks, the oldest living jawed vertebrates, which emerged ~500 million years ago. MHC class I molecules are composed of two types: classical (class Ia) and nonclassical (class Ib). Although they both exhibit structural similarities, in contrast to class Ia MHC the class Ib genes and proteins exhibit low polymorphism, tissue-specific expression, and the ability to bind to non-peptide antigens. Currently, one MHC class Ia gene (UAA) and six class Ib (UBA, UCA, UDA, UEA, UFA, and UGA) genes have been identified in sharks. Previous studies demonstrated that several of these genes are about as old as UAA, including UDA, UFA, and UGA, all of which exhibit similarities to human class Ib genes. In this study, we further characterized UDA, UFA, and UGA to better understand the evolutionary history of MHC class Ib genes and potentially shed light on human class Ib for downstream applications. Immunohistochemistry (IHC), Fluorescence-Activated Cell Sorting (FACS), and western blots were performed with monoclonal antibodies for UFA and UGA to identify the cell types expressing these genes. Fluorescence In Situ Hybridization (FISH) was used to probe RNA expression of these genes and to confirm previous hypotheses about their cell type-specific expression. UDA, UGA, and UFA were expressed in gill and in immunized and unimmunized (naïve) spleen, all in unique areas of the tissues. Additionally, UFA and UGA were expressed in spiral valve (shark small intestine) but only UGA was expressed in the rectal gland. Finally, we performed a single-cell RNAseq analysis of a peripheral blood lymphocyte (PBL) to further characterize cells expressing UFA. Examining genes expressed by the UFA-positive cells, we determined that UFA may be expressed in activated B cells. From this study, we confirmed expression patterns of UDA, UFA, and UGA and discovered a potential identity of UFA-expressing cells, which provides a basis for further analysis, such as cell types and their functions.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State and by the NIH (NIHR01AI140326)

O5.06 Testing AI-Powered Camera Effectiveness to Study Red Zone Precautions in Preventing the Spread of Antibiotic-Resistant Bacteria

Presenter: Robert McLuckey
Mentor(s): Anthony Harris, MD, MPH[1]
[1]Department of Epidemiology and Public Health, University of Maryland School of Medicine, Baltimore, MD

Antibiotic resistant bacteria, such as Methicillin Resistant *Staphylococcus Aureus* (MRSA) are a major threat to public health globally. Adding to this burden are conflicting recommendations on approaches to the use of contact precautions by healthcare workers to manage in-patient MRSA cases and prevent spread between patients. It is proposed that requiring healthcare team to wear personal protective equipment only when entering the "red zone" surrounding a patient's bed will result in a more cost-effective intervention than wearing contact precautions all or none of the time. This research also comes at a time where the role and benefits of artificial intelligence (AI) in healthcare are being heavily explored. Our study aims to 1) test a camera that will eventually use AI to detect workers while in the red zone and 2) test a break beam technology that will detect entries into the red zone. Two trials will be done, both in a simulation center and patient rooms, where a human observer will be used as a gold standard to test sensitivity and specificity of the devices at detecting red zone entries. It is hypothesized that the camera will effectively detect entries, defined with a sensitivity above 80% and specificity above 90%. We predict that this technology will be useful in a future study to determine the effectiveness of red zone precautions and have numerous other uses in patient care. Preliminary testing in the simulation center has shown poor performance by the break beam technology. The camera has yielded promising sensitivity and specificity (85% and 96%, respectively) with entries from the sides and foot of the patient's bed.

This research was supported in part by the Maryland Infection, Immunization, Intervention, and Impact Training Program (M4I), University of Maryland School of Medicine Office of Student Research.

O6.01 CAR-Tregs: Could they be the key to solving xenotransplant rejection?

Presenter: Charlotte Noyes
Mentor: Nevil Singh, PhD
Co-Authors: Mohammed Mohuiddin, MBBS[1]; Javier Galindo, MD[1]; Katrina Gorga[2]
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Xenotransplants, or transplants involving animal organs, have emerged as a potential solution to overcome organ shortage in cardiac and kidney transplantation. In practice, despite strong immunosuppressant medication, fatal organ rejection occurs in about 8 weeks. We hypothesize that a treatment that forces the immune system to stop recognizing the xeno-organs as a threat, can increase the survival of xeno-transplant recipients. We propose to use immunosuppressive T cells known as Tregs and target them to pig tissue using Chimeric Antigen Receptor (CAR) technology. CAR is usually made from an antibody that is specific for the target. Therefore, the first phase of the project is to generate anti-xeno monoclonal antibodies using hybridoma technology.

Mice immunized with pig antigens were tested by using their sera in an ELISA with pig tissue lysates. Splenocytes from the top two responding mice were isolated and fused through electrofusion with mouse myeloma cells. This allows individual B cells to survive in culture and become hybridomas, secreting single specificity of antibodies. 10 days after the fusion, we screened wells with growing colonies by ELISAs to test for antibodies reactive to pig heart lysates, live pig cell lines, and live human cell lines. Over a total of 179 colonies, we identified 54 strongly pig-reactive hybridomas and 60 borderline clones. We are currently re-testing these clones to get stable hybridoma lines continuing to produce anti-xeno monoclonal antibodies. In the next phase, the Ig genes encoding the antibody will be cloned in order to be introduced into Tregs to generate CAR-Tregs.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

O6.02 Evaluation of Platelet Adhesion and Hemodynamic Influences on ECMO Thrombosis: A Comparative Study of Biomaterial Surfaces

Presenter: Mustafa Siddiqui[1]
Mentor(s): Zhongjun Jon Wu, PhD[2]; Shigang Wang, MD[2]
[1]University of Maryland School of Medicine, Baltimore, MD
[2]Department of Surgery, University of Maryland School of Medicine, Baltimore, MD

Extracorporeal Membrane Oxygenation (ECMO) is a life-saving intervention for patients with severe cardiac and respiratory failure; however, thrombosis remains a major challenge, leading to device failure and heightened morbidity. Because of direct contact with blood, the materials used in ECMO circuits significantly influence thrombogenicity, yet a comprehensive comparative analysis of commonly used biomaterials under standardized conditions is lacking. The aim of this study was to evaluate platelet adhesion and thrombus formation on the biomaterial surfaces of silicone, polyvinyl chloride (PVC), polycarbonate (PC), polyurethane (PU), polyurethane (PE), polymethylpentene (PMP), and titanium to determine which materials exhibit the lowest thrombogenicity. Utilizing a custom-built thrombosis-on-a-chip model, we used fresh human blood to perform microscopy-based platelet adhesion assays on the surfaces of different biomaterials and under different shear stresses. Platelet adhesion on the biomaterial surface was measured under the fluorescence microscope. The coverage areas of clot formation were analyzed using Fiji ImageJ-based image analysis software and MATLAB-based programming. In addition, computational fluid dynamics (CFD) modeling was used to analyze shear stress distribution on clot formation across the different material surfaces. Preliminary data showed that platelet adhesion significantly decreased with increased shear stress from 500 s-1 to 5000 s-1 after 10 minutes, and the mean sizes of the clot clusters were greater under higher shear stress. Platelet adhesion on PVC was higher than PC at lower shear stress. Polishing titanium alloy (Ti-6Al-4V) displayed better biocompatibility. Other biomaterial assessments are currently in progress. The findings from this study will have help to develop ECMO circuits with improved hemocompatibility, ultimately enhancing patient outcomes.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O6.03 The Effect of Mechanical Circulatory Devices on Blood Components

Presenter: AyoOluwakiitan Oluwafemi
Mentor: Zhongjun Jon Wu, PhD[1]
[1]Department of Surgery, University of Maryland School of Medicine, Baltimore, MD

Mechanical circulatory support devices such as extracorporeal membrane oxygenation (ECMO) are critical in managing patients with severe cardiopulmonary failure. However, the high mechanical shear stress (HMSS) induced by these devices can cause structural damage to blood components, contributing to thrombosis, impaired hemostasis, and increased risk of infection. Previous in vitro studies have shown that morphological distortion of platelets and neutrophils correlates with functional impairments. In this study, we analyzed blood samples from patients on ECMO at multiple time points—before cannulation, during support, and after decannulation—to assess changes in cellular morphology and receptor expression. Using microscopy, we found a progressive increase in neutrophil morphological distortion over time. Analysis of neutrophil receptor CD62L expression is ongoing. Interestingly, we observed a transient resurgence of morphologically normal platelets and restored expression of CD41/61, CD42, and GPIV between days 14 and 21 on ECMO, followed by another decline. These findings suggest dynamic changes in blood cell integrity during ECMO support and raise important questions about the physiological mechanisms underlying platelet recovery. Future work will investigate these mechanisms and evaluate whether targeted therapeutics can reduce or reverse the damaging effects of HMSS on blood components, with the goal of improving the safety and efficacy of mechanical circulatory devices.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O6.04 Examining sociodemographic characteristics by place of death for Marylanders who died of heart disease or COVID-19 from 2020 to 2023

Presenter: Mubassira Nur[1]
Mentor(s): Kristen Polinski, PhD[2]; Monique Wilson, DrPH[2]
[1]Department of Biostatistics and Epidemiology, University of Maryland, College Park, MD
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Place of death is an important indicator of end-of-life care and can inform public health planning. Heart disease and COVID-19 were leading causes of death in Maryland from 2020-2023. This analysis examined sociodemographic characteristics by place of death among Marylanders who died from heart disease or COVID-19 from 2020-2023. Data from death certificates of Maryland residents were used to identify decedents with heart disease (I00-I099, I110-I119, I130-I139, I200-I519) or COVID-19 (U017) as the underlying cause of death. For the analysis, place of death was grouped as hospital inpatient, outpatient/ER, hospice, nursing home, and residence. Chi-square tests and logistic regression were used to examine associations of sociodemographic characteristics and place of death. Between 2020 and 2023, approximately 43% of heart disease deaths occurred in the decedent's home. In contrast, 7.5% of COVID-19 deaths occurred at home. Notably, inpatient COVID-19 deaths decreased from 74.8% in 2021 to 51.2% in 2023. Among married decedents, the highest proportion of deaths occurred at home for heart disease deaths (41.0%) or in inpatient care for COVID-19 deaths (75.0%). For both causes of death, compared to dying at home, non-Hispanic black decedents had higher odds of inpatient/outpatient deaths and lower odds of nursing home deaths than non-Hispanic white decedents (p-values <0.05). Compared to dying at home, women had a higher likelihood than men to die of heart disease or COVID-19 in a nursing home or hospice facility (p-values <0.05). These findings demonstrate sociodemographic differences by setting among Marylanders.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

O6.05 Factors Influencing Clinical Decision-Making for Pulmonary Vasodilation Therapies in Premature Neonates with Chronic Lung Disease

Presenter: Julia Whiteleather [1]
Mentor: Alicia Chaves, MD[1]
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Pulmonary hypertension (PH) is a common and serious complication in premature neonates with chronic lung disease (CLD), contributing to increased morbidity and mortality. While pulmonary vasodilators such as inhaled nitric oxide (iNO) and sildenafil are widely used, the short-term clinical factors guiding escalation or de-escalation of these therapies remain poorly defined. This study aims to identify which patient, clinical, and provider variables most influence physician decisions to initiate, adjust, or discontinue pulmonary vasodilation therapy in response to acute changes in respiratory status. We are conducting a retrospective chart review of neonates born at <32 weeks gestational age who were diagnosed with CLD and underwent echocardiography for suspected PH. Clinical data, including FiO_2 changes, blood gas results, ventilator settings, and echocardiographic findings (e.g., right ventricular pressure, PDA size, shunting direction), are being analyzed in relation to treatment decisions. By clarifying the relationship between acute clinical events and therapeutic response, this study seeks to support the development of more standardized, evidence-based guidelines for managing PH in this vulnerable population.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O6.06 Deep Vein Thrombosis and Thrombus Resolution

Presenter: Shaiza Mansoor[1]
Mentor: Brajesh Lal, MD [2]
Other Co-Author(s): Georges Jreij, MD [2]; Minerva Carlin [2]
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Deep vein thrombosis (DVT) can result in long-term, serious consequences such as post-thrombotic syndrome and limb-loss, a consequence of incomplete thrombus resolution. While the standard of care (SOC), anticoagulation therapy, mitigates pulmonary embolism (PE), it does not impact thrombus resolution within veins. Residual thrombus may lead to venous reflux and obstruction leading to increased pressure in distal veins, which can result in post thrombotic syndrome (PTS) in 20-25% of patients. Studies have shown that rapid thrombus resolution decreases the potential of PTS; therefore, we are focusing on increasing thrombus resolution. The purpose of this study is to determine if exercise improves outcomes in acute DVT by increasing thrombus resolution via increased venous flow. We first tested whether exercise improves thrombus resolution and then determined the contribution of venous hemodynamics to thrombus resolution. Patients with acute DVT of the lower extremity in the past 30 days were included in the study. 97 patients were randomized 1:1 to the control group, which received SOC, and the experimental group, which received SOC plus 30 minutes of exercise 3 times a week for 3 months. 3-D duplex ultrasound assessments were performed at baseline and then at follow-up at 1 month, 3 months, 6 months, 1 year, and 2 years. We are determining the relative contribution of flow to thrombus resolution using regression and mediation analysis. Data analysis is currently ongoing. Preliminary data with 5 patients indicates that exercise does result in thrombus resolution with acute DVT.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research, NIH Grants (NS080168, NS097876 and AG000513), and Veterans Affairs Merit Awards (HSR&D C19-20-407, RR&D RX000995 and CSR&D CX001621).

O7.01 SARM1 Drives Schwann Cell Dysfunction and Axonal Degeneration following Peripheral Nerve Injury

Presenter: Abel K. Lindley, BS[1]
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Peripheral nerve injury (PNI) often leads to long-term sensorimotor deficits, with current treatments offering limited recovery. Schwann cells (SCs) play a critical role in supporting axonal regrowth and remyelination but are vulnerable to metabolic stress, which activates SARM1, a pro-degenerative protein that drives axonal degeneration. Differentiated adipose-derived stem cells (dADSCs) support neuroregeneration by acquiring SC-like properties and modulating the neural microenvironment. This study investigates the effects of FK866 (a SARM1 activator)-induced metabolic stress on SCs and peripheral nerve regeneration, including: (1) the neurotoxic impact of FK866, (2) the neuroprotective potential of dADSCs using an in vitro SC injury model, and (3) a rat PNI model with or without FK866 (PNI vs. FK866+PNI). In vitro, cell viability and SC marker expression were assessed via MTT assays and immunofluorescence staining. In vivo, Wistar rats were treated with PBS or FK866 after sciatic nerve crush injury (n=6). FK866 was administered intraperitoneally plus a direct nerve injection. Functional recovery was monitored over four weeks using CatWalk XT Gait Analysis. Gastrocnemius muscle atrophy was evaluated by H&E staining, with quantification of the gastrocnemius muscle weight index and collagen deposition. Sciatic nerve regeneration was analyzed via immunostaining for NF200, MBP, SARM1, and S100. FK866 reduces SC viability and myelination markers in vitro. dADSC treatment mitigated these effects and reduced SARM1 levels under FK866 exposure. FK866+PNI rats showed greater muscle atrophy and impaired functional recovery compared to PNI only. These findings highlight the role of SARM1-mediated metabolic stress in driving SC dysfunction following PNI.

This research was supported in part by the National Institute of Neurological Disorders and Stroke (NS117102 to X. J.). A.L. was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O7.02 Characterizing Symptoms of Trichomoniasis

Presenter: Annabella Rinaldi
Mentor: R. Gentry Wilkerson, MD

Background: Trichomoniasis is a sexually transmitted disease caused by the parasite *Trichomas vaginalis*. Diagnosis of trichomoniasis is not reportable, therefore estimates of incidence and other epidemiological data are incomplete. Data from 2018 suggests that the annual incidence of infection in the United States was greater than 2.5 million cases (Kreisel et al. 2018). Additionally, making the diagnosis is challenging due to the diagnostic tools available to most clinicians. Previous research compared clinician-performed wet mount testing to nucleic acid amplification testing for the detection of *T. vaginalis* in female patients presenting to University of Maryland Medical Center Urgent Care with vulvovaginal complaints. The sensitivity was found to be 21% with a 98% specificity.
Objectives: Improving the selection of patients who are at higher risk for *T. vaginalis* based on presenting symptoms could help control the costs of care. Trichomonas is characterized as a disease with a variety of symptoms, with little research on correlation between symptoms and diagnoses. As a secondary aim, we also measured the association of these symptoms to gonorrhea and Chlamydia in hopes to improve the specificity of the empirical treatment for these diseases.
Methods: Using data from this previous research, which included 443 subjects who presented with a variety of clinical symptoms, we measured the association of these symptoms to the presence (or absence) of disease (trichomoniasis). Additionally, throughout data analysis the prevalence of empirical treatment for gonorrhea and Chlamydia (other diseases tested as part of routine care in the patient population) was 198 and of those, 14 tested positive for one of these diseases. This further indicates the need to better understand the symptoms of gonorrhea and Chlamydia.

This research was supported in part by UM Scholars at SOM (University of Maryland scholars at School of Medicine).

O7.03 Early Preeclampsia Risk Assessment: A Window into Congenital Heart Defect Development

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Mentor Name(s): Shifa Turan MD, RDMS[1]
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Preeclampsia (PE) is a pregnancy-specific condition characterized by hypertension and signs of organ dysfunction developing after 20 weeks of gestation[1]. While its maternal risks are well recognized, its potential relationship with fetal congenital heart disease (CHD) remains underexplored. Both PE and CHD share overlapping embryological origins and developmental pathways, suggesting a possible association that may be underappreciated in clinical practice. PE affects approximately 2%–8% of pregnancies worldwide, and CHD remains the most common congenital abnormality and a leading cause of neonatal morbidity and mortality[2,3,4,5]. Despite this plausible link, PE is not currently listed among the standard indications for referral for fetal echocardiography, meaning affected pregnancies are not routinely screened for CHD unless other risk factors exist[6]. With recent advances, first-trimester risk prediction models for PE, such as the Fetal Medicine Foundation (FMF) triple test at 11–14 weeks, can now identify pregnancies at elevated risk for PE early in gestation. This raises the question of whether these same risk scores could be leveraged to detect fetuses at increased risk of CHD, improving early diagnosis and management. To investigate this, we propose a retrospective cohort study of approximately 5,100 pregnancies screened for PE risk in the first trimester. Participants will be stratified into high- and low-risk PE groups to evaluate the incidence of CHD and assess whether incorporating first-trimester PE risk assessment could add value as an indication for targeted fetal echocardiography in the second trimester. Findings may inform more comprehensive screening strategies that reflect shared developmental origins.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O7.04 Patient Satisfaction with IUD Insertion with IUD Pain Management Option Checklist

Presenter: Lauren Jacobs[1]
Mentor: Jessica Lee, MD, MPH[1]
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Pain during intrauterine device (IUD) placement is a common barrier to uptake, yet patients are rarely informed of available anesthesia options prior to the procedure. This study aimed to evaluate the implementation of an anesthesia checklist to facilitate shared decision-making and improve patient satisfaction with IUD insertion. In this single-arm study, patients presenting for IUD placement were offered a standardized anesthesia checklist outlining available pain management options. Participants completed a pre-procedure survey assessing previous IUD experience, pain expectations, mental health, and satisfaction with care. To date, 10 participants have enrolled. The sample represents a wide age range (20–46). Most (80%) had previously been pregnant, and 40% had prior IUD placement. Pain expectations and anxiety levels varied widely, suggesting individualized needs. Among those who received the anesthesia checklist, all reported that it improved their satisfaction and that they would recommend it to others. Across all participants, satisfaction with care was high. Most rated providers as friendly and felt they had adequate time with them. Enrollment is ongoing to increase the sample size and allow for more definitive analysis. Preliminary findings suggest that the anesthesia checklist is a simple, acceptable tool that may enhance patient autonomy and satisfaction during outpatient gynecologic procedures.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O7.05 Determination of the Rate and Accuracy of Hypertensive Disorder Diagnosis in Pregnant People with Opioid Use Disorder

Presenter Name: Ashwini Pugazhendhi[1]
Mentor(s): Katrina Mark, MD, FACOG[1]
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Hypertensive Disorders of Pregnancy (HDP) are among the most common complications during pregnancy and postpartum and can occur in up to 10% of pregnancies. HDPs can include chronic hypertension, gestational hypertension, preeclampsia and eclampsia, and are usually diagnosed during prenatal care. Opioid Use Disorder (OUD) can involve the use of heroin, other illicit opioids, or legally prescribed opioids that are diverted or misused. Opioid withdrawal during pregnancy can present with autonomic symptoms, such as hypertension, which may overlap with the clinical features of HDPs and complicate accurate diagnosis and treatment. Management for HDPs typically includes antihypertensives; however, patients experiencing acute opioid withdrawal can benefit from both symptom-based therapy (ie., antihypertensives) and OUD pharmacological therapy. This study aims to assess the accuracy of hypertensive disorder diagnoses in pregnant individuals with OUD. This study will retrospectively determine the frequency, accuracy of diagnosis, and treatment practices for hypertension in pregnant patients with OUD. We will use chart reviews of pregnant patients with an OUD diagnosis admitted to the University of Maryland Medical Center from 2018 to 2024. We hypothesize that individuals with OUD may be more frequently diagnosed with HDPs, which may represent a potential miscategorization of opioid withdrawal symptoms. The findings of this study aim to improve clinical differentiation between HDPs and opioid withdrawal and can help inform future large-scale studies aimed at developing clinical guidelines for diagnosing and treating hypertension in pregnant patients with OUD.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research

O8.01 Investigating immune markers correlated with intestinal permeability

Presenter: Lina Teka Berhaneyessus[1, 2]
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Necrotizing enterocolitis (NEC) is a life-threatening gastrointestinal disease that affects approximately 7–10% of preterm infants. The disease's key contributing factor is the compromised intestinal barrier, or "leaky gut", which allows bacteria to traverse the gut wall, triggering severe inflammation, infection, and intestinal tissue necrosis. With a 50% mortality rate for NEC, there is an urgent need for early diagnostic tools and a deeper understanding of the factors that contribute to intestinal barrier development. In an immature intestinal barrier, microbe-gut interactions cause an inflammatory cascade characterized by an excessive, innate immune response; however we don't fully understand its exact patterns. Upon further understanding, immunological markers associated with gut-barrier injury could be potentially used as early diagnostic tools for high intestinal permeability (IP) leading to NEC. In addition to premature birth and high IP, other known risk factors for NEC include low gestational age, antibiotic exposure, absent or decreased anaerobic and facultative bacteria, and the lack of enteral exposure to maternal breast milk. In our study, we will use ELISA to analyze fecal samples from newborns for immune markers correlated with elevated IP as well as other NEC risk factors, as their relationships reflect the intestinal immune environment and maturation. Our study seeks to contribute to finding potential diagnostic biomarkers for early-stage NEC and elucidate how the intestinal barrier matures, ultimately advancing NEC prevention.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

O8.02 Genomic Specialization of Bifidobacterium for Human Milk Oligosaccharide Utilization in Preterm Infant Gut Maturation

Presenter: Margaret Kato[1]
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Preterm infants are born with an underdeveloped gut and elevated intestinal permeability (IP), predisposing them to life-threatening infections such as necrotizing enterocolitis and late-onset sepsis. Establishing a functional gut barrier soon after birth is essential, and previous studies, including ours, revealed breastmilk feeding is the key driver of this process. Human milk oligosaccharides (HMOs), a major component of breastmilk, are abundant and indigestible by the infant alone, selectively promote the growth of *Bifidobacterium*. Our previous studies identified *Bifidobacterium breve* as the only microbial biomarker associated with lower IP in preterm infants. However, the biological processes underlying this association remain incompletely understood. To address this gap, we performed a comparative genomic analysis of 15 *Bifidobacterium breve* strains isolated from stool samples of breastfed preterm infants with improved gut barrier functions. The genomes were pre-processed, assembled, annotated, and analyzed alongside 105 publicly available *B. breve* genomes. Pangenomic analysis identified 859 genes conserved across all 120 samples, comprising just 46% of genes in the average *B. breve* genome and indicating a highly versatile genome. Phylogenetic and pangenomic analyses revealed strain-specific gene clusters involved in HMO metabolism, including glycosyl hydrolases and transport systems, which likely confer a competitive advantage in the preterm gut environment. Because breastmilk is often insufficient or absent following preterm birth, defining the specialized HMO-assimilation mechanisms of *B. breve* that support intestinal barrier development provides a foundation for developing targeted live biotherapeutics. Such intervention could enhance postnatal gut maturation and prevent leaky gut-associated morbidities in this vulnerable population.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State, NIH NIDDK, and NICHD.

O8.03 Using STING agonists to reshape tumor environment in hepatocellular carcinoma, to improve response of anti-PD1 immunotherapy

Presenter: Angela Yang
Mentor: Daniel Shu, MD[1]
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Primary liver cancer is the sixth most diagnosed cancer and the third most common cause of cancer-related mortality worldwide. Hepatocellular carcinoma (HCC) is the most common cause of primary liver cancer, and its prevalence is rising. While immunotherapy has been shown to be a clinically effective treatment for patients with HCC, response rates remain as low as 30%, suggesting a need to improve treatment options. Here we will use a humanized mouse model of HCC to evaluate the effectiveness of combined inhibition of the programmed death 1 protein (anti-PD1), an established treatment, with a novel lipid-nanoparticle bound activator of the stimulator of interferon genes pathway (uniSTING). Response to treatment will be evaluated by measuring tumor volume using Gaussia luciferase assay and confirming with caliper measurements, as well as by visualizing histologic changes in tumor microenvironment associated with treatment, including the presence of tertiary lymphoid structures (TLSs), which are immune cell clusters that have previously been shown to be good prognostic markers after treatment. We will use immunofluorescence staining to identify TLSs and count the number of TLSs formed in each tumor, and we will use ImageJ software to count how many immune cells each TLS has. These studies will provide insight into whether uniSTING may improve clinical responses to anti-PD1 immunotherapy in patients with HCC.

O8.04 Primary and Revision Hip Arthroscopy Patients Report Similar Outcomes 2-years After Surgery

Presenter: Tyler Przygocki, BS[1]
Mentor(s): Sean Meredith, MD[1]
Other Co-Author(s): Joseph Blommer, MD[1]
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Background: Hip arthroscopy is a minimally invasive surgical procedure for patients with femoroacetabular impingement (FAI), hip micro-instability, and labral pathology. Within the last two decades the overall incidence of hip arthroscopy procedures performed within the U.S. has increased by over 600%. Despite the significant increase in utilization of hip arthroscopy, there is limited understanding of which patient characteristics, surgical indications, and arthroscopic procedure types predict better 2-year patient reported outcomes (PROs) following surgery.

Objectives: The purpose of the study was to determine differences in 2-year patient-reported outcomes between patients under primary versus revision hip arthroscopy. The hypothesis was that primary hip arthroscopy would result in better pain, physical function, and mental health outcomes. Secondary aims were to investigate the effects of preoperative patient characteristics, indications, and operative details.

Methods: 146 eligible patients treated with either primary or revision hip arthroscopy for femoracetabular impingement and/or labral tear between October 2015 and December 2022 were identified from a prospective orthopaedic registry, with 105 completing 2-year surveys (72%). A retrospective chart review was conducted to assess operative details and surgical indications. PROs included Patient-Reported Outcomes Measurement Information System (PROMIS) domains, pain scales, and activity scores collected at baseline and 2-years postoperatively. Statistical analysis was performed to compare primary and revision hip arthroscopy groups.

Results: The study included 96 primary and 9 revision hip arthroscopy patients. The revision group was more likely to be current smokers, use preoperative narcotics, have previous orthopaedic and any surgery ($p<0.05$). The revision group had worse preoperative PROMIS social satisfaction than the primary group. The primary group was more likely to undergo surgery for a labral tear. Revision patients reported lower social satisfaction and tended to have higher fatigue and pain at baseline than primary patients. There were no differences in 2-year PROs or change in PROs between the two groups ($p<0.05$).

Conclusions: In patients undergoing hip arthroscopy, revision patients had worse baseline psychosocial PROs than primary patients, but similar PROs at 2 years postoperatively. These findings suggest revision hip arthroscopy can be expected to result in similarly good outcomes to primary surgery and may help surgeons to better counsel patients undergoing revision hip arthroscopy.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O8.05 Mobile is the Goal: Validating Functional Outcomes and Return to Baseline Activity with Apple Health Metrics in Patients Undergoing ACL Reconstruction

Presenter: David Diep[1]
Mentor(s): Jonathan Packer, MD[1]
Other Co-Author(s): Joseph Blommer, MD[1]; Brian Shear, MD[1]
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Primary goals of most orthopaedic procedures include improvement in pain, function, and quality of life. Significant improvement in these metrics, as measured by patient-reported outcomes (PROs), has been previously shown after anterior cruciate ligament (ACL) reconstruction. Although PROs can capture patient recovery at discrete postoperative timepoints, surgeons currently lack a comprehensive understanding of a patient's day-to-day postoperative functional recovery. In the current age of technology, wearable health technology has become increasingly common. Perhaps the most widely available health data can be found in the health app on our smartphones. Apple Health data includes various validated mobility measures, including number of steps, walking distance, and gait imbalance. These metrics can be used alongside PROs to illustrate a more comprehensive understanding of a patient's postoperative recovery. The study aims to evaluate whether gait measurements from Apple Health mobility data correlate with PROs after ACL reconstruction. We hypothesize that Apple Health data, such as step count, distance walked, and gait imbalance, will accurately detect changes in function during the postoperative recovery of ACL reconstruction. Patients were screened and either downloaded the novel application OrthoSteps or exported Apple Health data to secure servers at follow-up appointments. The app OrthoSteps facilitates uploading Apple Health data to secure servers throughout a patient's postoperative recovery. Patients then filled out a survey asking them about their Apple iPhone use and physical health. To date, data collection is still occurring, and there are 13 patients enrolled in the study.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O8.06 Private Vehicle vs. Ambulance for Pediatric Orthopaedic Transfers: A Retrospective Analysis

Presenter: Daniel Polsky[1]
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Interhospital transfers are vital for delivering specialized care to pediatric patients with orthopedic injuries. However, the choice of transport modality (ambulance versus private vehicle) may significantly impact patient outcomes, system efficiency, and costs. While prior studies have explored transport-related outcomes in adult and trauma populations, limited data exist for pediatric orthopaedics. This study aims to evaluate and compare the effectiveness, safety, and logistics of ambulance versus private vehicle transfers in pediatric patients referred to a tertiary care orthopedic center. We conducted a retrospective chart review of patients aged 0–17 who received interhospital transfers to the University of Maryland Medical Center between 2012 and 2023. Patients were identified through ICD-9 and ICD-10 codes linked to orthopedic diagnoses. Extracted variables included transport type, timing of transport and triage, pain scores, diagnosis, management, complications, and functional outcomes. Data was de-identified and stored securely per HIPAA standards. We hypothesize that private vehicle transfers may be associated with shorter transport times and reduced costs, while maintaining similar outcomes to ambulance transfers. By identifying differences in efficiency and safety, our findings aim to provide evidence-based guidelines that optimize pediatric transfer protocols, reduce unnecessary EMS use, and support shared decision-making with families. This work addresses a critical gap in pediatric emergency care literature and may contribute to resource optimization in hospital systems.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O9.01 Investigating the Functional Role of WNT5A in Prurigo Nodularis

Presenter: Joshua Wilmer[1]
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Prurigo Nodularis (PN) is a chronic inflammatory skin disorder marked by intensely pruritic nodules and an itch-scratch cycle that exacerbates neural sensitization. Affecting approximately 72 per 100,000 individuals in the U.S., disproportionately patients of color, PN has limited FDA-approved therapies. Fibroblasts in lesional PN skin have been shown to adopt a cancer-associated ($WNT5A^+$, $POSTN^+$) phenotype, and patients with this profile may be at higher risk for other fibroblast-driven malignancies. Machine learning analysis of dorsal root ganglia (DRG) identified two itch-specific neuronal clusters with enriched WNT5A signaling, suggesting a potential fibroblast-to-neuron communication axis mediated by WNT5A.

We hypothesize that WNT5A is overexpressed and secreted by PN lesional fibroblasts, driving extracellular matrix remodeling, angiogenesis, and proinflammatory cytokine production. Additionally, we propose that WNT5A enhances peripheral sensory neuron excitability through noncanonical signaling pathways, contributing to chronic itch.

To investigate this, we performed Western blotting on PN lesional fibroblasts and their conditioned media. Intracellular WNT5A was reduced in PN cells, but secreted levels were equal or elevated compared to controls, indicating increased extracellular release. To evaluate functional effects on neurons, mouse DRGs were cultured and labeled with Cal-520 AM, a calcium-sensitive dye. Neurons were stimulated with recombinant WNT5A, capsaicin, or conditioned media from PN fibroblasts. Preliminary imaging showed elevated intracellular calcium changes of neurons after adding patient derived conditioned media from diseased fibroblasts as compared to healthy controls.

These findings suggest WNT5A contributes to PN pathogenesis via fibroblast activation and sensory neuron sensitization, revealing a potential therapeutic target for chronic itch.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O9.02 Expression of Growth Differentiation Factor 6 (GDF6) in Acral Melanoma

Presenter: Alisa Forsberg[1]
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Acral melanoma (AM) is a distinct clinical subtype of melanoma comprising 2-5% of all melanomas. Unlike cutaneous malignant melanomas (CMMs) which occur on hair-bearing skin, AM arises on the palms, soles, fingers, toes, and nailbeds. Compared to CMM, AM has worse survival rates and a lower level of ultraviolet radiation-induced mutations.

GDF6 was identified as a gene of interest in melanoma because it is amplified in both zebrafish and human melanomas. In CMMs, GDF6 signals through the bone morphogenetic pathway (BMP) and is associated with tumor progression. GDF6 represses the transcription factors MITF and SOX9 and promotes expression of SOX10, a melanoma marker, to maintain melanoma cells in a less-differentiated state.

We tested the hypothesis that GDF6 is also expressed by AM and is correlated with SOX10 expression in these tumors.

In 2/2 CMMs analyzed by GDF6/SOX10 Immunohistochemistry, areas of invasive melanoma cells showed a strong correlation between SOX10 and GDF6 expression, confirming prior findings. 5 AM specimens representing 2 primary lesions and 3 metastatic lesions were studied. 5/5 AM specimens from 4 distinct patients expressed GDF6 to varying degrees; where the highest levels of expression were noted in a metastatic lymph node specimen.

Studying AM tumorigenesis may provide more insight into potential AM therapeutic targets to improve its lower survival rate.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research, the DoD CDMRP W8IXWH2010288P00001 (subaward to Thomas J. Hornyak), and by the Baltimore Research and Education Foundation (BREF).

O9.03 Comparison of Outcomes in Transperineal vs. Transrectal Prostate Biopsy in the VA Patient Population

Presenter: Christopher Huang[1]
Mentor: Mohummad Minhaj Siddiqui, MD[1]
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BACKGROUND: Prostate cancer is the most common cancer and the second leading cause of cancer death for men worldwide. Men undergoing prostate cancer screening will be encouraged to perform a prostate biopsy for confirmation. Traditionally, the Transrectal (TR) prostate biopsy has been the gold standard for diagnosis, however, recent concerns about infectious complications (especially sepsis) associated with penetration of the rectal wall has driven urologic surgeons to explore alternative approaches. The Transperineal (TP) prostate biopsy has emerged as a viable alternative to TR that can be performed freehand under local anesthesia, and may be associated with lower infectious risk due to avoiding the rectal wall entirely. In addition, studies have suggested that TP has a noninferior cancer detection rate vs. TR, and may be more effective at detecting anterior or apical prostate tumors. In particular, the veteran patient population is an understudied and underserved group that may be uniquely susceptible to various complications associated with prostate biopsy.

OBJECTIVE: This research project aims to determine whether freehand Transperineal (TP) prostate biopsy vs. Transrectal (TR) prostate biopsy results in significantly different clinical outcomes (including cancer detection rate, infectious, as well as non-infectious complications) in the VA patient population.

METHODS: This project will utilize chart review and data analysis to perform a retrospective cohort study examining the prostate cancer detection rate, as well as various infectious and non-infectious outcomes associated with TP vs. TR prostate biopsy within the patient population at Baltimore VA Medical Center.

RESULTS: While still pending final results, preliminary data analysis on the first 82 patients (51 TR, 31 TP) suggests that rates of prostate cancer detection between TP and TR are similar (45.1% in TR vs. 54.84% in TP). In addition, rates of adverse infectious as well as non-infectious outcomes in both TP and TR are similarly low, with one case of sepsis in each cohort (1.96% in TR vs. 3.23% in TP), one case of urinary retention in TR (1.96%), and two cases in TP (6.45%).

CONCLUSIONS: Preliminary results suggest that TR and TP have a comparable prostate cancer detection rate and infectious risk, while TP is associated with a higher risk of urinary retention in the VA patient population. True outcome rates will be better approximated by screening more patients.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O9.04 An Analysis of Socioeconomic Deprivation and Cancer Trial Enrollment at the University of Maryland's Greenebaum Comprehensive Cancer Center: A Retrospective Cohort Study

Presenter: Priyanka Ravi, BS[1]
Mentor: Benjamin Powers, MD, MS[1]
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Introduction
Lack of diversity in cancer clinical trials remains a major barrier to equitable treatment advancements. However, most studies have focused on race/ethnicity or gender disparities in cancer trials. Although socioeconomic status has been described as a barrier to cancer clinical trial participation, few studies have assessed it using granular, comprehensive, or normalized measures. Therefore, this study used the Area Deprivation Index (ADI) to assess cancer clinical trial eligibility and enrollment at an NCI-designated Comprehensive Cancer Center.

Methods
Patients approached for cancer clinical trials (2010–2024) were identified from the University of Maryland Greenebaum Cancer Center OnCore database. Inclusion criteria were age ≥18, a cancer diagnosis, and available ADI. The ADI is a validated, publicly available dataset ranking census block groups (1–100), with 100 indicating greatest socioeconomic disadvantage. Descriptive statistics and linear regression were performed using R (v4.4.2).

Results
Patients eligible but not enrolled in a trial comprised 4.0% of the analytic cohort. Non-enrollees were 4.5% of the least deprived and 2.4% of the most deprived cohorts. The analytic cohort included 6858 patients. The median age was 59; 52% were female. By race, 61.2% percent were White, 32.6% African American, and 3.0% Asian. The cohort was 3.0% Hispanic. ADI was associated with 5 variables. The median national ADI of the cohort was 33. By national ADI, the least and most deprived groups comprised 28.3% and 8.5%, respectively. By state ADI, these groups comprised 18.0% and 21.5%.

Conclusion
These results are the first assessment of cancer clinical trial enrollment at an NCI-designated cancer center. The findings suggest inequity in cancer clinical trial enrollment at the national level; however, there is no clinically meaningful difference in enrollment at the state level. Several covariates were associated with ADI and evaluation suggesting that the intersection of socioeconomic status and other demographic factors may impact cancer clinical trial enrollment.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

O9.05 Ultrasound-mediated Drug Activation for the Treatment of Infiltrating Gliomas

Presenter: Renee Jaranson[1,3]
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Sonodynamic therapy (SDT) has emerged as a promising noninvasive modality for treating high-grade gliomas, including glioblastoma. SDT utilizes ultrasound to activate otherwise nontoxic drugs termed 'sensitizers', which in turn generate cytotoxic reactive oxygen species (ROS). Although SDT is currently being used in clinical trials, the underlying mechanisms behind ROS generation remain elusive, raising concerns for efficacy. The two prevailing theories behind sensitizer activation, sonoluminescence and pyrolysis, rely on bubble formation and cavitation during ultrasound exposure. Sonoluminescence (e.g., light emitted from collapsing bubbles) may activate sensitizers, while pyrolysis (heat-mediated sensitizer breakdown) may lead to the formation of radicals.

We have characterized an agarose-based brain tissue-mimicking phantom to investigate bubble cavitation dynamics, sensitizer activity, and singlet oxygen generation during SDT, all of which could contribute toward understanding the mechanism. At various sensitizer concentrations and focused ultrasound parameters, we have not observed significant singlet oxygen radical generation, suggesting that sonoluminescence may not be the mode by which sensitizers are activated. Sensitizer-mediated fluorescence readouts remained at similar levels pre- and post-SDT, indicating that sensitizers remain intact and that pyrolysis does not likely occur.

We hypothesize that increasing the concentration of sensitizers may contribute to increased bubble formation and lower cavitation thresholds, leading to more radicals associated with inertial cavitation (e.g., non-stable bubble oscillation). We are also investigating how sensitizers affect the rate of heating in phantoms, which may lead to hyperthermia effects.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

O9.06 Practical Clinical Interventions to Help Improve Cancer Care for Patients Who Experience Food Insecurity

Presenter: Delara Rajabi Abhari, MPH[1]
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Patients undergoing definitive chemoradiation (CRT) for head and neck, gastrointestinal, thoracic, or gynecologic malignancies often face nutritional challenges in the months following treatment, which can contribute to long-term health complications and reduced quality of life. Food insecurity and poor dietary intake during this recovery period may go unrecognized, leading to weight loss, diminished functional status, and increased symptom burden. This pilot study aims to proactively identify patients at increased risk for food insecurity 1–3 months post-CRT and provide them with cost-effective nutritional resources alongside individualized dietary education. The primary objectives are to evaluate the feasibility of early intervention and to assess its impact on food security, BMI/weight stability, and overall symptom burden. We hypothesize that we can achieve ≥ 85% retention among enrolled participants and demonstrate improvements in post-treatment nutritional outcomes. We are conducting a small, single-arm, interventional study. Eligible patients are recruited 1–3 months after completing CRT and will receive a structured nutritional support program lasting 6 months. Data will be collected through validated short-form surveys administered at baseline (time of consent), during the intervention period, and post-intervention. Surveys will capture BMI, self-reported symptom burden, and measures of food insecurity. The intervention will consist of regular contact with a dietitian, access to nutritious and affordable food options, and education tailored to each patient's dietary needs and treatment history. This study will inform future strategies to address nutritional insecurity as a critical, modifiable factor in cancer survivorship care.

This research was supported in part by the Radiation Oncology Medical Student Summer Fellowship Program and the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

P1.01 Targeting Medulloblastoma Cells with Novel Therapeutic Compound VNPP-433.3β.

Presenter: Gabrielle Olibris[1]
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Medulloblastoma is a highly aggressive pediatric brain tumor, accounting for approximately 20% of all childhood brain cancers. About 30% of cases relapse, with a survival rate of less than 5% in recurrent cases. Current treatments, including surgery and radiation therapy, often lead to severe side effects and limited long-term success, highlighting the urgent necessity for novel therapeutic strategies. Our previous studies demonstrated that the compound VNPP-433.3β effectively inhibits the growth of medulloblastoma cell lines, DAOY and D341, and medullospheroids by inducing apoptosis and suppressing the expression of the oncoprotein FoxM1 and phospho-β-catenin, both of which are normally overexpressed in medulloblastoma. This study further investigated the molecular mechanisms underlying this drug's effects. At a dose of 1 μM of VNPP-433.3β for 48 hrs, increased cleaved caspase 3, CHOP protein levels, and a decreased BAX/Bcl-2 ratio indicated activation of the apoptotic pathway, which is associated with reduced ATP levels. Additionally, BrdU incorporation assays using immunofluorescence showed a significant reduction in cell proliferation. RNA-sequencing demonstrated the involvement of cell cycle-regulating proteins such as cyclin D1, cyclin B1, and cyclin A. The gene and protein expression of cyclin D1, cyclin B1, and cyclin A was validated using qPCR and immunoblotting, respectively. Our results demonstrated that gene and protein expression were significantly ($p<0.05$) downregulated in the drug-treated cells compared to untreated cells. This data indicates that VNPP-433.3β induced apoptotic cell death through the involvement of the cyclin-dependent pathway, supporting its potential as a promising therapeutic agent for medulloblastoma.

This work was supported in part by the American Cancer Society- DICR INTR-23-1253710-01-DICR INTR (Tonya J. Webb), National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P1.02 Hijacking the Endogenous Repair Mechanisms of DNA to Cure Genetic Disorders

Presenter: Maawiah Mohammed[1]
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CRISPR-Cas9 machinery relies on double-strand DNA breaks (DSBs) to induce edits in a gene of interest. In mammalian cell lines, non-homologous end joining (NHEJ) is the primary repair pathway for DSBs. NHEJ is an error-prone pathway which results in insertion-deletion mutations in a DNA sequence. In repeat-expansion diseases like Huntington's disease, mismatch repair (MMR) is an error-prone repair pathway which results in further pathology of repeat-expansions. The mechanism by which MMR results in increased mutations is strand slippage which introduces loops or extra helical extrusions in DNA leading to a greater number of pathological repeats. In contrast to these error prone repair pathways, homology-directed repair (HDR) utilizes a homologous DNA sequence as a template for precise mutations and knock-in at the gene of interest; however, HDR is limited in post mitotic neurons. Utilizing a high throughput modular cloning workflow, we propose developing CRISPR based fusion editors that bias the endogenous DNA repair pathways to allow for error free repair by either enhancing HDR or inhibiting the NHEJ/MMR. We hypothesize using DNA repair proteins like FANCI associated nuclease 1 (FAN1), dominant negative MLH1 (dnMLH1), and UPF1 RNA helicase and ATPase (UPF1) to develop Cas9 fusion editors will allow the bias of HDR and the inhibition of MMR and NHEJ. These fusion editors will be tested for genome restoration efficacy in various Huntington's disease models.

This research was supported in part by the Nathan Schnaper Intern Program in Translational Cancer Research, University of Maryland School of Medicine Office of Student Research.

P1.03 Investigating the antileukemic mechanism of action of ART838 in acute myeloid leukemia (AML)

Presenter: Amaka Chukwura[1]
Mentor(s): Curt Civin, MD, ScD[1]; Ye Jun Kim, BA[1]
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Previous studies in our lab have shown that the eIF2apha-kinase heme-regulated inhibitor (HRI), a core sensor in the activation of the Unfolded Protein Response (UPR), is necessary for the mechanism of action (MOA) of the antileukemic drug ART838 in THP1 acute myeloid leukemia (AML) cells. However, whether HRI is sufficient for ART838's antileukemic MOA is unknown. To investigate this, we used lentiviral transduction to generate THP1 cell lines genetically overexpressing UPR pathway genes HRI, EIF2alpha, IRE1, or CHOP, and we evaluated whether HRI, HRI's downstream effector molecules, and/or a separate UPR sensor IRE1 are genetically sufficient for ART838's antileukemic MOA. ART838 sensitivity in THP1 cells was assessed by alamarBlue cell viability assays. Western Blots were utilized to investigate the expression of key UPR pathway proteins induced by ART838 treatment. THP1 EIF2alpha and IRE1 OE cells displayed no substantial differences in sensitivity to ART838 treatment compared to wild-type and empty vector (EV) transduced THP1 cells, suggesting that EIF2aplha and IRE1 are not sufficient for ART838 sensitivity/antileukemic efficacy. Western blot data has been collected and will be analyzed to determine whether IRE1 or EIF2alpha OE impacts ART838-mediated changes in gene expression. These results suggest that the ART838 antileukemic MOA involves the HRI pathway, but its function is independent from EIF2alpha. Additionally, the IRE1 results suggest that the HRI pathway may be the only relevant UPR pathway in the ART838 MOA.

This work was supported in part by the American Cancer Society- DICR INTR-23-1253710-01-DICR INTR (Tonya J. Webb), National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P1.04 Investigating Cytokine Influence on Tumor Dynamics in Ovarian Cancer Cell Models

Presenter: Karem Sandagorda
Mentor(s): Kyla Roland, PhD Candidate; Tonya Webb, PhD

Ovarian cancer, the fifth leading cause of cancer-related death in women, is notoriously difficult to detect in its early stages. Due to a lack of identifiable symptoms, patients are often diagnosed at a late stage, which contributes to chemoresistance, increased mortality, and a reduced five-year survival rate. Current 2D cell models aid researchers in investigating the dynamics and complexity of tumor microenvironments. However, they fail to accurately replicate the environment of the tumor within a human. In comparison, 3D cellular models more accurately mimic the structure of a cancerous mass, and potential therapies can be tested within a laboratory before initiating animal studies. As vital components of the immune system, cytokines also play a role in cancer development and treatment. Some promote anti-tumor responses while others may contribute to their growth and resistance to therapy. Understanding their role within the ovarian cancer microenvironment can provide information on how to develop more effective targeted cancer therapies. In addition to cytokines, other genetic factors may contribute to the differences in treatment efficacy between 2D and 3D in-vivo microenvironments by inducing drug resistance, increasing cell proliferation and promoting migration. Specifically, we hypothesize that genes such as FASN, HER2, CDK1, and Hsp70 may be differentially regulated in 2D models, compared to 3D multicellular cultures due to differences in tumor associated cytokines. In this study, we assessed the impact of IL-6, IL-10, and VEGF-A cytokine levels on ovarian cancer cell growth. The overall goal will be to investigate how these cytokines lead to genetic alterations that impact drug susceptibility in the 3D in-vivo environment that are not accounted for in 2D models.

This research was supported in part by the American Cancer Society – Internships for Mentored Projects Advancing Cancer Translation (ACS-IMPACT), University of Maryland School of Medicine Office of Student Research.

P1.05 Rewiring T Cell Signaling To Make a Better CAR-T Cell

Presenter: Nicole Brown
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Chimeric antigen receptor (CAR) T cell therapy involves using genetically engineering T cells that encode specific receptors to recognize and attack cancerous cells. This approach is highly effective in blood cancers; however, the therapy faces several limitations, most notably being the short-lived activity and exhaustion of the CAR T cells. Our hypothesis is that adjusting signaling molecules involved in the activation of T cells, can help improve and prolong the activity of CAR T cells. In this context, we selected two proteins, LAT and ZAP70, which are unique to T cell activation but are also in the early stages of the signal transduction pathway. The lab found that LAT, an adaptor protein, is cleaved in T cells but it is unclear which fragment potentiates T cell activation. Additionally, ZAP70 was found to increase in memory and tolerant T cells but levels that favor optimal function are not known. Therefore, our experimental strategy is to increase the amounts of ZAP70 or LAT isoforms in CAR T cells and evaluate changes in killing target tumor cells. We generated retroviruses expressing CD19 to generate MB49, LL2, MC38, B16 and CT26 tumor cells that can be killed by anti-CD19 CAR T cells. We then co-transduced the CAR as well as LAT constructs into mouse T cells. Ongoing experiments are comparing how LAT-modified CAR T cells kill these targets relative to control cells before combining it with ZAP70 modification. The most promising variants will be tested using in vivo models.

This research was supported in part by the American Cancer Society- DICR INTR-23-1253710-01-DICR INTR (Tonya J. Webb), National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P1.06 The Cellular Response of Bladder Cancer Cell Lines to Gemcitabine

Presenter: Grace Ayomide Hountangni
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Gemcitabine is a chemotherapy drug and the standard of care treatment against bladder cancer. Our goal is to observe the effects of gemcitabine in bladder cancer. The Seahorse and MTT Proliferation Assay were used on the various cultured cell lines that were grown to 80% - 90% confluency. The Seahorse Assay XF Glycolysis Stress Test measured the oxygen consumption rate [OCR] and extracellular acidification rate [ECAR] which allowed us to measure ATP activity and glycolysis pathways of the cell lines. Multiple MTT assays were performed with and without Hoechst dye to observe cell viability and migration. All images of assays using Hoechst dye were derived using the BioTek Cytation 1 Imaging Reader. When treated with gemcitabine, Scaber cell growth decreased by 45% and 53% for 5 μM and 10 μM concentrations, respectively. J82 responded similarly with a 40% decrease with a 5 μM treatment and a 50% decrease with 10 μM treatment. The CRC organoids also experienced at least a 21% decrease with 10 μM with gemcitabine having the biggest effect on VA0004 cell growth out of all of the organoids. Over the course of 24 hours, cell migration was inhibited in Scaber by 21%, VA004 by 42%, mBCA26 by 33%, mBCA36 by 24%, BCA418 by 17%. According to the results from the assays, gemcitabine does deplete the bladder cancer cell lines from growing, migrating, and normal ATP activity.

This work was supported in part by the American Cancer Society- DICR INTR-23-1253710-01-DICR INTR (Tonya J. Webb), National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P1.07 Transcriptional Profiling of Cancer-Related Fatigue

Presenter: Ronit Shah[1,3]
Mentor: Amber S. Kleckner, PhD[1]
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Most patients with cancer will experience cancer-related fatigue during treatment; however, there is not much to offer to address their fatigue, as treatments typically focus solely on curing the cancer. Through this research, we hope to understand the underlying mechanisms causing this fatigue to improve the quality of life during and after treatment.

Previously, the TRIXIE Study used bulk RNA sequencing (RNA-Seq) on blood cells among patients with breast cancer undergoing chemotherapy and described differences in transcriptomic profiles between people reporting high vs. low fatigue. Because blood cells are heterogeneous, we are now building on these data to explore differences in gene expression between those with high vs. low fatigue within each cell type.

We hypothesize that there are distinct differences in single-cell transcriptomic profiles of patients experiencing low fatigue vs. high fatigue. For this experiment, peripheral blood mononuclear cells (PBMCs) were isolated from whole blood samples, 4 from patients with high fatigue and 4 from patients with low fatigue, and single-cell RNA-seq was performed. The data were integrated into 32 clusters using Principal Component Analysis. The clusters were annotated using both machine learning and manual approaches to identify cell type (e.g., T cell, B cell). Differential gene expression is being run to compare the low-fatigue group to the high-fatigue group and determine whether there are distinct differences between them.

If conclusive, distinct profiles could help with understanding the biological pathways that need to be targeted to treat fatigue and identify biomarkers to predict fatigue before patients undergo chemotherapy.

This research was supported in part by the Nathan Schnaper Intern Program in Translational Cancer Research, University of Maryland School of Medicine Office of Student Research.

P1.08 In Vitro Study of Granzyme B-Dependent Antitumor Immune Response

Presenter: Mikayla Wills[1]
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Granzyme B (gzmB) is a serine protease secreted by cytotoxic T lymphocytes (CD8+) and natural killer (NK) cells that induces apoptosis in infected or cancerous cells, a process facilitated by the pore-forming protein perforin. While gzmB is primarily associated with tumor cell clearance, prior research[1] suggests that tumor-induced expression of gzmB in regulatory T cells (Tregs) may paradoxically suppress cytotoxic immune responses. Given the relative scarcity of Tregs to effector cells during immune responses, the net effect of gzmB and perforin in antitumor immunity requires further investigation. This study examined the role of gzmB and perforin in regulating B16-F10 melanoma proliferation in vitro. Co-culture experiments were performed using naive splenocytes from wild-type (WT), gzmB-knockout, perforin-knockout, and gzmB/perforin-double knockout mice, with interleukin-2 (IL-2) treatment to stimulate T cell activation. Results showed that immune cells deficient in gzmB and perforin were less effective at suppressing B16 melanoma cell growth compared to WT controls, suggesting a critical role for these cytotoxic effector molecules in mediating antitumor immunity.

P1.09 The Impact of Financial Stress on Overall Survival in Acute Myeloid Leukemia

Presenter: Tayah Reid[1]
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Acute Myeloid Leukemia is a rapidly progressive blood cancer with poor overall survival rates, and the impact of socioeconomic factors such as financial stress on patient outcomes remains to be under-explored. Patients who experience financial hardship face obstacles that affect their availability to receive proper treatment such as chemotherapy, stem cell transplants, and other supportive care, further causing health disparities in leukemia care. We performed an analysis of AML patients using a comprehensive database that highlighted patients demographics including gender, race, treatment intensity, and survival outcomes which are linked with socioeconomic indicators such as insurance type, income, and area deprivation quartiles (ADI).Area deprivation index (ADI) is a scientifically validated measure of the relative socioeconomic conditions of neighborhoods. It is created from publicly available data on income, education, employment, and housing quality. Areas with greater socioeconomic disadvantages are ranked higher. Survival curves were created to evaluate differences in overall survival. A total number of 228 leukemia patients were included. Patients in higher ADI quartiles and lower income categories exhibited shorter overall survival. Issuance status was also associated with outcomes; private insurance has improved survival compared to those with Medicare or uninsured status. Socioeconomic factors, particularly financial stress indicators such as insurance type, and ADI, significantly impact survival in AML. These findings highlight the importance and need for policy interventions to reduce disparities and improve outcomes.

P1.10 Building Molecular Tools to Identify Protein Binding Partners of AUF1 Involved in Post-Transcriptional Regulation

Presenter: Elliot Stratton[1]
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AUF1 is an RNA-binding protein that targets AU-rich elements (AREs) within RNAs and is commonly upregulated in various cancers, with both pro- and anti-tumorigenic effects depending on cellular context. AUF1 binding to an ARE-containing mRNA can induce transcript stabilization, destabilization, or enhanced translation. However, the mechanisms selecting and executing each outcome remain largely unknown. Among its four isoforms, $p37^{AUF1}$ has the greatest mRNA-binding affinity, and is thus most closely associated with post-transcriptional gene regulation. To better understand the mechanisms of $p37^{AUF1}$, our goal was to identify protein-protein interactions involved in AUF1-mediated mRNA regulation. Using $p37^{AUF1}$-BioID fusion proteins, we aimed to tag all interacting proteins in the vicinity of AUF1 for isolation and identification, while differentiating between RNA-dependent and -independent binding partners via parallel experiments with a non-RNA binding mutant $p37^{AUF1}$[C207E] protein. We began by growing and purifying His_6-$p37^{AUF1}$wt and His_6-$p37^{AUF1}$[C207E], then measured the ARE-binding affinities of both recombinant proteins using fluorescence anisotropy. We observed that the [C207E] mutation caused a 65-fold penalty in RNA-binding affinity compared to $p37^{AUF1}$wt, confirming this mutant should engage only RNA-independent binding partners. We then subcloned coding sequences for sh-Resistant $p37^{AUF1}$wt and mutant shR-$p37^{AUF1}$[C207E] into pBioID plasmids. After screening and sequencing, we transfected them into cancer cell lines with endogenous AUF1 knockdown to ensure expression. Future steps will test the fusion proteins' ability to bind and destabilize RNA. Moving forward, these proteins will be utilized to isolate other proteins interacting with AUF1 via proximity ligation experiments, revealing candidate RNA-dependent and -independent binding partners of AUF1.

This work was supported by the National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P1.11 Photodegradation Shifts Indocyanine Green (ICG) Aggregation, Modulating Fluorescence and Thermal Response

Presenter: Amogh Shetty
Mentor: Vikas Kundra

Delivering targeted thermal doses in photothermal therapy (PTT) requires precise control of heat generation to avoid damaging surrounding tissue. Indocyanine green (ICG) is a clinically approved near-infrared (NIR) dye widely used in PTT due to its strong NIR absorption, fluorescence, and efficient heat generation. In water, ICG exists in a dynamic equilibrium between monomeric and H-aggregated forms, each with distinct optical and thermal properties. Under continuous laser illumination, ICG undergoes photodegradation, decreasing its total concentration. This study demonstrates that photodegradation causes a time-dependent shift in ICG's aggregation equilibrium, favoring monomers as dye concentration decreases. Using spectroscopic and thermal measurements, we quantify how fluorescence intensity and heat output evolve under irradiation conditions. Initially, fluorescence increases as aggregates break apart, but it eventually declines due to dye degradation. Similarly, the thermal response changes over time as the distribution of absorbing species shifts. Our findings reveal that the photophysical properties of ICG are not constant during illumination; instead, its aggregation state and resulting fluorescence and heat generation change dynamically. This dynamic behavior has important implications for PTT, where consistent and precise thermal dosing is critical. Understanding these shifts is essential for improving treatment accuracy and optimizing protocols that use ICG as a photothermal agent.

This work was supported by the National Cancer Institute grant R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P1.12 Identifying antigen-driven tolerance signatures in tumor-reactive T cells

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T cells failing to swiftly eliminate routinely-arising tumors become "tolerant", losing their ability to proliferate and kill effectively. Previous studies have attributed this dysfunction to two main factors: chronic exposure to tumor antigens and the influence of the tumor microenvironment. Our project aimed to dissect the individual contributions of these two factors by identifying genes associated with each context. We used a mouse model comparing T cells chronically exposed to an antigen to those acutely stimulated once with the same antigen. T cells chronically stimulated in vivo exhibit many hallmarks of tumor-infiltrating tolerant T cells, including upregulation of PD1, LAG3, TIM3, and FR4, and impairment in proliferation and effector functions compared to memory T cells generated in acutely immunized animals. These two populations of T cells were isolated, and single-cell RNA sequencing was performed to profile gene expression. T cells (9,778 acute and 9,650 chronic) were identified via a marker-based artificial intelligence followed by manual confirmation, and were clustered and visualized using UMAP for dimensionality reduction. Analysis revealed differential abundances of T cells within specific clusters between the chronic and acute states and significant gene expression differences within those clusters. Gene expression signatures identified from this tumor-free model will next be compared to publicly available datasets of T cells exposed to both tumor antigen and the tumor microenvironment. This approach allows us to identify which changes are specifically attributable to chronic antigen exposure versus microenvironmental factors, highlighting targets for drug development aimed at preserving T cell function and improving immunotherapy.

This work was supported by the National Cancer Institute grant R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P1.13 Investigating the Impact of JAK-1 Inhibitors on Tumorigenesis Through Regulation of JAK-1 Suppressed Genes

Presenter: Kristal Chicas
Mentor: Darren Perkins

Type I Interferons (IFNs) are cytokines that play a critical role in inflammation, immunoregulation, and the body's defense against viral infections. IFNs work by halting cellular replication and signaling immune cells to respond, and are also known to directly inhibit tumor growth and induce apoptosis. Many cancers hijack the JAK/STAT signaling pathway, which regulates cell growth and differentiation, allowing cancer cells to produce cytokines and bypass normal regulatory circuits to sustain uncontrolled growth. JAK-1, a key component of this pathway, has emerged as a potential therapeutic target since inhibiting JAK-1 limits the cytokine-driven signals that cancer cells rely on for proliferation. Prior research in our lab suggests that JAK-1 may regulate a group of suppressed genes, and disrupting its activity could reveal important insights into how cytokine signaling contributes to tumorigenesis. This project aims to explore how JAK-1 and TYK-2 inhibition affect gene expression in the presence of IFNβ treatment. Two cancer cell lines, A549 (human lung cancer) and B16 (mouse skin cancer), were used as models. Changes in gene expression were assessed using qPCR, along with cell morphology, to understand how the inhibitors may impact the growth of the cell lines at certain timepoints. Western blots were performed to determine if the JAK-1 and TYK-2 inhibitors affected the phosphorylation of the transcription factor phospho-STAT1. Previous data from the lab showed IFNβ impacts p53 regulatory genes. This finding was validated in BMDMs and further explored in B16 cell lines. This project contributes to a better understanding of how JAK-1 and TYK-2 inhibition modulate gene expression and cell behavior, giving insight into potential therapeutic strategies for targeting cytokine signaling in cancer.

P1.14 Impact of Methotrexate on Motor Learning and Motor Function

Presenter: Lauren Savage[1]
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Acute lymphoblastic leukemia (ALL) is the most prevalent pediatric cancer. Methotrexate is a widely utilized chemotherapeutic agent in the treatment of children with ALL and has demonstrated efficacy in improving overall survival outcomes. Despite its success in prolonging survival in patients with ALL, methotrexate has neurotoxic properties and is known to induce chemotherapy-induced cognitive impairment (CICI), a side effect that can lead to irreversible deficits in attention, working memory, and executive function. Furthermore, these deficits can persist into adulthood, as treatment regimens are taking place during a crucial stage of brain development. Evidence suggests that chemotherapy-induced cognitive impairment (CICI), particularly from methotrexate-induced neurotoxicity, may arise from disruptions in neuroplasticity and adaptive myelination. Although methotrexate has been associated with cognitive deficits, it remains unclear whether these impairments extend to or impact motor skill learning and motor function. Accordingly, we propose that the neurobiological mechanisms underlying CICI, such as disrupted neuroplasticity, also contribute to the motor impairments frequently observed in pediatric survivors of ALL. In a literature review examining motor function in both children and adult survivors of pediatric ALL, articles were identified that examined motor outcomes in children treated with methotrexate-containing chemotherapy protocols. These studies found that both children who had recently undergone chemotherapy and adult survivors of pediatric ALL exhibited reduced motor function, particularly in areas such as knee extension strength, dorsiflexion strength, and fine motor skills, when compared to healthy controls. Additionally, studies have found that methotrexate administration has been associated with decreased brain-derived neurotrophic factor (BDNF) expression, which plays a crucial role in motor learning processes. Furthermore, it has been established that BDNF levels can be increased through exercise and rehabilitation. These findings suggest a complex interplay between methotrexate-induced cognitive impairments, motor skill acquisition, and physical activity. However, the mechanisms linking these domains remain poorly understood. Future research examining the impact of exercise on both functional outcomes and underlying neurobiological processes may help clarify these relationships and inform rehabilitation strategies for pediatric ALL survivors.

This work was supported by the National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P1.15 A comparison of various MRI acquisition methods for visualization of MRI findings critical to the new multiple sclerosis diagnostic criteria

Presenter: Anna Li
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Multiple sclerosis (MS) is a chronic autoimmune disease of the central nervous system characterized by demyelination and axonal loss, leading to progressive neurological disability. MS is driven by a complex combination of genetic predisposition and environmental triggers. The estimated 2010 prevalence of MS in the US adult population cumulated over 10 years was 309.2 per 100,000, representing 727,344 cases. Early and accurate diagnosis of MS is crucial for patients. The 2017 McDonald diagnostic criteria improved diagnostic sensitivity, but these criteria have raised concerns regarding misdiagnosis. Advanced MRI biomarkers, such as cortical lesions (CLs), the central vein sign (CVS), and paramagnetic rim lesions (PRLs), have shown promise in improving diagnostic accuracy but may not be consistently visualized with standard clinical MRI protocols.
This study aims to evaluate the diagnostic performance of specialized MRI protocols, including 7 Tesla (7T) MRI, compared to standard 3T MRI imaging for detecting MS-specific lesions. We will conduct a retrospective review of MRI images from 103 subjects – including MS patients, other neuroinflammatory diseases and healthy volunteers – who underwent both 3T and 7T MRI scans. Three independent reviewers will assess each MRI for specific MS-related lesions, including WMLs, CLs, CVS, and PRLs. The diagnostic performance of each protocol will be compared, with sensitivity and specificity calculated using established diagnostic criteria.
We hypothesize that 7T MRI will demonstrate superior lesion detection and diagnostic accuracy compared to 3T MRI. This study has the potential to guide the development of standardized imaging protocols, improving MS diagnostic accuracy and patient outcomes.

This research was supported in part by the Maryland Infection, Immunization, Intervention, and Impact (M4I) Training Program, University of Maryland School of Medicine Office of Student Research.

P1.16 Characterizing the Synergistic Immunosuppressive Effect of Mesenchymal Stromal Cells on Peripheral Blood Mononuclear Cells

Presenter: Jehan Idsassi[1]
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Background: Immune-mediated diseases such as graft-versus-host disease (GVHD) and chronic inflammatory disorders affect approximately 8% of the U.S. population, with dysregulated immune responses and abnormal activation of peripheral blood mononuclear cells (PBMCs) playing a central role in disease pathogenesis. Mesenchymal stromal cells (MSCs) possess potent immunomodulatory properties and may offer a promising therapeutic approach by suppressing PBMC proliferation and function. This study aimed to optimize a flow cytometry– based assay to evaluate MSC immunosuppressive potential in vitro.

Methods: This study was approved by the University of Maryland Institutional Review Board. Peripheral blood (15 mL) was collected from healthy donors, and PBMCs were spun down and isolated via the buffy coat method. Samples were stained with carboxyfluorescein succinimidyl ester (CFSE) to track proliferation through successive generations. PBMC proliferation was first validated using the following groups: (1) unstimulated CFSE-stained PBMCs and (2) CFSE-stained PBMCs stimulated with 0.8 µL phytohemagglutinin (PHA) and incubated for 5 days. PBMCs were analyzed by flow cytometry using the following gating strategy: singlets → live cells → CD45+ → CFSE+ PBMCs. Following validation of PBMC proliferation, MSC and PBMC co-incubation assays were performed using the following groups: (1) MSCs co-cultured with CFSE-stained, PHA-stimulated PBMCs at a 1:1 ratio (90,000 MSCs and 90,000 PBMCs per well), (2) stimulated PBMCs alone (90,000 PBMCs per well), and (3) unstimulated PBMCs (90,000 PBMCs per well). PBMCs were stained with 1.75 µL of CD45 antibody, 1.0 µL of CD73 antibody, and 2.5 µL of Fc Block to prevent nonspecific binding. Flow cytometry was performed with the following gating scheme: singlets → live cells → CD73- → CD45+ → CFSE+ PBMCs. CFSE median fluorescence intensity (MFI) and proliferation fold change were quantified using FlowJo v10.

Results: PHA-stimulated PBMCs demonstrated robust proliferation, while co-culture with MSCs significantly suppressed CFSE dilution across all three replicates. MFI values of CFSE were lower in MSC and PBMC co-culture groups (mean MFI = 4747.33) compared to stimulated PBMCs alone (mean MFI = 5363), indicating reduced cell division. Proliferation fold change in the MSC and PBMC stimulated groups ranged from 2 to 4, compared to 8 to 9 in the PBMC stimulated groups; thus, reflecting a 2- to 4-fold reduction in proliferation. One-way ANOVA revealed statistically significant differences between the MSC co-culture and control groups ($p < 0.05$), confirming a reproducible and quantifiable immunosuppressive effect of MSCs on activated PBMCs.

Conclusion: The findings suggest that MSCs do have an immunosuppressive effect on PBMC proliferation in vitro, supporting their therapeutic potential in immune-mediated diseases. Future work will focus on enhancing MSC potency through red blood cell (RBC) preconditioning to upregulate key immunosuppressive molecules such as IL-10, TGF-β, and PD-L1. Further analysis of cytokine secretion, immunoregulatory surface markers, and gene expression will help elucidate MSC-driven pathways to optimize their clinical application.

This research was supported in part by the American Cancer Society – DICR INTR-23- 1253710-01-DICR INTR (Tonya J. Webb), National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P1.17 The Impact of Structured Provider Education on HIV Pre-Exposure Prophylaxis Delivery in a Pediatric Primary Care Office

Presenter: Gabriel Tobin-Xet
Mentor(s): Matthew Grant, MD[1]; Vandana Racherla, MD[1]
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Adolescents and young adults remain disproportionately affected by Human Immunodeficiency Virus (HIV). Methods of primary prevention of HIV, such as pre-exposure prophylaxis (PrEP) taken as a daily pill, are the most effective means of reducing transmission. Unfortunately, studies have shown that while many adolescents would benefit from PrEP, the overall uptake remains low for many reasons. There is existing evidence of healthcare associated barriers such as provider knowledge of and comfort in prescribing PrEP. This study aims to examine the impact of a structured education curriculum on the provision of PrEP counseling and prescriptions for PrEP in the adolescent population within an academic general pediatrics practice. Clinical encounters were examined for adolescents aged 16-21 seen within the general pediatrics practice at UMMC Midtown Pediatrics from March 2024 to September 2024, spanning the time frame two months before and five months after a provider education intervention. In addition to studying overall rates of PrEP counseling and prescriptions, analyses will be conducted on subgroups within the patient sample – from basic demographics to different HIV risk factors such as patients with multiple partners, patients who report having unprotected sex, patients with a prior diagnosis of a sexually transmitted infection, and males who have sex with males. Studying the extent to which PrEP has been implemented into provider workflow at adolescent visits at this practice will hopefully provide an example of how to integrate similar programs in other clinics to continue reducing HIV transmissions.

This research was supported in part by the Maryland Infection, Immunization, Intervention, and Impact Training Program (M4I), an initiative of the University of Maryland School of Medicine Office of Student Research, Center for Vaccine Development and Global Health, and Institute of Human Virology.

P1.18 SIX family proteins drive human erythropoiesis

Presenter: Angela Tan[1]
Mentor(s): Curt I. Civin, MD[2]; MinJung Kim, PhD[2]
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Successful *ex vivo* expansion of a patient's own hematopoietic stem-progenitor cells (HSPCs) to generate mature and maturing blood cells could potentially enhance blood transfusion and transplantation. In our molecular engineering approach toward developing *ex vivo* HSPC expansion strategies, our lab demonstrated previously that SIX1 overexpression (OE) in the human TF1 erythropoietic cell line model and in primary human $CD34^+$ HSPCs increased generation of erythroid cells (Creed *et al.*, *Development* 2020), and loss of SIX1 reduced human erythropoiesis. SIX family proteins, including 6 isoforms (SIX1-SIX6), are members of the human PAX-SIX-EYA-DACH transcriptional regulatory network (PSEDN). To further investigate the role of the SIX proteins in human erythropoiesis, we transduced TF1 cells with lentiviruses overexpressing each SIX protein isoform, and SIX OE TF1 cells were then analyzed for erythroid differentiation using qRT-PCR, western blot, and flow cytometry. OE of SIX2, SIX3, or SIX6 in TF1 cells markedly enhanced erythroid differentiation, as indicated by frequency of progeny cells expressing erythroid cell membrane markers (CD71, CD235a) with elevated mRNA/protein levels of erythroid molecules (e.g., hemoglobin, GATA1). Fewer progeny of TF1 cells with OE of SIX2, SIX3, or SIX6 expressed the $CD34^+$ HSPC marker, and total numbers of cells were reduced. SIX3 or SIX6 OE TF1 cells generated two distinct populations, erythroid ($CD34^{hi}CD235a^{hi}$) and non-erythroid ($CD34^{low}CD235a^{low}$), suggesting that SIX family proteins might also regulate differentiation into non-erythroid cells. Future studies will investigate the molecular mechanisms by which SIX family proteins regulate the types and numbers of progeny generated by primary human HSPCs.

P1.19 Physiologically Relevant Ca2+ and Mg2+ Concentrations to Regulate the BP2 Region of the STRA6-Calmodulin Complex

Presenter: Phoenix Wang
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STRA6 is a cellular receptor for retinol reuptake and Calmodulin (CaM) is known as a calcium signaling protein. Previous CryoEM data showed that STRA6 is commonly found bound to CaM, forming a STRA6-Calmodulin complex, which suggested that CaM could help regulate STRA6. Our lab previously showed how the BP2 region of the STRA6-Calmodulin structure changes in conformation when Ca2+ concentrations reflect those of an intracellular calcium signaling event. However, our lab previously used Zebrafish STRA6, which only conserves less than half the amino acid sequence compared to Human STRA6. Our objective of the research was to use a much closer homolog to Human STRA6, Sheep, which is 80% conserved. We conducted NMR experiments where we observed NMR chemical shift perturbations (CSPs) in the BP2-CaM complex due to the competitive binding of EGTA for Ca2+, modeling the structure where free calcium levels reflect those of a calcium signaling event. This helps us progress towards understanding the CaM-BP2 interaction in humans.

This work was supported by the National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P1.20 Low-Intensity Focused Ultrasound: A Non-Invasive Treatment Alternative for Opioid Use Disorder

Presenter: Ariel W. Abraham[1]
Mentors: Sandesh Kamdi, PhD[1]; Linda Chang, MD, MS[1]
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Background and rationale: Opioid use disorder (OUD) affects approximately 9.4 million Americans and accounted for over 70% of the 107,000 annual overdose deaths in the United States. OUD disrupts the brain's reward circuitry, particularly the nucleus accumbens (NAc) and anterior insular cortex (AIC) by enhancing dopaminergic and glutamatergic neurotransmission while suppressing GABAergic signaling. These neuroadaptations promote compulsive drug seeking, craving, and high relapse rates. Despite available treatments, clinical outcomes remain suboptimal due to low adherence and relapse rates nearing 70%, highlighting the need for novel therapeutic strategies. Low-intensity focused ultrasound (LiFU), a non-invasive neuromodulation technique, offers a promising therapeutic alternative for various neurological disorders. This study investigates whether magnetic resonance (MR) guided LiFU targeting the NAc and AIC can reduce fentanyl-seeking behavior in an oral fentanyl self-administration rat model.

Methods: All animal procedures followed institutional guidelines and were approved by the University of Maryland Baltimore Institutional Animal Care and Use Committee (AUP-00002527). Male and female rats (n=20) were trained to self-administer oral fentanyl (70 µg/mL) in 5% sucrose solution via active lever presses for 30 days, including 4 progressive ratios (PR) schedules to assess drug-seeking behavior. LiFU was administered for 10 minutes, targeting the NAc and/or AIC. Post-treatment, rats underwent behavioral testing for PR. Following the behavioral testing, rats will be euthanized, and brain tissue will be harvested for histopathological studies investigating the mechanistic role of LiFU in the treatment of opioid addiction.

Results and conclusion: We observed a significant increase in active lever presses and fentanyl infusion in rats compared to the sucrose-only group over a 30-day training period and subsequent PR sessions indicating the development of OUD. In the dose-dependent study, LiFU was delivered at 1.5 MHz frequency, with a mechanical index of 0.73, and a 30% duty cycle, producing a substantial reduction in both active lever presses and fentanyl infusion in self-administering animals. Currently we are further evaluating these parameters to determine their efficacy in suppressing fentanyl self-administration, aiming to reduce drug craving and relapse through the neuromodulatory effects of LiFU.

Acknowledgement: This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State and funded by NIH grant (DP1- (DA053719, PI: L.Chang).

P1.21 Quality assessment in orthopedic randomized controlled trials: Retracted vs non-retracted studies

Presenter: Kirie Frederick[1]
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Randomized controlled trials (RCTs) are foundational to evidence-based orthopedic care, informing treatment decisions for musculoskeletal conditions. However, scientific misconduct and poor reporting have undermined the reliability of some orthopedic RCTs, posing risks to patient outcomes. There is an urgent need for tools that assess RCT trustworthiness to ensure clinical practice and research are based on credible evidence.

We hypothesize that an adapted checklist can detect significant differences in trustworthiness indicators between retracted and non-retracted orthopedic RCTs. Our study builds on the TRACT (Trustworthiness in RAndomised Controlled Trials) checklist proposed by Mol et al., which offers a structured approach to identifying red flags in trial reporting. Additionally, Usman et al. demonstrated the effectiveness of comparing retracted articles with matched controls and highlighted the harmful downstream effects of citing retracted studies—providing methodological precedent for our work.

We adapted the TRACT checklist for orthopedic RCTs through expert consultation and assembled a dataset of 204 articles: 102 retracted orthopedic RCTs (1995–2025) and 102 matched controls identified via the Retraction Watch database and PubMed's "similar articles" function. Each article was independently assessed by two reviewers using the adapted checklist. Articles received a score of 1-10 based on the number of criteria met, and group means were compared to determine statistical significance.

After analyzing the data, the retracted group had a mean checklist score of 4.59 (95% CI: 4.25–4.93), whereas the matched control group had a mean score of 5.98 (95% CI: 5.57–6.39). Since the 95% confidence intervals do not overlap, the matched control group demonstrated a statistically significantly higher checklist score compared to the retracted group. However, the findings also indicate that many control articles failed to meet key criteria, as only 5 out of 10 criteria were met by more than 50% of the matched group, raising broader concerns about the overall standards of published orthopedic research.

This study aims to validate a practical tool for evaluating RCT trustworthiness, with potential applications in peer review, guideline development, and meta-research. Future work will focus on refining the checklist to emphasize the most predictive criteria and promoting its adoption across the field.

This work was supported in part by the American Cancer Society- DICR INTR-23-1253710-01-DICR INTR (Tonya J. Webb), National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P1.22 Assessing the Effects of Exercise During Chemotherapy on Physical Function Among Women with Breast Cancer

Presenter: Benick Mbaya[1]
Mentor(s): Ian Kleckner, PhD, MPH[1]; Javier Rosales, MS, ACSM EP-C[2]
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Chemotherapy is known to cause changes in physical function, causing bodily changes that can limit the quality of life. This includes changes in cardiovascular function and musculoskeletal function. Physical function is defined here as cardiovascular function (measured by a six-minute walk test (6MWT)) and musculoskeletal function (measured by a handgrip device and the Fullerton Balance Test). Not much is known about the effects of exercise on physical function in chemotherapy patients. This project sought to answer two questions via a statistical analysis of collected data:

1. How do outcomes in physical function change over time?
2. Does exercise affect overall outcomes?

Eighty women with breast cancer were randomly assigned into one of two treatments, with the experimental exercise arm performing 12 weeks of resistance band exercises and step count monitoring via Fitbit. The control arm involved 12 weeks of nutrition education, which controls for time, attention, and expectation of benefit. Regarding changes over time in physical function, we expect to see lower values in all three tests over time, as patients undergoing chemotherapy will experience fatigue and physical disfunction. Regarding differences between the exercise and control arms, we expect to see higher scores in all three tests in the exercise group compared to the control group.

This research was supported in part by the Nathan Schnaper Intern Program and the University of Maryland School of Medicine Office of Student Research. This work was sponsored by grants from the National Cancer Institute grants R25CA6872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), K07CA221931 (Ian Kleckner), and the UMMS Foundation Nathan Schnaper Fund.

P1.23 Impact of Intraoperative Autologous Blood Donation in Heart Transplantation on Hemodynamics and Transfusion Requirements: Interim Analysis

Presenter: Xiwei Peng[1]
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Introduction: Blood transfusion is critical in heart transplantation (HTx), but high usage of allogeneic blood products is associated with increased adverse outcomes. Intraoperative autologous blood donation (IABD)—the collection and reinfusion of a patient's own blood during surgery—is associated with reduced allogeneic transfusion requirements in cardiac surgery, yet its role in HTx remains unstudied. Given the hemodynamic instability that can occur during HTx surgery, understanding the impact of IABD on intraoperative hemodynamics is essential. This study evaluates associations between IABD use and transfusion needs, hemodynamics, morbidity, and mortality in heart transplant recipients. We hypothesize that IABD use is associated with reduced allogeneic transfusion volumes without significant differences in hemodynamic stability, morbidity, or mortality.

Methods: Adult HTx recipients aged 18–80 years at UMMC from 2014–2024 were identified via Epic records; 97 patients met inclusion criteria. For this interim analysis, 5 IABD-treated patients were age- and sex-matched to 5 controls. Baseline demographics, left ventricular assist device (LVAD) use, anticoagulant use, and prior HTx history were compared. Surgical factors including cardiopulmonary bypass (CPB) and aortic cross-clamp times, and early postoperative metrics such as ICU length of stay and duration of mechanical ventilation were analyzed. Primary outcomes included transfusion volumes, and hemodynamic metrics (mean arterial pressure [MAP] and vasopressor use) measured at three intraoperative timepoints: before, during, and after IABD (with equivalent timepoints in the control group).

Results: Baseline characteristics and cross-clamp times were comparable; CPB time was longer in controls. There were no strokes or 90-day mortalities. New continuous renal replacement therapy rates did not differ (OR = 0.41, 95% CI 0.005–11.8, p = 1). Platelet transfusions were significantly lower in the treatment group (mean 0.2 vs. 1.7 units, p = 0.044), while PRBC and FFP use did not differ significantly. Cryoprecipitate use was too infrequent to analyze. Norepinephrine equivalents were low and similar at all timepoints. MAP trends across three intraoperative timepoints did not differ by group (Group × Timepoint interaction p = 0.51, aligned rank transform ANOVA); post-hoc contrasts showed no significant differences (all p > 0.27).

Conclusion: Preliminary findings suggest IABD use is associated with reduced platelet transfusions without compromising hemodynamics or increasing morbidity in HTx. Analysis of the full cohort is ongoing to validate these results.

Funding: This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

P1.24 The New Imperative: Measuring Primary Palliative Care Education for Undergraduate Nursing Students

Presenter: Georgia Bardi[1]
Mentor(s): Hannah Murphy-Buc, PhD, RN[2]; Janet Wulf, DNP, CRNP, ACHPN[2]; Melissa McClean, MSN, ANP-BC, NP-C, ACHPN, CNE[2]
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[3]University of Maryland, College Park

An estimated 13 million people in the U.S. could benefit from palliative care, a number that is expected to rise as the population ages. However, pre-licensure nursing education often fails to prepare students to deliver high-quality palliative or end-of-life care. New graduates frequently report insufficient confidence, knowledge, communication skills, and emotional competence to care for seriously ill patients and their families. This gap between academic preparation and practice readiness results in moral distress among nurses and can negatively affect the care they give to their patients.

To address this critical practice gap, faculty at the University of Maryland School of Nursing created and piloted a required, stand-alone primary palliative care course for baccalaureate students—the first of its kind in the U.S., where palliative care education is typically elective or partially integrated into other courses. Despite positive evaluations and promising outcomes, limited empirical evidence exists on the course's effect.

This mixed-method, longitudinal study will address this gap by exploring the impact of UMSON's required primary palliative care course on undergraduate nursing students' development of primary palliative competence. Validated assessment tools will be used to measure changes in undergraduate nursing students' knowledge, experience, self-awareness, and performance before and at several points after completion of the course, with these 4 domains serving as indicators of primary palliative competence.

Findings will support strengthening generalist palliative care skills and inform competency-based curricular reforms, guiding future research and broader integration of palliative care education in nursing programs.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

P1.25 From Conference Presentation to Journal Publication: Restorative Practices to Strengthen Student-Faculty Relationships

Presenter: Alejandra Mata
Mentor: Hannah Murphy Buc, PhD, RN[1]
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Turning a conference presentation into an academic article requires more than reformatting slides into sections and paragraphs. It involved expanding on the original idea through in-depth research, analyzing publication guidelines, and tailoring the writing for a specific audience. This poster uses a conference presentation on restorative practices at UMSON to show how an organized approach can help outline the development of a publishable article. The goal of the article is to further support the integration of restorative practices in healthcare academia to continue encouraging respectful dialogue and connection between faculty and students.

This structured process includes conducting a full-text literature review, identifying appropriate journals, determining central arguments and hypotheses, synthesizing themes, and incorporating feedback to meet journal expectations. To narrow the focus of the article, JANE will be used to search for relevant, high impact journals for potential publication. A structured framework using two worksheets, Planning and Framing and Evaluation Content and Format, will be used to organize key information from the presentation into these components of the article.

The worksheets help to identify the differing goals, audiences, and needs of the presentation and article. They highlight what needs to be changed, what should be added, and what should remain. Additionally, they support the structure of the article by outlining the required sections and where relevant background, data, and supporting information should be added. As a result of this process, a comprehensive, publication ready academic article will be created.

Acknowledgement:
This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State

P2.01 Emerging Clinical Patterns Among Young Adults Living With HIV

Presenter: Jeffrey Fenn
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The introduction of combination antiretroviral therapy (cART) has transformed HIV from a fatal diagnosis to a manageable chronic condition, resulting in a growing population of individuals living into adulthood with HIV and facing new health challenges. Despite effective treatment, people with HIV remain at increased risk for non-AIDS-associated comorbidities. Individuals with perinatally acquired HIV (PHIV) represent a particularly vulnerable group, having been exposed to both HIV and antiretroviral therapy since birth, and may experience distinct patterns of comorbidity compared to those with non-perinatally acquired HIV (NPHIV). This study aims to assess and compare the prevalence and patterns of non-AIDS comorbidities among young adults with PHIV and NPHIV. We conducted a retrospective cohort study using clinical, virologic, and sociodemographic data from medical records. Participants were categorized by HIV acquisition status, and NPHIV participants were age-range-matched and randomly selected. Descriptive statistics characterized the study population, and univariate and multivariate logistic regression models were used to examine associations between clinical predictors and comorbidity outcomes. Categorical variables were compared using chi-squared and Fisher's exact tests, as appropriate. We found PHIV patients demonstrated a nonsignificant trend toward increased chronic kidney disease ($p = 0.0518$), along with a statistically significant higher mortality rate compared to NPHIV patients ($p = 0.0015$). PHIV acquisition was also associated with higher odds of hypertension, but this did not reach statistical significance (OR = 1.561, 95% CI: 0.917–2.659; $p = 0.1011$), which suggests a potential trend toward increased hypertension risk among individuals with PHIV. These findings demonstrate the need for continued monitoring and clinical management of young adults with PHIV, who may be at elevated risk for certain non-AIDS comorbidities.

This research was supported in part by the M4I: Maryland Infection, Immunization, Intervention, and Impact Training Program which was supported and administered by the Office of Student Research, Center for Vaccine Development and Global Health, and Institute of Human Virology.

P2.02 Bordetella *pertussis* infection downregulates the angiotensin system in the murine right ventricle

Presenter: Maame Sarpong
Mentor(s): Karen M. Scanlon, PhD[1]
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Bordetella pertussis (*Bp*) is a gram-negative bacterium that causes the respiratory infection known as pertussis, or whooping cough. The severity of *Bp* infection is age-dependent, with infants experiencing increased morbidity and mortality compared to adults. Adults and older children infected with *Bp* present with a paroxysmal cough while infants display more severe symptoms such as leukocytosis, cyanosis, and pulmonary hypertension (PH). Specific to *Bp* infection outcomes in infants, the development of PH is a major risk factor for fatal infection, triggering the onset of damaging and fatal effects including cardiac ischemia, cardiac arrest, and cardiac failure. Previously, our lab found that infant mice display significantly higher levels of the vasoconstricting angiotensin peptide, ANGII. This study examines the impact of *Bp* infection on the angiotensin system, which is a known regulator of cardiovascular pathologies. We hypothesize that during infant infection, the angiotensin system is dysregulated. To test this hypothesis, the right ventricle (RV) wall was isolated from the hearts of infant mice inoculated with *Bp* or vehicle buffer. The mRNA levels of *agt, agtr1a, agtr1b, agtr2, ace1,* and *ace2* were quantified and normalized to the expression of the housekeeping gene *hprt,* using quantitative RT-PCR. We found that *Bp* infection in infant mice downregulated the expression of angiotensinogen and the angiotensin type I receptor. These data indicate that pathological functions of the angiotensin system are reduced in the RV upon *Bp* infection in infant mice. Future studies will explore the impact of pertussis toxin on the activity of angiotensin receptors.

P2.03 Role of OAS1 rs1131454 SNP in Modulating Enterovirus Replication

Presenter: Paige Bonnet[1]
Mentor(s): Michael Wagner, PhD Student[2]; Saumen Sarkar, PhD[2]
Other Co-Author(s): Madeline Large[3]
[1]Nathan Schnaper Internship Program (NSIP) Student
[2]Molecular Microbiology and Immunology at the University of Maryland Baltimore (UMB)
[3]CATALYST Teacher

Enteroviruses, like EVD68, are a group of RNA viruses that infect the gastrointestinal and have no current vaccine or treatment available, making research of their mechanistic modes of action valuable knowledge.

The immune system is used to fight off both exogenous agents and diseases originating in host cells, like cancer. Similar immune factors can serve dual functions in antiviral and tumor-suppressor activities, like OAS1.

OAS1 is a protein that detects the presence of double-stranded RNA during viral replication. Isoforms of OAS1 correlate to different phenotypic abilities, but the extent to which these isoforms alter autophagy mechanisms within enterovirus replication is unknown. Preliminary data suggests that OAS1 limits enteroviral infection by producing 2'-5' oligoadenylate to induce RNase-L degradation and can stabilize IFN-beta. It also has been suggested that OAS1 plays a role in autophagy based on the associated proteins SQSTM1 and SIRT1. We hypothesize that OAS1 isoforms in the rs1131454 SNP, being 162G and 162S variants, will exhibit a different enteroviral regulation mechanism through the autophagy pathway. The use of infection assays, plaque assay for viral titers, and western blot autophagy protein analysis allows for these ideas to be investigated. Results revealed 162G reducing viral replication greater than the 162S variant. Studying differences of OAS1 isoforms like this could allow for understanding of disease severity and viral invasion based on phenotypes in patients.

This research was supported in part by the Nathan Schnaper Internship Program (NSIP), University of Maryland School of Medicine Office of Student Research.

P2.04 Does Integrin β4 Function as a Host Receptor for Fungi?

Presenter: Janice Baffoe-Bonnie
Mentor(s): Vincent Bruno, PhD[1]
[1]Department of Microbiology and Immunology, University of Maryland, Baltimore

While advances in modern medicine have improved outcomes for many patients, they have also increased the incidence of fungal infections by disrupting normal immune function. *Lomentospora prolificans* is an environmental fungus that can cause life-threatening infections when fungal conidia are inhaled. This project investigates how fungal conidia interact with human lung epithelial cells and explores potential strategies to block these interactions. We hypothesize that the integrin β4 protein functions as a host receptor for fungi, and that blocking the function of this protein might lead to decreased fungal accumulation in the lungs. To test this, I am attempting to overexpress the integrin b4 protein human lung epithelial cells (A549). Western blot analysis is being used to verify expression of the integrin β4 protein. In a parallel experiment, I am treating A549 cells with siRNA to knock down integrin β4 expression, while a control group received no siRNA. Both groups will then be infected with *Lomentospora prolificans* to determine whether integrin β4 facilitates fungal invasion using a CFU-based fungal adhesion assay.

P2.05 The role of integrin β4 in *Candida albicans* adhesion to human cells

Presenter: Lucy Murr
Mentor(s): Vincent Bruno, PhD[1]
[1]Department of Microbiology and Immunology, University of Maryland, Baltimore

Candida albicans is a commensal fungus that causes diverse infections after antibiotic use or immune debilitation. To improve our understanding of how *C. albicans* interacts with host cells, my project examines the role of integrin β4 in the adhesion of *Candida albicans* to human cells. Using a CFU-based adhesion assay, I am testing the ability of *C. albicans* to bind to cervical epithelial cells (HeLa) in the presence or absence of an antibody that binds to integrin β4. This assay measures how many *C. albicans* cells are able to attach to human epithelial cell cultures when ITGB4 is inhibited by the antibody compared to untreated cells. If integrin β4 is indeed promoting the interaction between *C. albicans* and the host cells, then we would observe a reduction in adhesion in the presence of the antibody. Future experiments will test the role of integrinβ4 in the adherence of *C. albicans* to vaginal epithelial cells (VK2). Understanding this mechanism will inform the development of new treatments or strategies to treat fungal infections in the future.

P2.06 MMP-9 Enhanced Mesenchymal Stem Cells Improve Islet Xenograft Survival in Diabetic Mice

Presenter: Pratham Chopra[1]
Mentor(s): Raphael Meier, MD[1]; Anjali Verma, PhD[1]
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Abstract
Diabetes requires lifelong insulin therapy, often leading to complications such as hypoglycemia and kidney failure. Encapsulated pancreatic islet xenotransplantation shows promise in diabetes treatment but is limited by poor islet engraftment. Matrix Metalloproteinase-9 (MMP-9), secreted by mesenchymal stem cells (MSCs) enhances islet function, engraftment, and vascularization. This study investigates whether encapsulated human islets co-transplanted with MMP-9-enhanced MSCs can reverse diabetes in mice.

Human MSCs were genetically modified to overexpress MMP-9 using two methods (GM-MSC1 and GM-MSC2), achieving 6,000-fold and 15-fold increases confirmed by qPCR and ELISA. We evaluated the impact of co-culturing human islets with MSCs on glucose-stimulated insulin secretion (GSIS), morphology, and viability (via FDA/PI staining). Diabetes was induced in C57BL/6 mice using streptozotocin (220 mg/kg). Diabetic mice received intraperitoneal xenotransplants of encapsulated human islets, with or without MSCs or GM-MSCs.

Mice receiving islets with GM-MSC1 showed the strongest insulin response and glycemic control. Forty percent of mice achieved long-term (>30 days) graft survival without immunosuppression, including three receiving GM-MSCs. Results suggest MMP-9-enhanced MSCs improve islet xenograft outcomes by creating a supportive microenvironment.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State and by the Maryland Stem Cell Research Fund (MSCRF).

P2.07 Identifying Immune Cells in Nurse Shark Tissues.

Presenter: Najah Johnson
Mentor(s): Helen Dooley; Thomas Hill

Sharks possess one of the oldest immune systems that features B cells and T cells, and lymphocytes, which also form the foundation of the adaptive immune system in humans. The spleen is a vital component of the shark's immune system, playing a crucial role in the development and maturation of B cells. Recent research from our lab has shown that immune responses can be generated in the shark pancreas. However, little is known about immune responses in other shark organs. To explore this question, I began by investigating whether lymphocytes are present in other nurse shark organs, with a specific focus on the kidney. First, I performed PCR analysis measuring the RNA transcript levels of the B cell marker IgM and the T cell marker CD3e in the kidney, pancreas, and spleen. I observed both IgM and CD3e transcripts in the kidney, suggesting there are B cells and T cells are present in this tissue. Next, using Hematoxylin and Eosin (H&E) staining, I evaluated whether lymphocyte aggregates were present in the kidney. To confirm the presence of B cells specifically, I employed immunohistochemistry (IHC) staining to identify B cells in the selected tissues. The results of these experiments will be presented. Future work continues to evaluate how the adaptive immune system functions in animals that lack lymph nodes.

This research was supported in part by the Dooley lab, the Institute of Marine and Environmental Technology (IMET), and the SUMMIR program

P2.08 Developing an agent based model of the pancreatic ductal adenocarcinoma microenvironment

Presenter: Joshua Naranjo
Mentor(s): Daniel Bergman, PhD; Jeanette Johnson, PhD; Elana Fertig, PhD
Co-Author(s): Lynijah Russell

Pancreatic ductal adenocarcinoma (PDAC) is a challenging and often fatal disease. One of the major factors contributing to PDAC's lethality is the immunosuppressive tumor microenvironment produced by PDAC. The "immune cold" condition of the tumor microenvironment (TME) contributes to decreased efficacy of the immunotherapies that have been revolutionary for treatment of many other tumor types; this despite the confirmed presence of antigen specific T-cells in the PDAC TME. Developing a better understanding of the TME landscape, specifically the factors that play roles in activating and deactivating antigen specific T-cells in the TME, will lead to improved outcomes for PDAC patients and possibly improve the outcome for immunotherapies targeting PDAC. To this end, we use an agent based modeling system that simulates cellular interactions between the immune, stromal, and neoplastic cell types and chemokines present in the TME. We build T cell antigen recognition and activation in the PDAC microenvironment, with model conditions derived from a scRNAseq dataset of untreated human PDAC. These models approximate T cell activation, and expansion using hill functions. These models focus on the mechanism of T cell activation, including signals 1, 2, and 3 obtained from antigen-presenting cells and other cells in the TME,and how these play a role in the overall antitumor response. This allows us to directly explore the mechanisms that may contribute to the differentiation of T-cells into antitumor effectors vs. protumor T-regulatory cells in the PDAC TME. In subsequent work this will allow a systematic investigation of the implications of ectopic MHC class II expression, that has been consistently reported in the PDAC microenvironment in the stromal and neoplastic compartments. In addition to this we go on to model the effects of T-regs on effector T-cell response. We finally go on to model the effects of the previously listed dynamics on both CD4 and CD8 compartments and their ability to control tumor response.

This research was supported in part by the Bridges to The Doctorate Program at Towson University.

P2.09 Effects of modulating ERK 1/2 on tristetraprolin expression in A375 melanoma cells

Presenter: Vivien Van't Hof[1]
Mentor(s): Paul Shapiro, PhD[2]
[1]Nathan Schnaper Intern Program, University of Maryland Marlene and Stewart Greenbaum Comprehensive Cancer Center, Baltimore, Maryland
[2]Department of Pharmaceutical Sciences, University of Maryland School of Pharmacy, Baltimore, MD

Melanoma is an aggressive skin cancer marked by chronic inflammation and constitutive activation of the extracellular signal-regulated kinase (ERK). This pathway has been implicated in suppressing tristetraprolin (TTP), RNA-binding protein that promotes the degradation of pro-inflammatory cytokine mRNAs. Current ERK inhibitors can lead to resistance and toxicity, understanding how ERK signaling affects TTP expression may reveal more targeted strategies to control inflammation in melanoma. **This study aims to determine how modulation of ERK1/2 activity influences TTP expression in A375 melanoma cells. Cells were co-treated with** Phorbol 12-myristate 13-acetate (PMA), an known ERK activator, and a panel of ERK-modulating compounds (SF-3-030, SP-2-025, SP-2-038, SP-2-090, SP-2-096, SP-2-098, SP-2-151, SP-2-153, SP-2-156) for fifteen minutes. TTP and phosphorylated ERK (pERK) expression were measured by Western Blot and compared to cells treated with PMA and BVD-523, a clinical stage ERK inhibitor given an Expanded Access grant to be used when all other options have been exhausted. PMA alone significantly increased TTP expression. SF-3-030, SP-2-038 and BVD-523 elevated pERK levels, while SP-2-153 and SP-2-156 markedly suppressed pERK. Notably, SP-2-028 and SP-2-156 partially reversed PMA-induced TTP upregulation. To further validate ERK activity, FosB, a downstream ERK-responsive transcription factor, expression was also assessed. A375 melanoma cells were co-treated with PMA and the ERK modulating compounds in varying concentrations. These findings suggest that pharmacological modulation or ERK alters TTP levels, providing insights into potential therapeutic strategies for targeting inflammation in melanoma.

This work was supported by the National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P2.10 The role of nuclear receptor NR1D1 in tumor response to anticancer therapeutics

Presenter: Arryn Berroya[1,2]
Mentor(s): Remi Klotz, PhD[1]; Min Yu, MD[1]
[1]Department of Pharmacology and Physiology, University of Maryland School of Medicine, Baltimore, MD, USA
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Brain metastasis is fatal condition within advanced cancer that affects 20% of cancer patients, and understanding tumor metastatic mechanisms is essential in developing effective anti-tumor strategies. Previous data proven in our lab have demonstrated the potential role of the nuclear receptor NR1D1, on brain metastasis, especially in its activity being correlated to tumor proliferation and immune evasion. Our current focus is understanding the role of NR1D1 in brain metastasis regulation specifically on disseminated tumor cells that exhibit characteristics of dormancy in the niche beyond the blood brain barrier (BBB). It has been found within our project that NR1D1 promotes cell death through the immune response *in vitro* particularly by stimulating the release of interferon and cytokines. We then wanted to understand this in correlation within the perivascular niche and of its effects when treated with anti-tumor therapeutics. Breast cancer cells lines, HCC1954 and SUM190 which were chosen due to their high and low NR1D1 expression respectively, were used as models to undergo chemotherapy and irradiation. To determine the appropriate levels of chemotherapy and irradiation to use on these cell lines, a separate pilot experiment was conducted, and it was found that the appropriate paclitaxel concentration to use was 4 nM and 10 Gy for irradiation. We will then use these concentrations in our co-culture experiments containing tumor cells with endothelial cells, pericytes, and astrocytes to mimic the BBB to determine the regulation of NR1D1 activity and anti-tumor therapeutic effects within this perivascular niche.

This research was supported by the National Cancer Institute grants R25CA186872 to Bret A. Hassel and P30CA134274 to Taofeek Owonikoko, and the UMMS Foundation Nathan Schnaper Fund; in part of the Nathan Schnaper Internship Program, University of Maryland Baltimore.

P2.11 Investigating the Role of Drug Inhibition of hnRNP A18 In Suppressing Hypoxia-Induced Glioblastoma Proliferation

Presenter: Andrew Parakhoodi[1,2]
Mentor: France Carrier, PhD[2]
Co-Author(s): Shirin Azarbarzin, PhD[2]; Eduardo Solano-Gonzalez, PhD[2]
[1]Nathan Schnaper Intern Program
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Glioblastoma multiforme (GBM) is a highly malignant brain cancer in adults with poor prognosis and limited response to chemotherapy. A key feature of GBM is tumor hypoxia, which is based on substantial evidence that low levels of oxygen play a role in tumor initiation, differentiation, and survival. In Dr. France Carrier's lab, we have identified the heterogeneous ribonucleoprotein A18 (hnRNP A18) as a regulator of protein translation in cancer cells. Targeting the hypoxia inducible factors regulated by hnRNP A18 has thus been suggested as a potential therapeutic strategy. hnRNP A18, a stress responsive RNA-binding protein, supports tumor cell viability and proliferation under hypoxic conditions. D54 (p53 wildtype) and U118 (p53/PTEN mutant) GBM cell lines were cultured and exposed to normoxic and hypoxic (1% O_2, 5% CO_2, 94% N_2) conditions to investigate how p53 status influences GBM proliferation and response to hnRNP A18-targeted treatments. Western blot analysis confirmed basal hnRNP A18 expression in normoxic and hypoxic conditions after 4, 8, and 18 hours. Subsequent studies will evaluate 3 candidates of small molecule inhibitors against hnRNP A18 and determine their IC50 values for suppressing GBM proliferation using a WST-1 assay. In here, we hope to show that inhibition of hnRNP A18 under hypoxic conditions significantly reduces glioblastoma proliferation. Further invitro and in vivo mouse xenograft studies will evaluate the efficacy of the drug candidates alone and combined with radiation, chemotherapy, or immunotherapy to assess additive effects on tumor invasion. Ultimately, this work may enhance anticancer therapy responses and improve prognosis in GBM patients.

This work was supported by the National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P2.12 Fluid Shear Stress-Mediated Activation of Ras in Primary and Metastatic Brain Cancer

Presenter: Grace Acle[1]
Mentor: Pavlos Anastasiadis, PhD[1,2,3]
Co-Author(s): Sarah Benjumea[1]; Alexandra Seas[1]; Masanobu Komatsu[4]
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R-Ras is a small GTPase that is a member of the Ras superfamily and interacts with structural proteins to modulate cytoskeletal integrity. Aberrant activation of R-Ras has been observed in a variety of cancer types, and has been related to increased migration and invasion in cancer cells.

The changes observed as a result of fluid shear stress (FSS) have been variable across cell types and methods of study, requiring further investigation on a signaling level. This study seeks to quantify the relationship between FSS and Ras activity in both primary and metastatic brain cancer cell lines.

To mimic the effect of FSS on a tumor model *in vitro*, primary glioblastoma cells were cultured in suspension to form neurospheres (e.g., suspension), and metastatic breast cancer cells were cultured adherently. The cells were then transferred to spin pods that allowed for continuous rotation for 1 hour and 24 hours. A Ras pulldown assay was used to quantify Ras activity levels, and western blotting identified R-Ras expression in FSS versus cells cultured statically. In order to observe the effect of FSS on adherent cells, GBM1 cells were seeded in Ibidi flow channels that allowed for media to flow over the cells in a continuous loop. After 24 hours, flow was halted, and the cells were stained using immunofluorescence to observe R-Ras expression. Preliminary results of these experiments suggest that FSS plays a role in the increased activation of Ras and may have implications as a therapeutic target for primary and metastatic brain cancer.

This work was supported by the National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P2.13 A Novel Construct that Enables Simultaneous Tracking of Cell Cycle Progression and Calcium in Glioblastoma

Presenter: Laasya Ravipati[1]
Mentor(s): Jinghui Wang, PhD[2]; Yajie Liang, PhD[2]
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Cell cycle progression and calcium signaling are both critical, dynamic processes in cancer biology, particularly in highly aggressive and heterogeneous tumors such as human glioblastoma (GBM). However, current tools do not allow for simultaneous, long-term tracking of both parameters at the single-cell level. To address this gap, we integrated a modified fluorescent ubiquitination-based cell cycle indicator (FUCCI) with a genetically encoded calcium sensor (GCaMP6s) resulting in pLV-EF1A-Cherry-hCdt-p2a-miRFP670nano3-hGem1(1- 110):t2a-GCaMP6s). Specifically, we replaced the blue fluorescent protein, which exhibits high background fluorescence due to the presence of autofluorescence from culture media, with a near-infrared fluorophore (miRFP670nano). The three fluorophores (mCherry, miRFP670nano, and GCaMP6s) were multiplexed for precise reporting of cell cycle and calcium parameters. We tested this system with a GBM cell line (GBM1) and HEK293 cells as a control by performing longitudinal imaging at 15-minute intervals over 24 hours. We successfully observed dynamic changes in the fluorescence intensity of the system regarding cell cycle progression and calcium dynamics in both HEK293 and GBM1 cells. Afterwards we quantified and performed a correlation on the following parameters: mean intensities of cell cycle, mean intensity of calcium indicator, and migration features. In conclusion, our novel functional cell tracking platform enables simultaneous tracking of cell cycle progression and calcium in GBM. Future studies should evaluate tumor heterogeneity across different cell lines to see to what extent the degree of heterogeneity differs. Quantifying such heterogeneous behaviors could inform the development of new cancer therapies and more effective deterrents against cancer recurrence.

This research was supported in part by the Nathan Schnaper Intern Program in Translational Cancer Research (NSIP), University of Maryland School of Medicine Office of Student Research.

P2.14 Modeling Pancreatic Cancer and Precancer Microenvironments Using PhysiCell Agent-Based Simulations

Presenter: Lynijah Russell
Mentor(s): Daniel Bergman, PhD

Pancreatic ductal adenocarcinoma (PDAC) is an aggressive and complex cancer due to its late detection and poor understanding of early progression. Understanding how pancreatic tumors evolve from precancerous lesions is essential to improving early detection and therapies. Tumor development requires interactions between several types of cells within the tumor microenvironment (TME), including epithelial, immune, and stromal cells. While we have incredible genomics data that tell us about a human TME at a point in time, these datasets only capture static snapshots of these dynamics, limiting our ability to model cancer progression over time. Agent-based modeling (ABM) is a mathematical framework for creating mechanistic and dynamic digital models of biological systems, such as PDAC. However, these models currently lack standards to connect to experimental data. Here I develop a Python-based pipeline for doing this in PhysiCell, a community-driven ABM framework. This allows rapid construction of model TMEs, overcoming the one-at-a-time limitation of traditional PhysiCell workflows. Building a digital library of cancer and precancer cell types will enable us to create reproducible, standardized patient-specific in silico models to systematically test interventions and visualize tumor progression. I use this pipeline to generate simulations of interacting CD8, CD4, fibroblasts, and epithelial cells, producing dynamic models that represent mechanisms underlying early cancer proliferation. In future work, I will compare these results with condition-matched experimental data from tissue culture systems to assess performance, iterate upon them, and investigate predictions from the ABM. This work lays the foundation for patient-customized TME simulations. A powerful tool for precision oncology.

P2.15 A novel PD-1 and LAG-3-targeting bispecific molecule in a murine glioblastoma model

Presenter: Justin Obi
Mentor(s): Pavlos Anastasiadis, PhD

Glioblastoma (GBM) is an aggressive primary brain tumor originating from neural stem or progenitor cells that carry tumor-initiating genetic alterations. Immunotherapy presents therapeutic options for GBM patients with poor prognosis. Immune checkpoint inhibitors have been extensively studied as promising targets for T cell upregulation in solid tumors. Still, their use in the brain has been historically underutilized due to therapeutic barriers, such as the blood-brain barrier (BBB). In this study, we investigated T cell infiltration and blockade of a bispecific molecule (e.g., tebotelimab), PD-1 and LAG-3, through immunofluorescent staining and histological methods. We hypothesized that there would be significantly more T-cell infiltration in the treated mice as opposed to the control mice. To answer these questions, we first performed orthotopic glioma inoculation in 8-10-week-old mice with 10,000 CT2A cells (syngeneic cell line). Tumor-bearing mice were treated with a novel bispecific antibody targeting PD-1/LAG-3 or with a nonspecific antibody targeting IgG (control) at days 10 and 13 post-inoculation. Animals that reached endpoint criteria were euthanized, and their brains were harvested for histology and immunofluorescence staining. Furthermore, cell experiments were also conducted through subjection *in vitro* to fluid shear stress to mimic hemodynamic shear forces as well as other techniques such as paraffin wax embedding and sectioning, Hematoxylin and Eosin staining. By examining the difference in expression of PD-1 and LAG-3, we can utilize the effectiveness of dual checkpoint inhibition in other combination therapies, including focused ultrasound and chemotherapy.

This work was supported in part by the American Cancer Society- DICR INTR-23-1253710-01-DICR INTR (Tonya J. Webb), National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P2.16 Qualitative Analysis of Cancer Survivors' Experience in a Nutritional Clinical Trial

Presenter: Jackson Kerner
Mentor(s): Amber Kleckner, PhD[1]
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Background: Cancer-related fatigue is a prevalent and persistent issue in many cancer survivors. Time-restricted eating is a promising nutritional intervention that may help to combat this fatigue.

Purpose: Use qualitative methods to describe participants' lived experiences in the FREDA study, which tested if addition of time-restricted eating (TRE) with a 14-hour fasting period to nutritional counseling is a feasible and effective way to relieve cancer-related fatigue (CRF) in cancer survivors.

Methods: The FREDA study was a two-arm, randomized control trial. Participants were eligible if they were adult cancer survivors 2 months to 2 years post-treatment. A nutritionist was assigned to work 1:1 with FREDA participants to develop individualized nutrition plans for 12 weeks. The study required participants, should they be randomized to the TRE group, to eat within a 10-hour period of their choosing. At the conclusion of the study, exit interviews were conducted to gauge participants' experiences in the trial. The interviews were transcribed and two independent coders thematically analyzed the transcripts using inductive and deductive coding to create a codebook. NVivo software was used for data organization and analysis.

Results: Participant responses varied on whether they thought the study impacted their fatigue levels. Despite this, the majority of participants would recommend the FREDA study to others, as they found that being in the study helped them to set and achieve lifestyle goals for themselves. Participants who were randomized to the TRE group occasionally noted that it was difficult to switch to a 14-hour fasting schedule. Additionally, many participants noted they were happy that cancer-related fatigue was gaining more attention, hoping to find some solutions for themselves and other cancer survivors.

Conclusions: Many participants enjoyed the study and regardless of its impact on their fatigue, the majority of participants found the study helped them to gain better control of their dietary habits.

This research was supported by the NIH (UL1TR003098), Maryland Department of Health's Cigarette Restitution Fund Program (no.CH-649-CRF), and UM Scholars Partnership

P2.17 Evaluating the *in vitro* Radiosensitizing Effects of Oxaliplatin in Comparison to the Current Standard of Care Treatment: Temozolomide

Presenter: Chloe Seluchins[1]
Mentor(s): Ananya Elati, BS[2,3]; Jeffrey Winkles, PhD[2,3]; Anthony Kim, PhD[2,3]
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Glioblastoma (GBM) is one of the most common and aggressive forms of brain cancer in adults. The standard of care treatment includes surgical resection of the tumor followed by concurrent administration of temozolomide (TMZ) chemotherapy and radiation therapy (RT) to kill off residual tumor cells. Although this combination therapy has the potential to be successful, it ultimately has low efficacy due to the inherent radioresistance of GBM and TMZ resistance that develops in more than 50% of GBM patients, resulting in a low survival rate of only 12 to 14 months. Platinum-based drugs are amassing more interest for the treatment of GBM due to their various radiosensitization mechanisms. This study focuses on the use of oxaliplatin (OXA) as a GBM radiosensitizer due to its ability to target key oncogenic signaling pathways. To evaluate whether the interaction between oxaliplatin and radiation is synergistic at sub-cytotoxic doses, a dose-finding cell viability assay was performed on three GBM cell lines. Results from this experiment demonstrated synergy at certain sub-cytotoxic doses of oxaliplatin, which were then used for clonogenic assays to assess the efficacy of OXA and radiation as a combination therapy. However, in contrast to TMZ, OXA cannot cross the blood brain barrier, so future studies aim to develop a delivery system that can effectively deliver OXA to GBM tumors.

This work was supported by the National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P2.18 Pulmonary Complications Following Lateral Beam Total Body Irradiation: A Retrospective Analysis of Dose-Dependent Outcomes

Presenter: Nina Kolodgie[1]
Mentor(s): Zaker Rana, MD[1]
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Background: Hematopoietic stem cell transplantation (HSCT) is a vital treatment option for patients with refractory lymphoma and has demonstrated improvements in overall survival. The optimal approach for approach for radiation regimens remains unknown. The lateral total body irradiation (TBI) technique has been utilized as an alternative to AP/PA methods, offering improved dose uniformity across midline structures but does not allow for physical organ shielding. The objective of this study was to retrospectively evaluate outcomes and complications in patients who underwent transplantation following the lateral beam TBI.

Methods: A total of 84 patients underwent lateral TBI-based conditioning at a single institution between 2022 and 2025. TBI ranging from 2-12 Gy in 1-6 fractions was combined with high-dose cyclophosphamide, melphalan, fludarabine, or etoposide, depending on the underlying disease. Acute and chronic toxicity was scored using Common Terminology Criteria for Adverse Events (CTCAE) criteria. Overall survival and the cumulative incidence of toxicity were calculated and compared using a log-rank test.

Results: Median follow-up was 364 days. Overall survival was 84.6% in the high-dose group and 71.8% in the low-dose group. Three patients (3.6%) died during transplant admission, all receiving 4 Gy in 2 fractions. Fisher's exact test showed no statistically significant association between dose and survival (OR = 2.14, 95% CI: 0.41–21.57, p = 0.50), although there is uncertainty due to small sample size. The most common toxicities were fevers (25%) and acute respiratory compromise (42.9%).

Conclusions: Lateral total body irradiation was well-tolerated showing comparable toxicity and survival outcomes when compared to historical total body irradiation treatment.

This research was supported by the Department of Radiation Oncology, University of Maryland School of Medicine.

P2.19 ROR1 CAR T-Cells As a Novel Treatment for Prostate Cancer

Presenter: Sophia Bredar[1]
Mentor(s): Djordje Atanackovic, MD[2]; Aerielle Matsangos, MHS[2]
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Chimeric Antigen Receptor (CAR) T-cells engineered to kill tumor cells have revolutionized the treatment of hematologic cancers. To do this, CAR T-cells use an artificially expressed antibody to bind to surface antigen on tumors. The efficacy of this approach against solid tumors has been limited, which is primarily due to (1) the absence of appropriate targets from solid tumors and (2) the presence of immunosuppressive factors like TGFβ in the tumor microenvironment. Our lab is for the first time investigating the tumor-targeting ability of novel CAR T-cells specific for the Receptor Tyrosine Kinase-like Orphan Receptor 1 (ROR1) antigen. We analyzed ROR1 surface expression on various prostate cancer cell lines by flow cytometry. Next, tumor cells underwent GFP-luc lentivirus transduction and were sorted to yield pure target cells for cytotoxicity assays. Finally, we evaluated the tumor-killing ability of ROR1-specific CAR T-cells and compared them to ROR1 CAR T-cells armored with a dominant-negative form of TGFβRII. Our novel CAR T-cells were able to bind to and produce various cytokines in response to recombinant ROR1 protein. We found that most of the prostate tumor cell lines express substantial ROR1 levels. Finally, unmodified and “armored” CAR T-cells both efficiently killed prostate cancer cells, even at low effector/target ratios. Our findings suggest for the first time that ROR1 CAR T-cells may serve as a promising treatment for prostate cancer, especially when combined with anti-TGFβ mechanisms. Additional studies are underway in our lab evaluating this novel CAR T-cell approach in other solid tumors.

This research was supported in part by the Nathan Schnaper Intern Program at the University of Maryland Marlene and Stewart Greenebaum Comprehensive Cancer Center.

P2.20 Engineered CAR T-cells targeting ROR1 for the treatment of lung cancer

Presenter: Ashley Gelin[1]
Mentor(s): Djorde Atanackovic, M.D.[1,2]
Co-Author(s): Aerielle Matsangos, M.S[1,2], Daniel Yamoah, B.S.[1,2], Rediet Mulato, B.S.[1,2]
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Chimeric antigen receptor (CAR)-modified T cells (CAR-T) targeting tumor-associated surface molecules have transformed the treatment of hematologic malignancies. However, effective CAR-T therapies for solid tumors remain elusive due to two major barriers: (1) the lack of tumor-specific antigens and (2) the immunosuppressive tumor microenvironment, particularly due to TGF-β signaling. To address this, we developed ROR1-specific CAR-T cells armored with a dominant-negative TGFβ receptor II (TGFβRIIDN). We investigated, for the first time, ROR1 surface expression across lung cancer subtypes and evaluated the anti-tumor activity of TGFβRIIDN ROR1-CAR-T cells. We used 14 lung cancer lines to assess ROR1 expression by quantitative flow cytometry and we genetically modified the cell lines for the assessment of the antitumor function of ROR1-specific CAR-T.

ROR1-specific CAR-T were generated by lentiviral transduction of T cells. Lung cancer cell lines expressing luciferase-GFP were generated by lentiviral transduction. Cytotoxic responses and proteomic profiles were assessed using co-culture killing assays and codeplex secretome assays.

We detected substantial surface expression of ROR1 by flow cytometry on most small cell and non-small cell lung cancer lines. We found that ROR1-CAR-T cells secreted anti-tumor effector cytokines after stimulation with recombinant ROR1. Importantly, ROR1-CAR-T cells exhibited high anti-tumor activity against both types of lung cancer lines (NSCLC/SCLC).

This study presents a novel ROR1 CAR-T cell strategy for the treatment of NSCLC and SCLC. Our *in vitro* experiments build the basis for future in vivo evaluation of this approach and hopefully a clinical study using TGFβRIIDN ROR1-CAR T as a novel treatment for lung cancer.

This study was funded by two grants from the Kahlert Foundation (to D.A.), by the Maryland Department of Health's Cigarette Restitution Fund Program (to D.A. and X.F.) and by the National Cancer Institute - Cancer Center Support Grant (CCSG) P30CA134274. In addition, the D.A. lab received philanthropic support from the Becker Family Foundation, Marco Chacon and the Kassap Family Foundation.

P2.21 Exploring the Role of Race in Adolescent Dietary Choices: United States High School Students

Presenters: Keira Terrillion[1], Davis Arroyave[1], Imani Ward[2]
Mentor: Diane Marie St. George, PhD[3]
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Introduction: Adolescent dietary habits play a pivotal role in shaping long term health outcomes such as obesity. Racial and ethnic disparities in diet related health outcomes suggest that social, cultural, and environmental factors may influence the eating behaviors of high school students. The goal of this study was to highlight key nutritional/ dietary differences among different racial and ethnic groups to inform more equitable public health interventions. Methods: This study used data from the Centers for Disease Control and Prevention's 2023 Youth Risk Behavior Survey (YRBS), a nationally representative survey of high school students. Results: Among the 20,103 respondents, there was variability in vegetable, fruit, soda and breakfast consumption by race/ethnicity. Conclusion: This study supported previous research that showed racial and ethnic differences in dietary practices. While diet may play a role, other factors, such as disparities in physical activity levels, access to resources, or social determinants of health, could contribute to higher obesity rates among African American adolescents.

P2.22 Investigating the Functional Role of UBASH3B Isoforms in HPV-Negative Head and Neck Squamous Cell Carcinoma

Presenter: Cameron Westlake [1,2]
Mentor(s): Ishita Gupta[1,2], Madeleine Ndahayo[1,2], Daria A. Gaykalova[1,2, 3*]
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Ubiquitin-associated SH3 domain containing protein B (UBASH3B) is a protein tyrosine phosphatase involved in regulating epidermal growth factor receptor (EGFR) signaling. Our previous work has identified UBASH3B as a biomarker in HPV-negative head and neck squamous cell carcinoma (HPV- HNSCC), with higher expression correlating with advanced tumor stage. Through alternative splicing, UBASH3B creates two structurally distinct isoforms; however, their functions remain uncharacterized. Transcript variant 1 encodes the primary isoform (UBASH3B-1), while isoform 2 (UBASH3B-2) has an extended 5'UTR and produces a different amino acid sequence in Exons 1 and 2. We hypothesize that the UBASH3B isoforms have distinct functional roles in HPV- HNSCC progression, with differential effects on tumor biology. In this study, we performed in silico analysis alongside in vitro functional studies to determine the role of each isoform in HPV- HNSCC progression. Analysis of the Cancer Genome Atlas (TCGA) data was performed to determine gene expression patterns of UBASH3B isoforms. Additionally, qRT-PCR analysis and functional assays were conducted to examine differential expression and biological effects of UBASH3B isoforms in HPV- HNSCC cell lines. In silico analysis revealed isoform-specific mRNA and protein expression patterns across HPV- HNSCC. Additionally, pathway analysis (GSEA) of RNA-seq data correlated high expression of both isoforms with the TGF-β signaling pathway, while the HEDGEHOG pathway was correlated with high expression of only UBASH3B-1. Our in vitro experiments demonstrated that transient silencing of UBASH3B-1 decreased cell proliferation and migration, while silencing UBASH3B-2 enhanced proliferation as compared to UBASH3B-1. Based on this data, we conclude that UBASH3B-1 poses oncogenic effects, while UBASH3B-2 plays a potential role as a tumor suppressor, demonstrating how alternative splicing can impact oncogenic signaling pathways in HPV-HNSCC.

This work was supported by the National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P2.23 Liposomal HDAC8 inhibitor formulation for neuroblastoma treatment

Presenter: Eva Qiao[1, 2]
Mentor(s): Ryan M. Pearson, PhD[1, 2]
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Neuroblastoma is the most common cancer in infants with high-risk patients having a 5-year survival rate of 50%. Deacetylation of histones plays a key role in cancer development with histone deacetylases (HDACs) modulating the expression of tumor suppressor genes like p53, highlighting HDAC inhibitors as promising cancer treatments. Several pan-HDAC inhibitors have been FDA approved; however, significant side effects including arrythmias and fatigue hinder their widespread use as anti-cancer therapies. Targeting individual HDAC isoforms may mitigate the toxicity of pan-HDAC inhibitors. Specifically, HDAC8 has been identified as a potential target due to its overexpression in various cancers, like neuroblastoma, with several studies demonstrating HDAC8-specific inhibitors effectively hindering cancer growth. However, many small-molecule drugs suffer from poor water solubility, making direct administration challenging. Encapsulating hydrophobic therapeutics in liposomes can enhance circulation half-life and mitigate side effects. We hypothesize that liposomal encapsulation of a novel HDAC8 inhibitor will circumvent the compound's poor water solubility, increasing efficacy and improving the safety profile compared to direct administration of the drug. Liposomes were prepared using both thin film hydration and microfluidics-based methods. Various liposomal formulations were developed with sizes from 100 to 300 nm and PDIs below 0.2 with sufficient drug encapsulation. IC50 data of liposomal formulations was determined through an MTS assay, while HDAC8 inhibition was validated through confocal microscopy. In the future, the microfluidics formulation will be optimized for lyophilization and maximum tolerated dose studies will be conducted in mice.

This work was supported by the National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P2.24 The Effect of Combining MEK Inhibitors with Commonly Used Chemotherapies in KRAS Mutated Pancreas Cancer

Presenter: Karl Trageser
Mentor: Rena Lapidus, PhD; Aaron Ciner, MD
Greenebaum Comprehensive Cancer Center University of Maryland School of Medicine, Baltimore, MD

Pancreatic cancer is an unmet medical need in the US with 67,440 people diagnosed in 2025; the five-year survival rate is 13.3%. The oncogene, Kirsten rat sarcoma virus (KRAS), is mutated in over 90% of pancreatic cancers, giving cancer cells a growth advantage. Clinically, it was observed that patients with G12R mutations respond to chemotherapy and survive longer than patients with G12D mutations. Since KRAS is found upstream of MEK in the RAS/RAF/MEK/ERK pathway, we evaluated if the MEK inhibitor, trametinib, combined with commonly used chemotherapies would be more effective in cell lines with G12R mutations compared to cell lines with G12D mutations. Two human cell lines with G12R and two with G12D mutations were treated with trametinib in combination with 5-flurouracil (5-FU), everolimus, and erlotinib. The drugs were serially diluted, added to media, then added to each cell line separately and in combination for 72 hours. As single agents, everolimus and erlotinib had no effect. PATC50 (G12R) was inhibited 35% with 5μM 5-FU and only 18% with 5μM trametinib. But in combination, cell proliferation was inhibited by 79%. The other G12R cell line, PSN-1, also had low single agent effects, 28% inhibition for both, but the combination inhibited 68%. HPAF-II and Panc1, cell lines with the G12D mutation, had lower responses to both the single agents and the combination, demonstrating that the combination of 5-FU and trametinib is more effective in G12R cell lines than in G12D cell lines.

This work was supported by the National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P2.25 Caveolin-1 Upregulation Fuels EGFR-Mediated Resistance to Radiation and Osimertinib in Lung Cancer

Presenter: Grace Rawlett[1]
Mentor: Hem Shukla, PhD[1]
Co-Authors: Sanjit Roy[1], PhD; Martina Vize, MEd[1]
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Non-small cell lung cancer (NSCLC) is the leading cause of cancer-related death worldwide and accounts for 85% of all lung cancer cases. Typical treatments include surgery, chemotherapy, radiation therapy, and immunotherapy, or a combination of treatments. Patients may develop resistance to radiation therapy after long-term use. Thus, effective therapeutic options to overcome radio-resistance in lung cancer treatment are needed. Based on previous observations, the interaction of caveolin-1 (CAV1) with epidermal growth factor receptor (EGFR) could be responsible for resistance to radiation and Osimertinib in lung cancer.

In the present investigation, we have observed the upregulation of CAV1 and EGFR in A549 radio-resistant (RR) and HCC-827 EGFR mutant lung cancer cells when radiated. When HCC-827 cells were treated with 4 and 8 Gy, we observed significant upregulation of CAV1 (25% and 35% respectively) and EGFR, as compared to untreated control. The colony formation assay at 4 Gy resulted in HCC-827 cells being most sensitive to radiation (20% survival), while A549RR cells were the least sensitive (55%). The Western blot analysis showed high expression of phosphorylated EGFR (pTyr1068) in HCC-827, as compared to none in A549. We further investigated CAV1's and EGFR's roles in Osimertinib resistance by inhibiting CAV1 with Methyl-Beta-Cyclodextrin (MBCD) and EGFR with Osimertinib. The data showed that combination of both MBCD and Osimertinib significantly inhibited cell viability by 50% in HCC-827 cells. In conclusion, we observed a synergistic relationship between dysregulated CAV1 and mutant EGFR in Osimertinib resistance in lung cancer, which can be targeted for treatment.

This work was supported by the National Cancer Institute grants R25CA186872 (Bret A. Hassel) and P30CA134274 (Taofeek Owonikoko), and the UMMS Foundation Nathan Schnaper Fund.

P3.01 What Happens to Pain?: An Observational, Longitudinal Study in Patients with Aneurysmal Subarachnoid Hemorrhage Treated in Neurocritical Care Units (WHOL-PAIN)

Presenter: Ruchika Gadagkar[1]
Mentor(s): Nicholas Morris, MD[1, 2, 3]
[1]University of Maryland School of Medicine
[2]Department of Neurology
[3]Program in Trauma

Long-term follow-up of patients with aneurysmal subarachnoid hemorrhage (SAH) suggests that SAH-related chronic pain is an important contributor to opioid use disorders and reductions in health-related quality of life. A gap exists in understanding how sudden-onset worst headache of life transforms into chronic pain. To address this gap, we performed a multicenter, observational, prospective, survey-based study of pain after SAH to characterize pain after SAH throughout the ICU stay and in outpatient follow-up. We included adult patients with primary aneurysmal and perimesenecephalic pattern SAH with a GCS of 15. We excluded patients with secondary SAH. We have enrolled 42 patients with SAH (mean (SD) age 49 (13) y, 81% women, median (IQR) Hunt-Hess 2 (2-3), median (IQR) modified Fisher score 3 (3-3)). Preliminary analyses suggest that headache is near-ubiquitous immediately after SAH (endorsed by 39/42 (92.9%) of patients post-bleed day (PBD) 0-4) and remains common in outpatient follow-up (endorsed by 10/23 (43.5%) at PBD 90-120). Extremity pain, in contrast, increased from admission to outpatient follow-up (5/42(11.9%) at PBD 0-4 to 7/24(29.2%) at PBD 90-120). While headache was the most bothersome pain location for 35/42 (88%) of patients on admission, 5/23 (22%) of patients identified another pain location as most bothersome at outpatient follow-up. Further characterization of pain and analgesic response may inform more targeted therapeutic approaches. Long-term treatment strategies focused solely on headache may be misdirected in a substantial portion of patients after SAH.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

P3.02 Investigating the Role of Expectations in a Virtual Reality Based Intervention in people with Temporomandibular Disorder

Presenter: Ozioma Agoh
Mentor(s): Luana Colloca MD, PhD, MS,
Co-Author(s): Titilola Akintola, PhD
Department of Pain and Translational Symptom Science, University of Maryland School of Nursing, Baltimore, MD

Temporomandibular joint disorder (TMD) is a group of conditions characterized by pain in the jaw joints and the surrounding muscles and ligaments. TMD is often associated with pain, facial discomfort and headaches which can limit jaw movement and significantly decrease the quality of life. Estimates report that up to 30% of adults around the world deal with TMD-related to symptoms, without adequate treatment options, which makes this a critical unmet clinical need. Studies show that expectations about pain and pain treatments are able to modulate the experience of pain, especially for related outcomes such as anxiety, mood and pain unpleasantness that are highly associated with pain. Thus, expectations and expectation management has emerged as a possible solution to managing pain and improve clinical outcomes. This study investigated the effects of a Virtual Reality intervention on pain tolerance in patients with TMD. TMD patients were randomly assigned either an immersive/3D VR (active condition), a non-immersive/2D VR control group or a no VR control group. Measures of treatment expectation were collected before and after each condition and the effect on each treatment on anxiety, mood and pain unpleasantness was assessed after each session. We examined how expectations influence pain VR effects on pain tolerance as well as pain unpleasantness, anxiety and mood in 176 patients with TMD. Findings from this study will inform the design of future expectation-based digital therapeutics and support the use of immersive VR therapeutics in patients with TMD.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

P3.03 Efficacy of Exercise on Improving Cardiovascular Function in Breast Cancer Patients Undergoing Chemotherapy

Presenter: Rachel Ni[1]
Mentor: Ian Kleckner, PhD, MPH[1]
[1]Department of Pain and Translational Symptom Science, Cancer Control Mind & Body Lab, University of Maryland School of Nursing, Baltimore, MD

Chemotherapy, while an essential component of cancer treatment, often disrupts patients' physiological function, resulting in decreased physical activity and a lower quality of life. These effects can persist throughout and even after treatment. Understanding chemotherapy-induced symptoms and how they can be mitigated can enhance patient well-being throughout treatment and recovery. A 25-week single-site randomized controlled trial with a 12-week exercise or nutrition education control intervention was conducted to examine whether exercise improves physical function of patients and whether conditioning responses of the musculoskeletal, cardiovascular, neuropsychological, and immune systems are involved in its mechanism. The objective of this project is to assess the efficacy of exercise on improving cardiovascular function based on the study data. Eighty female breast cancer patients about to or who recently started chemotherapy were assessed at four timepoints: pre- (T1), mid- (T2), post-intervention (T3), follow-up (T4). Participants engaged in standard aerobic tasks, including a 6-minute walk test and 4-minute seated rest and recovery. Physiological data was collected through an ECG device and subsequently imported into HRVanalysis to extract cardiovascular metrics, including Maximum HR, Minimum HR, and Heart Rate Variability (HRV). Statistical models are applied to model the outcome at T2, T3, or T4 using T1 as the covariate and the study arm as the factor to analyze the difference in variables.

This research was supported in part by the UM Scholars at SON, University of Maryland School of Medicine Office of Student Research, and funding from the National Cancer Institute (K07CA221931).

P3.04 Ongoing Study Investigating Virtual Reality as an At-Home Intervention for Temporomandibular Disorder

Presenter: Daphney Waller
Mentor(s): Luana Colloca, MD, PhD, MS; Nandini Raghuraman, MS, PhD[1]
[1]Department of Pain and Translational Symptom Science, University of Maryland Baltimore, School of Nursing

Abstract: Temporomandibular disorder (TMD) is a chronic pain condition that significantly impairs quality of life and disproportionately affects women. Standard treatments are often inaccessible or carry risks of dependency, highlighting the need for effective non-pharmacological alternatives. Virtual reality (VR) offers a promising, low-risk intervention, but its feasibility, acceptability, and underlying mechanisms in TMD remain understudied. We are conducting an ongoing randomized trial aiming to enroll 78 adults with TMD to evaluate the behavioral and neural effects of VR. Here, we present preliminary findings from the first 10 participants who completed all three randomized conditions: Active VR, Sham VR, and No Intervention. At each lab session, participants underwent electroencephalography (EEG) recordings and at-home pre- and post-assessments of pain intensity, unpleasantness, mood, and anxiety.
EEG was used to measure peak alpha frequency (PAF), a neural marker of cortical excitability and pain sensitivity. Higher PAF has been associated with lower pain perception and greater treatment responsiveness.
A linear mixed model revealed no significant main effect of condition on pain intensity ($F(2, 204.104) = 0.303$, $p = 0.739$), though pairwise comparisons showed greater pain reduction with Active VR versus No Intervention (Mean Difference = 3.63, $p < 0.001$) and Sham VR (2.19, $p = 0.001$). A repeated-measures ANOVA revealed a trend toward a condition effect on PAF ($F(3, 4) = 3.41$, $p = 0.053$).
These preliminary findings support the potential of VR as an accessible, mechanistically informed approach for chronic pain management.

This research was supported in part by UM Scholars at the University of Maryland School of Nursing, an initiative of the University of Maryland: MPowering the State.

P3.05 Ongoing Investigation of The Impact of Virtual Reality on Orofacial Pain Intensity and Interference among Participants with Temporomandibular Disorder

Presenter: Kayla Morris[1]
Mentor(s): Luana Colloca, M.D., PhD, MS[1]; Nandini Raghuraman, MS, PhD[1]
[1]Department of Pain and Translational Symptoms Science[1], University of Maryland School of Nursing, Baltimore, MD

Virtual reality (VR) has emerged as a promising non-pharmacological tool for managing chronic pain, with studies showing improvements not only in pain intensity but also in mood, sleep, and anxiety. This study explores the impact of VR on orofacial pain in individuals with temporomandibular disorder (TMD), focusing on changes in pain perception and brain activity. Using a randomized crossover design, participants underwent EEG recordings, heat-pain sensitivity testing, and three intervention conditions: Active VR, Sham VR, and No VR. The goal was to evaluate whether VR could reduce clinical pain and alter cortical excitability, measured through peak alpha frequency (PAF). Participants also completed the Graded Chronic Pain Scale (GCPS) before and after the intervention month to assess changes in pain-related disability and intensity. The GCPS helps categorize individuals into high- and low-impact TMD groups, providing a clinical framework for evaluating the potential benefits of VR. This ongoing trial aims to recruit 78 TMD participants. Here, we report preliminary findings from the first 15 participants who completed all study procedures, including four laboratory visits and pre- and post-pain ratings. A linear mixed model showed no significant main effect of condition on pain intensity ($F(3, 23.741) = 2.179$, $p = 0.117$). Mean pain intensity was highest at baseline (M = 50.89) and decreased across Active VR (M = 35.78), Sham VR (M = 33.78), and No VR (M = 33.56). These early findings suggest VR may help reduce pain intensity in TMD, but additional data are needed to determine its clinical and neural impact.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

P3.06 Troubleshooting Alginate Microcapsules for Oral Mice Trial

Presenter: Madison Parrish[1]
Mentors: Scott Baliban, PhD[1]
[1]Center for Vaccine Development and Global Health, University of Maryland School of Medicine, Baltimore, MD

In sub-Saharan African, non-Typhoidal *Salmonella* (NTS), particularly serovars Enteritidis and Typhimurium, are a leading cause of invasive bacterial disease infants and toddlers. Recently, we developed a novel live-attenuated non-transmissible (LANT) vaccine platform for NTS that displays a shortened shedding period following oral vaccination. We found that our *S.* Typhimurium LANT strain was immunogenic in mice and protective against lethal challenge. However, protection was less robust compared to a non-LANT *S.* Typhimurium vaccine strain, which is associated with longer stool shedding.

LANT vaccines must be delivered orally and safely make it into the intestinal tract to ensure proper vaccination. One of the developmental hurdles for oral vaccine modalities is overcoming stomach acidity, which creates a harsh environment for vaccine antigens. To circumvent this, we are pursuing whether alginate-based microencapsulation can be used as a delivery vehicle for NTS LANT vaccines.

The objective of this project is to create an optimized microencapsulation protocol for NTS LANT vaccines and determine whether this imparts resistance to gastric acid and enzymes.

The hypothesis of this project was that a protocol could be used to encapsule LANT vaccines of *S*. Typhimurium and increase viability of bacteria in acidic conditions.

Bacterial culture incubates overnight at 37^0C. Culture was centrifuged and resuspended in PBS to get targeted OD_{600} per experiment. After achieving targeted OD_{600}, 100uL of culture was mixed with 1% alginate. Then using a 1mL syringe with a 34G needle, 20-30 drops of the bacteria and alginate solution were dropped into a calcium chloride and 1% chitosan solution. After a 10 - 60 minutes incubation period, microcapsules were homogenized in PBS and plated in serial dilutions.

This research was supported in part by the Program for Supporting Undergraduate Members in Microbiology and Immunology Research (SUMMIR), University of Maryland School of Medicine Office of Student Research.

P3.07 mRNA and miRNA Dual Omics Profiling of Ischemia-Reperfusion Injury in Liver Allografts

Presenter: Rishi S. Kumar[1,2]
Mentor(s): Haseeb Zubair, PhD[1]; Valeria R. Mas, PhD[1]
[1]Department of Surgery, University of Maryland School of Medicine, Baltimore, MD
[2]University of Maryland, College Park, College Park, MD

Ischemia-reperfusion injury (IRI) remains a leading cause of graft dysfunction in liver transplant recipients; however, its molecular mechanisms are not yet fully understood. Gene expression profiling can potentially demonstrate the molecular signatures of hepatic IRI, identifying the disrupted transcriptional networks that govern cellular survival, metabolic function, stress responses, and immune cell chemotaxis. MicroRNAs (miRNAs) represent critical post-transcriptional regulators that can simultaneously modulate multiple genes within these disrupted pathways, potentially serving as “master” switches controlling IRI-induced transcriptional remodeling. Thus, miRNAs may orchestrate the coordinated expression changes observed during hepatic allograft ischemia-reperfusion, making them both key mechanistic drivers and promising therapeutic targets. Our approach utilizes comprehensive microarray-based transcriptomic profiling and miRNA expression analysis across liver transplant samples to determine the regulatory networks governing IRI. The experimental design incorporates paired pre- and post-reperfusion comparisons, stratifying deceased donor samples based on injury severity to isolate IRI-specific transcriptional signatures and their regulatory controllers.

This research was supported in part by the Supporting Undergraduate Members in Microbiology and Immunology Research (SUMMIR) program, University of Maryland School of Medicine Office of Student Research.

P3.08 Investigating the Regulation and Expression of Colonization Factor CS1 in Enterotoxigenic *E. coli* (ETEC) Clinical Isolates

Presenter: Mariam Hassan[1]
Mentor(s): Elieen Barry, PhD[1]; Viktoria Van Nederveen, PhD[1]
[1]Center for Vaccine Development, University of Maryland School of Medicine, Baltimore, MD

Enterotoxigenic *Escherichia coli* (ETEC) is a Gram-negative bacterial pathogen that is a significant contributor to diarrheal disease in children under five, especially in low-resource countries. Colonization factors (CFs) like CS1 and CS3 enable bacterial adhesion to the intestinal epithelium, causing infection. Despite being essential to the development of vaccines, little is understood about how environmental factors control the expression of CFs in clinical isolates. This gap limits our ability to improve antigens for vaccinations with broad protection. We hypothesized that growth in different media influenced the expression of CS1 and or CS3, especially iron availability and bile salts, and that genetic variation among clinical isolates affects CS1 and/or CS3 expression. This was shown by initial studies demonstrating different CF expression across different strains and conditions. We conducted an experiment by growing geographically diverse clinical ETEC isolates on bile salt, CFA agar, iron-chelation, and CFA broth media. CF expression was measured by Western blotting, and PCR assessed the presence of structural genes. Only one strain, 60R75, cultured on CFA agar exhibited CS1 expression, whereas the majority of CS1-only clinical isolates displayed no CS1 expression. Although several strains displayed faint or smeared bands, indicating partial gene presence or degradation, PCR confirmed that multiple bacteria didn't have the CS1 genes. A set of $CS1^+$ $CS3^+$ isolates was next evaluated for CF expression. Interestingly, all strains expressed both CFs; the level of CS1 varied between strains, but CS3 was consistent. The future goal is to determine whether host-cell adhesion is enhanced by increased CS1 expression and whether antibodies can prevent this interaction. Also, complete operon sequencing will reveal CF sequence conservation between Isolates.

This research was supported in part by the NIH Pol AI125181 and NIH Rol AI177145

P3.09 Regulation of the Ferritin-like Protein PA4880 in Pseudomonas fluorescens Under Oxidative Stress

Presenter: Savannah Ford
Mentor(s): Amanda Oglesby, PhD; Khady Ouattara

Iron is essential for nearly all organisms, but its redox activity can catalyze harmful oxidative reactions, making it critical for cells to tightly regulate the uptake, use, and storage of iron – a process holistically referred to as iron homeostasis. Because iron can result in oxidative stress, regulation of the oxidative stress response is likely linked to regulation of iron homeostasis. This study investigates how a gene encoding an unusual class of proteins in the ferritin super family is regulated in two highly related bacterial species. Our study set included the PA4880 gene of the nosocomial pathogen *Pseudomonas aeruginosa*, and a homolog (Pf0-1_0567) in the closely related non-pathogenic environmental species *Pseudomonas fluorescens.* Preliminary data in *P. aeruginosa* shows PA4880 is iron-regulated and responsive to oxidative stress, and we sought to determine if this regulation was also observed for Pf0-1_0567. As a surrogate of Pf0-1/PA4880 expression, we used a Pf0-1/PA4880-lacZ reporter strain and conducted β-galactosidase assays on cultures of the reporter strain grown under various conditions: shaking liquid culture (250 rpm), static liquid cultures, and static liquid cultures treated with hydrogen peroxide. My results show that PA4880 reporter activity in Pf0-1 varies with growth conditions and is rapidly induced following H_2O_2 exposure, consistent with its proposed involvement in oxidative stress. These findings suggest that PA4880 and its role in iron homeostasis and oxidative stress are conserved in the Pseudomonas genus. Ongoing work aims to determine the specific regulation of PA4880 by small RNAs or other stress-responsive pathways in *P. aeruginosa* and *P. fluorescens*.

P3.10 A study of Rickettsial Lipase (R Lip.) effector protein in Host Pathogenesis

Presenter: Kinan Bazzi
Mentor(s): Mohammad Sadik, M. Sayeedur Rahman, and Oliver H. Voss
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Rickettsia species are obligate intracellular, gram-negative bacteria. They infect a significant percentage of eukaryotes, including vertebrates like humans, causing diseases such as rickettsial pox, bouton-nausea fever, and multiple strains of typhus. *Rickettsia* is incredibly good at their job, making their lethality rate as high as 30%. For this reason, it is important to study the mechanisms of pathogenicity in order to stop *Rickettsia* from plaguing humans. While *Rickettsia* employs a myriad of effector proteins to facilitate the infection process, one of the seemingly key players is a molecule named *Rickettsia* Lipase (RLip), which codes for a protein that breaks down host lipids. Consequently, we hypothesize that RLip plays an important role in promoting the phagosomal escape of the bacteria after being internalized by the host cell. In order to study the role of RLip, the gene is amplified by PCR, cloned into a plasmid and then transformed into a strain of *Rickettsia*, which does not have a functional gene copy due to transposon mutation. Bacterial replication, pathogenicity, and subcellular localization studies will be conducted to evaluate phenotypical differences between a wild-type *Rickettsia* strain, a RLip transposon mutant strain and a complementary strain that over-expresses the RLip gene. Observed differences are attributed to the presence of the RLip gene, effectively conveying the functional importance of RLip in modulating rickettsial infection in the host.

This research [FR5] was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

P3.11 Epitranscriptomics: Developing a Rapid Diagnostic to Identify Single-Based CRISPR-Induced Deletions in an Epitranscriptomic Writer in *Drosophila melanogaster*

Presenter: Samiatu Yussuf[1]
Mentor(s): Robin Bromley[2]; Julie Dunning Hotopp, PhD[2]
Co-Author(s): Kaylee Watson, PhD[2]
[1]Spelman College, Atlanta GA, USA.
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Epitranscriptomics, the biochemical modifications of RNA transcripts, in an emerging field of study with medical significance. The methylation of bases in mRNA such as 6-methyladenosine (m^6A) and 5-methylcytosine (m^5C) and can alter mRNA stability, translation, and processing. This is of particular interest to infectious disease research because mosquito borne RNA viruses like Dengue, Zika, and Chikungunya use RNA methylation to stabilize their RNA, enhance replication, and suppress host antiviral responses. By mimicking or hijacking host methylation machinery, these viruses gain an advantage in replication and immune evasion. Similarly, *Wolbachia,* a bacterial endosymbiont of some mosquitoes and invertebrates, can alter host RNA methylation and therefore alter transmission of RNA viruses, including transmission of the RNA virus to humans. In order to learn more about epitranscriptomics in insects, we turned to genome-engineering innovations on a lab strain of *Drosophila melanogaster* that is infected with *Wolbachia*. To investigate host RNA methylation, the genome-editing tool, CRISPR-Cas9, enables targeted modification of DNA sequences through precise, targeted deletions or insertions in DNA sequences. The Cas9 protein uses a guide RNA to identify a site in the genome to cut the DNA. Once the DNA is cut, it degrades losing anywhere from a single base to multiple bases. In our research, we employed CRISPR-Cas9 to induce deletions in *Dnmt2* in the *Drosophila melanogaster* genome. *Dnmt2* has been previously identified as a potential enzyme in flies that functions to create base modifications as an m^5C writer. The aim of this project is to create a fly that lacks *Dnmt2* to compare it to the wilde-type fly that has a normal *Dnmt2*. The methodologies I used entailed housing the flies in vials containing a standard cornmeal-agarose media under controlled temperature of 25 °C and relative humidity 60%. The flies then underwent DNA Isolation, Polymerase Chain Reaction (PCR), and Gel Electrophoresis to identify flies with the successful *Dnmt2* gene editing mutation in order to establish a line of m^5C writer mutants. Future work will focus on comparing the *Dnmt2* knockout flies to wild-type to understand the influence *Dnmt2* has on RNA methylation and viral pathogenesis.

This research was supported by the Supporting Undergraduate in Microbiology and Immunology Research, (SUMMIR) program which was funded with the grant MiNIH/NIAID R25AI181751-01.

P3.12 High-Dimensional Flow Cytometry Analysis on CAR-T Cells for Cancer Patients

Presenter: Alisa Davis[1]
Mentor: Xiaoxuan Fan, PhD[1]
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Chimeric antigen receptor (CAR) T cell therapy has shown great promise in treating different types of cancer by using genetically modified T cells that can specifically recognize and destroy cancer cells. However, not all patients respond well to the treatment, and some may experience side effects. Studying the immune system of patients, as well as the CAR-T cells themselves, could help us better understand why responses vary and how to predict which patients are more likely to benefit. To explore this, we developed a 28-marker flow cytometry panel to deeply analyze different T cell subsets. The panel includes 27 monoclonal antibodies, each labeled with a unique fluorochrome, along with a viability dye to exclude dead cells from the analysis. To validate our method, we used T cells from a healthy donor, stained them with all the antibodies, and performed flow cytometry analysis on a Cytek Aurora spectral cytometer. We also used single-color controls and compensation beads to collect the spectral signatures for each fluorochrome, which are used to unmix the data from the fully stained sample. Our goal was to see whether these antibodies can recognize the markers with acceptable resolution and whether all the markers can be distinguished without issues from overlapping fluorescence. The results showed that most markers could be resolved well, suggesting that our panel can be used to analyze T cells in cancer patients before and after CAR-T treatment. In the future, we hope this approach will help identify biomarkers that can predict treatment outcomes.

This research was supported in part by the Supporting Undergraduate Members in Microbiology Immunology Research (SUMMIR) program , University of Maryland School of Medicine Office of Student Research.

P3.13 Peri-hippocampal Mast Cells Influence Oligodendrocyte Development During the Early Postnatal Period

Presenter: Nimah Aime[1]
Mentor(s): Matthew Bruce, PhD[1]; Margaret McCarthy, PhD[1]
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Abstract
Early life immune activation, including allergy, can result in detrimental effects on neurodevelopment. Transient populations of immune cells with distinct transcriptional programming exist within the brain and associated border regions during the macrophages (PMID: 31262353). Activation of these immune cells during sensitive periods of development may contribute to effects seen in models of early life immune challenge. A uniquely proliferative CNS-resident mast cell population exists in the peri-hippocampal area during the first two postnatal weeks of life in rodents. Pharmacological activation of peri-hippocampal mast cells (phMCs) by intraventricular infusion of the specific degranulator c48/80 increases blood-brain barrier permeability, promotes transient monocytic infiltration, and has effects on the local microglial population (PMID:39662467). Additional roles suggested by analysis of gene expression in phMCs compared to other mast cell populations relate to regulation of insulin growth factor and thyroid hormone signaling, both of which are involved in oligodendrocyte development and myelination. We sought to answer whether mast cell activation affects oligodendrocyte maturation in the hippocampus. To examine this, we injected rat pups intracerebroventricularly (I.C.V) wither either c48/80 or saline from postnatal days 9-12, to model chronic mast cell activation. We then conducted immunofluorescent (IF) staining with oligodendrocyte lineage markers (CC1/SOX10/PCNA) to quantify the effects of phMC degranulation on oligodendrocyte maturation states in the hippocampus, with results suggesting enhanced oligodendrocyte maturation in response to c48/80 exposure compared to controls. The data will provide an insight into the role of phMCs in oligodendrocyte development, which may reveal novel mechanisms of early life inflammation induced changes in the brain.

This research was supported in part by the Supporting Undergraduate Members in Immunology and Microbiology Research (SUMMIR), University of Maryland School of Medicine Office of Student Research.

P3.14 Identification of genomic mechanisms of multi-drug resistance in the fungal pathogen *Candida auris*

Presenter: Rafael C. Gonzalez
Mentor(s): Mary Ann Jabra-Rizk, PhD
Co-Author(s): Tristan Wang, PhD
Department of Oncology and Diagnostic Sciences, University of Maryland School of Dentistry, Baltimore, MD.

Candida auris is a newly emerged fungal pathogen associated with invasive disease with mortality rates approaching 68% mainly due to its ability to develop multidrug resistance. In this project, we characterized *C. auris* isolates recovered from infected patients hospitalized at UMB Medical Center. A total of 35 isolates were classified into genetic clades based on ITS, *RHA1, RPB1,* and *RPB2* sequences, and drug resistance to antifungal drugs (amphotericin B, caspofungin and fluconazole) was determined using susceptibility testing. To identify mechanisms of resistance, genes associated with resistance (*FKS1, ERG11)* were amplified and sequenced to identify mutations. Furthermore, isolates were phenotypically evaluated for ability to aggregate and form biofilms, characteristics associated with pathogenesis. Findings demonstrated that 34 of the isolates belonged to clade I and one isolate to clade III. Suceptibility testing indicated that 100% of isolates were resistant to fluconazole, 30% to caspofungin, and 46% to amphotericin B with 17% of isolates resistant to all antifungals. Sequencing of *ERG11* gene identified mutations (Y132F, V125A, and F126L) associated with fluconazole resistance and a mutation (F635Y) in the *FKS1* gene associated with caspofungin resistance. Based on assessment of metabolic activity and using aggregation assays, the isolates were found to differ significantly in ability to form biofilm and to aggregate. Combined, these findings highlight the phenotypic plasticity of this pathogen and provide mechanistic insights that may aid in the identification of new drug targets. Transcriptional analysis to identify differentially modulated genes relevant to drug resistance are currently underway.

This research was supported in part by the Supporting Undergraduate Members in Microbiology and Immunology Research (SUMMIR), University of Maryland School of Medicine Office of Student Research. Investigator support to conduct this research was provided by NIH/NIAID R25AI181751-01. The work in this publication was supported by the National Institute of Allergy and Infectious Diseases of the NIH under award number R01AI130170 (NIAID) to M.A.J-R.

P3.15 Dynamics of "Double Duty" Dendritic Cells (XL Cells) during a Humoral Immune Response in the Amphibian *Xenopus*

Presenter: Jeremi Carlos-Figueroa[1]
Mentor: Martin Flajnik, PhD[1]
Co-Author(s): Erik Cruz[1], Xiwei Peng[1], Yuko Ohta[1]
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Dendritic cells (DCs) are essential antigen-presenting cells that coordinate immune responses, yet their evolutionary history remains underexplored. In mammals, there are two DC populations: conventional DCs (cDCs), which stimulate T cells, and follicular dendritic cells (FDCs), which present antigen to B cells. In the amphibian *Xenopus*, we hypothesize that there is only one DC type (Xenopus laevis cells or XL cells), that can stimulate both T cells in the splenic red pulp and B cells in the white pulp. These are referred to as "double-duty" DCs. We propose that two DC populations (XL1 in red pulp and XL2 in white pulp) differentiate from a precursor that can interconvert during an adaptive immune response. To test this, frogs were immunized with a foreign antigen, and spleens were collected at multiple time points from day 2 to day 9. Splenic fragments were processed for immunohistochemistry (IHC), and single-cell suspensions were analyzed by flow cytometry (FACS). Monoclonal antibodies (mAbs) specific for MHC class II and CD45, which label both DC populations, were used. Fc receptors for *Xenopus* IgM and IgG(Y) and a complement receptor-specific mAb were used to identify XL2. We also tested peanut agglutinin lectin as a predicted DC-specific marker. FACS and IHC were used to visualize DC migration and define subset distribution over time, with the prediction that XL1 presents antigen to T cells early and transitions into XL2 to sustain B cell stimulation.

This research was supported in part by the Summer Undergraduate Members in Microbiology and Immunology Research (SUMMIR) Program and NIH/NIAID R25AI181751-01.

P3.16 Gene Expression Differences in Cerebral vs. Asymptomatic Malaria in Malawian Children

Presenter: Yaneysi Quezada
Mentor(s): Mark Travassos, MD[1]
Co-Author(s): Jonathan Lawton, PhD[1]
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Introduction
Malaria, a mosquito-borne disease caused by the deadly parasite Plasmodium falciparum, can lead to a wide range of presentations in children. This includes cerebral malaria, the deadliest form of severe malaria, and asymptomatic infections, where no symptoms are present. Understanding how parasite gene expression differs between these clinical outcomes may reveal genes associated with disease severity and immune evasion.
Objective
Through computational work, we aim to determine which P. falciparum genes are differentially expressed in cerebral vs. asymptomatic malaria, to identify parasite contributors to infection severity.
Methods
We compared gene expression in P. falciparum samples collected from 19 Malawian children diagnosed with malaria, 9 with cerebral and 10 asymptomatic infections. RNA was extracted from whole blood samples and analyzed using RNA-seq. Differential gene expression analysis was performed in RStudio using the edgeR package to identify genes that were significantly upregulated or downregulated in severe cases compared to asymptomatic ones.
Results
Preliminary analysis revealed a distinct set of genes that were highly upregulated in cerebral malaria and significantly downregulated in asymptomatic cases. A key finding was that the two most differentially expressed genes in cerebral malaria PF3D7_0731900 and PF3D7_1150300 both belong to the RIFIN gene family.
Conclusions
The elevated expression of RIFIN genes in Malawian children with cerebral malaria suggests they may play a key role in the severity of the disease. Given their known involvement in immune evasion and cytoadherence, these genes may contribute to the parasite's ability to avoid host defenses and cause more severe neurological symptoms

UMScholars/MPower:
This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State

P3.17 Assessing EV-D68 entry following ATG-14 knockdown

Presenter: Princaya Sanders
Mentor(s): William Jackson, PhD

Enterovirus-D68 (EV-D68) is a member of the picornaviridae family, of positive-sense single stranded RNA viruses, associated with respiratory illnesses. EV-D68 is also associated with acute flaccid myelitis (AFM), a paralytic disease that primarily affects children. Work from our lab and others has identified the autophagy pathway, a degradative cellular process, to be critical in EV-D68 replication. We have identified the host autophagy protein ATG14 as a critical component for EV-D68 replication, although the exact mechanism is currently unknown. This goal of the project is to identify which stage of the EV-D68's life cycle ATG-14. We first asked whether ATG-14 has an impact on the virus's entry into cells. To begin answering this, we knocked down ATG-14 expression in HeLa cells using small interfering RNA (siRNA). We then performed an entry assay to see if the virus's ability to enter the cells was affected by the loss of ATG-14. A western blot confirmed a successful knockdown, and a plaque assay was used to quantify viral entry. Our results showed that both the ATG-14 knockdown samples and the scrambled RNA control samples had a similar number of viral entry events. These results show that knocking down ATG-14 is unlikely to influence viral entry of EV-D68. With this step ruled out, our lab will now investigate whether ATG-14 impacts the later steps in virus replication.

P3.18 Evaluation and selection of optimal antigen expression cassettes to maximize the immunogenicity of non-replicating adenoviral vectored vaccines.

Presenter: Sydney Weir[1]
Mentor(s): Aisha Souquette, PhD[2]; Lynda Coughlan, PhD[2]
[1]Department of Biological Sciences, Towson University, Towson, MD
[2]Department of Microbiology and Immunology, University of Maryland Baltimore School of Medicine, Baltimore, MD

Non-replicating adenoviral (Ad) vectors are promising vaccine platforms and offer many benefits including rapid, cheap production, a well-established safety profile in humans, and simultaneous induction of cellular and humoral immunity. These vectors can be easily manipulated to incorporate and express a vaccine antigen of choice. However, the optimal combination of pre- and post-transcriptional regulatory elements to maximize expression of the encoded antigen is unknown. As maximizing antigen expression may improve vaccine potency, this could allow for reduction in the cost-per-dose and reactogenicity, and thus an enhanced ability to distribute vaccines worldwide. To address this, we have designed and tested six Ad constructs with modified CMV promoters, leader signal sequences, and/or post-transcriptional regulatory elements to determine which candidate elicits maximal immunogenicity.

Seven groups of Balbc/J mice were vaccinated with PBS (G1), or distinct modified Ad vaccines (G2-G7) encoding enhanced green fluorescent protein (EGFP). Spleens and serum samples were collected 28 days post-vaccination. Preliminary flow cytometry data showed that Groups 4 and 7 induced T cells with increased production of anti-viral IFNγ, and Group 6 induced more CD107a+ expression. Anti-EGFP IgG ELISAs showed that Group 6 was the most immunogenic construct, as determined by induction of higher anti-EGFP antibodies at lower serum concentrations, and high area under the curve (AUC) and endpoint titers.

These data suggest that using the tPA signal sequence, and post-transcriptional regulatory element WHVPRE in combination, may be optimal for maximizing immunogenicity to Ad-encoded vaccine antigens. Future experiments will investigate the mechanism underlying the observed immunogenicity, such as assessing differences in EGFP expression across the constructs, and testing at lower vaccine doses to evaluate maintenance of immunogenicity. These results provide novel insight on optimal Ad vector design and could ultimately contribute to improved vaccination against pathogens with pandemic potential, such as influenza and SARS-CoV-2 when encoding a disease-specific antigen.

This research was supported in part by the Supporting Undergraduate Members in Microbiology and Immunology Research (SUMMIR) Program, University of Maryland School of Medicine Office of Student Research (NIH/NIAID R25AI181751-01) and NIH/NIAID R01AI148369 (L.C.).

P3.19 Impact of Social Discrepancy on Placebo Hypoalgesia: Preliminary Analyses

Presenter: Zuzana Huserova[1]
Mentor(s): Belina Rodrigues, PhD[1]; Luana Colloca, MD, PhD, MS[1];
[1]Department of Pain and Translational Symptom Science, University of Maryland School of Nursing, Baltimore, MD

Prior research has proven observation influences pain perception in the form of the placebo and nocebo effect. However, the role of expectation—and violations of expectation—in social contexts remains poorly understood. This study investigates the effect of social discrepancy between expected and observed social cues on placebo-induced hypoalgesia.
Participants were exposed to color cues, different levels of painful stimuli, and facial expressions. Specifically, on day 1, following individualized pain calibration, participants learned to associate the red fearful face with high pain, the yellow neutral face with medium pain, and the green happy face with low pain. On day 2, participants rated their pain while fMRI/EEG was simultaneously recorded. Half of the color-face cues were randomly mismatched to violate expectations. Unbeknownst to participants, the pain intensity was fixed at a medium level for all trials.
Pain ratings were extracted from text files, along with information on each trial (type of trial-match/mismatch, and type of color cue) using MATLAB. Our preliminary behavioral results demonstrated the interaction between the condition and cues was significant ($p<0.001$). Pair-wise comparisons applying Bonferroni corrections indicated that only within the matched condition, pain intensity ratings for the red cues were significantly greater than that for the yellow cues ($p<0.001$) and green cues ($p<0.001$). No cues differences were observed in the mismatched condition.
Future integration of EEG/fMRI data will clarify the neural mechanisms involved in expectancy violation.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

P3.20 A Comparative Analysis of Decipher Genomic Classifier and Artera Multi-Modal AI for Risk Stratification and Treatment Optimization in Non-Metastatic Prostate Cancer

Presenter: Olivia Jordan[1]
Mentor: Phuoc Tran, MD, PhD[2,]
Co-Author(s): Adeniyi A. Olabumuyi[2]; Claudia Datnow-Martinez[2]; Phuoc T. Tran[2]
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Prostate cancer remains a prevalent and clinically complex malignancy among men. Traditional risk assessment uses tumor stage, PSA levels, and Gleason score. Recently, molecular and imaging-based tools like Decipher Genomic Classifier (GC) and Artera Multi-Modal AI (MMAI) have been introduced to improve prognostic accuracy for localized disease. GC guides treatment intensification in unfavorable intermediate-risk (UIR) patients in the NRG GU010 trial, while MMAI has shown utility in predicting short-term androgen deprivation therapy (ST-ADT) sensitivity. Both are clinically validated and increasingly inform treatment strategies. Based on this framework, our study focused on non-metastatic prostate cancer patients treated at the University of Maryland Medical Center who received both tests between discrete timepoints. We evaluated the correlation between GC and MMAI risk scores, analyzed correlation differences between non-metastatic and oligometastatic disease, and assessed variation within UIR patients compared to broader risk groups. We compared MMAI's ST-ADT treatment recommendations with GC-based cut-off points employed in the GU010 trial. GC scores were generated through RNA profiling. MMAI scores were derived from pathology images combined with clinical data. Statistical analyses included Pearson correlation, cross-tabulation, and diagnostic performance metrics. By December 2024, 76 patients showed moderate overall correlation between GC and MMAI scores, with stronger correlation in UIR patients. Risk groupings correlated significantly. GC and MMAI demonstrated variable outcomes for ST-ADT classification. GC and MMAI offer distinct but complementary insights into prostate cancer risk and treatment decision-making.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research and the Radiation Oncology Summer Program.

P3.21 Continuing the CATALYST Program: Teacher Researchers Connecting Cancer Research to the Classroom

Presenter: Cynthia F. White
Mentor(s): Bret Hassel, PhD; Stuart Martin PhD.; Michelle Vitolo PhD.; Keyata Thompson M.S., M.B.A.
The Martin Laboratory, University of Maryland School of Medicine, Baltimore, MD

Cancer is one of the leading causes of death within the United States. Creating an educated population of young cancer-literate students is of utmost importance to inspire future leaders in cancer research and prevention for our society. The ***CA***ncer ***T***raining ***A***ffecting ***L***ives of ***Y***oung ***S***cientists and ***T***eachers (***CATALYST***) program at the University of Maryland Greenebaum Comprehensive Cancer Center (UMGCCC) recruits middle and high school teachers to conduct summer research with UMGCCC faculty mentors. Teacher research methods and outcomes are piloted in their own classrooms and used to develop curriculum material for scholars in the UMB CURE program.

Circulating **T**umor **C**ells derived from TNBC (Triple Negative Breast Cancer) create tubulin-based projections called microtentacles (McTNs). Accumulating evidence indicates the significant role of microtubule **P**ost **T**ranslational **M**odifications, such as polyglutamylation brought about by tubulin tyrosine-like ligases, like TTLL-11, on cancer behavior. Altered polyglutamylation is linked to tumorigenesis and resistance to chemotherapeutic drugs. We studied the impact of the enzyme TTLL-11 on BT-549 tumor cell McTN invasiveness as measured by gelatin degradation.

Following their own research, teachers will create curriculum for the UMB CURE program and their own students to learn basic lab techniques and concepts relating to cancer research. Lessons from year one were piloted in teachers' individual classrooms and will be presented to UMB CURE scholars during the fall of year 2l. Biotechnology and scientific journal articles and posters were incorporated into existing science curricula to create cancer connections and expose students to research methodologies.

P3.22 Bringing Cancer Research to the Classroom: a novel immunotherapeutic approach for Ovarian Cancer as foundational material for curriculum development

Presenter: Brittney Shaw Ahenda[1]
Mentor(s): Tonya Webb
Co- Authors: Yuyi Zhu[3]; Bret Hassel [1,2,3,]; Tonya Webb[2 ,3]
[1] Cancer Training Affecting the Lives of Young Scientists and Teachers
[2] Department of Microbiology & Immunology
[3] Marlene and Stewart Greenebaum Comprehensive Cancer Center

Ovarian Cancer (OC) is a solid tumor with a poor prognosis. OC uses multiple mechanisms to suppress anti-tumor immunity. In particular, OC has been shown to downregulate antigen-presentation machinery that is essential for its recognition by the immune system and elimination by cytotoxic T cells. To circumvent this, we have developed chimeric antigen receptor T (CAR-T) cells, which utilize unique single chain antibodies (nanobodies) derived from the target growth factor receptors EGFR and HER3 that are highly overexpressed in OC. We hypothesize that the small size, stability, high affinity of nanobodies as well as their capacity to target multiple antigens, will provide an advantage over standard antibodies which have not been effective for OC. We have demonstrated that OC cell lines express EGFR and HER3 that will be used as a model system to test nanobody-CAR-T killing. This study was conducted by middle school (MS) teachers who gain experience in laboratory research with the goal of developing novel curriculum material that will educate students about biomedical research and inspire their interest in STEM subjects. The broad concepts and technical approaches of this work will be adapted into accessible research experiences for middle and high school students to explore topics such as antibody-mediated immunity, genetic engineering, nanotechnology, and gene/cellular therapy.

The research reported in this presentation was supported by the National Cancer Institute of the National Institutes of Health under the Catalyzing Cancer Research among Urban Underrepresented Minority Youths and Teachers (CATALYST) Program with Award Number R25CA274166 to Drs. C. Adebamowo, B. Hassel and S. Adebamowo, and the P30 Cancer Center Support Grant under Award Number P30CA134274 to Dr. T. K. Owonikoko. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

P3.23 Randomized Control Trial of Flange Fitting for NICU Pumping Parents to Explore Improved Milk Production and Satisfaction

Presenter: Hooria Umer
Mentor(s): Tonya Rachel[1] Breman PhD, MPH, RN, FAWHONN; Hannah McGraw[2] MS, RNC-OB, C-EFM, IBCLC; Christina Berlett[2] BSN, RN, IBCLC
[1]University of Maryland School of Nursing, Baltimore, MD
[2]University of Maryland Medical Center, Baltimore, MD

Breastfeeding is highly recommended because it promotes long-term growth and development of infants. Newborn infants in the NICU often struggle with latching. Since early breastfeeding success is a strong indicator of future breastfeeding success, NICU mothers often use a breast pump to supply breast milk for their infant. To use a breast pump, one must place a flange on their nipple. The purpose of this study is to observe any difference between the Flange FITS™ Guide and traditional care. We will focus on milk output and comfort measures. This study will utilize a randomized controlled trial. We will use a sample size of 130 participants. 65 participants will be from the control group that will receive traditional care. 65 participants will be from the intervention group that will be fitted for their flange using the Flange FITS™ Guide. These participants will attempt to pump 6-8 times a day with their assigned flange size. Throughout the study, their breast milk output will be collected and weighed. The participants will also answer a survey questionnaire about their comfort level during breast pumping. This study is still in its initial stages, but we hypothesize that the participants fitted for their flange using the Flange FITS™ Guide will produce more milk and have a more comfortable experience than participants who receive traditional care.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

P3.24 Ongoing Study: Virtual Reality as a Home-Based Pain and Wellness Intervention in Cancer Survivors

Presenter: Megha Chander
Mentor: Luana Colloca, MD, PhD, MS[1]
[1]Department of Pain and Translational Symptom Science, University of Maryland School of Nursing, Baltimore, MD

Breast cancer survivors often experience persistent symptoms such as chronic pain, anxiety, fatigue, and sleep disturbances long after treatment concludes. These symptoms can contribute to polypharmacy, decreased quality of life, and increased healthcare burden. This ongoing clinical study investigates a novel, home-based Virtual Reality (VR) intervention as a complementary, non-pharmacological approach for managing chronic symptoms induced by cancer and cancer treatment. The immersive VR experience promotes relaxation, emotional regulation, and self-efficacy. A total of 30 breast cancer patients will be enrolled in a 10-week crossover pilot study, alternating between four weeks of Active VR therapy and four weeks of a control condition using a standard MP4 audio module, with a brief washout period between phases. All sessions are completed remotely in participants' homes, with onboarding, user training, technical support, and check-ins to encourage adherence. Participants report symptom data, pain, fatigue, anxiety, and sleep quality daily via an online platform, enabling real-time tracking of symptom trends and engagement. A repeated-measures ANOVA will be used to assess within-subject changes across conditions and determine the intervention's effectiveness on the key outcome measures of pain, fatigue, anxiety, mood changes, and sleep. If successful, this scalable, low-risk intervention could serve as a low-risk treatment alternative or adjunct to conventional symptom management strategies for cancer survivors coping with chronic symptoms.

P3.25 Effects of Immersive Virtual Reality on Experimental and Clinical Pain in Patients with Temporomandibular Disorder (TMD)

Presenter: Ayanna Arroyo[1]
Mentor: Luana Colloca, MD, PhD, MS[1]; Titilola Akintola, PhD[1]
[1]Department of Pain and Translational Symptom Science, University of Maryland School of Nursing, Baltimore, MD

Temporomandibular joint disorder (TMD) refers to a group of conditions affecting the temporomandibular joint leading to persistent jaw pain and dysfunction. While TMD is estimated to affect up to 30% of the general population with a higher prevalence in women, and despite decades of research, there are still limited effective treatments. Virtual reality (VR) is a therapeutic, non-pharmacological tool shown to reduce pain and is being explored as a potential therapeutic solution for chronic pain. While previous studies have demonstrated VR's effectiveness in various chronic pain populations, its role in TMD, remains underexplored. The aim of this study was to evaluate the effects of immersive VR on ischemic pain tolerance in patients with TMD and to assess the impact of VR on clinical TMD pain, as an exploratory outcome. TMD patients were randomly assigned to one of three groups: immersive/3D VR (active condition), non-immersive/2D VR (sham condition), or no VR (control). Ischemic pain tolerance was assessed using a blood pressure cuff inflated to induce ischemic pain, and participants were timed on how long they could tolerate the pain stimulus. We found a main effect of the VR group ($p = 0.008$) such that patients in the immersive VR group showed increased ischemic pain tolerance compared to the no-VR groups ($p = 0.006$) but not the 2D control ($p = 0.13$). These preliminary results show that VR may be an effective strategy to increase pain tolerance in patients with TMD. Future studies will investigate the role of the endogenous opioid system in VR effects. Overall, these findings support the use of VR as a potential therapeutic approach in chronic pain management, particularly for those with TMD. This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

P4.01 Investigating Differential Gene Expression in Two Severe Malaria Syndromes in Mali

Presenter: Haley Kim
Mentor(s): Mark Travassos, MD[1]
Co-Author(s): Jonathan Lawton, PhD[1], Emily Stucke, PhD[1], Bryan Cummings[1]
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Malaria is one of the leading causes of death in sub-Saharan Africa, disproportionately affecting infants and young children. The disease occurs when the *Anopheles* mosquito bites its host and transmits the parasite, *Plasmodium falciparum.* It has a wide range of symptoms categorized as uncomplicated, mild, and severe. The primary types of severe Malaria are severe malarial anemia (SMA) and cerebral malaria (CM). SMA is marked by an extremely low red blood cell count, where a blood transfusion is crucial and CM occurs when infected erythrocytes clump together, clogging the small vessels in the brain, leading to seizures, comas, and death. The aim of our research is to compare gene expression in that of SMA vs CM to find specific genes and biological pathways that are attributed with each of these severe malaria syndromes. We hypothesize that there are gene expression patterns that are distinct to each of these phenotypes. Furthermore, we expect that many of our previously identified genes of interest will be present in this comparison which will reveal shared pathological mechanisms between SMA and CM. In a case study in Mali, RNA sequencing data was collected from blood samples of 129 subjects. We used EdgeR to analyze the data for 4439 genes in the reference genome 3D7 and identified 335 of those that were differentially expressed between SMA and CM using an False Discovery Rate (FDR) value of less than 0.05. Another list of 137 genes of interest was compiled from a previous study looking at differentially expressed genes between uncomplicated malaria (UM) vs CM (without a history of CM), UM vs CM (with a history of CM), and concurrent CM and SMA (without a history of CM). These genes were cross-analyzed to our list of 4439 genes. In this comparison, we found 6 genes of interest were differentially expressed (FDR < 0.05) between SMA and CM. A pathway analysis was also performed on these genes indicating most classified genes played a role in catalytic activity and binding in the cell. In conclusion, we found shared gene expression profiles associated with SMA vs CM showing shared molecular processes between the two syndromes contributing to the mechanisms of the disease. A whole transcriptome analysis of SMA vs CM can give more insights on the pathogenesis of severe malaria and help identify targets for future treatments.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

P4.02 Time to Patent Bloodstream Infection for Males Versus Females in Controlled Human Malaria Infection at the University of Maryland

Presenter: Paige Bristow[1]
Mentor: Matthew Laurens, MD, MPH[1]
[1]Center for Vaccine Development and Global Health, University of Maryland School of Medicine, Baltimore, Maryland, USA

According to the World Health Organization, in 2023, there were an estimated 263 million malaria cases, with 94% occurring in Africa. The University of Maryland, Baltimore, Center for Vaccine Development and Global Health (UMB-CVD) is a major site for controlled human malaria infection (CHMI) studies that serve to advance treatments and to develop effective vaccines. Previous studies have shown biological sex-based differences may play a role in *Plasmodium falciparum* malaria infection and host response. In particular, sex hormones are thought to influence the human immune response, leaving males more vulnerable to greater parasite burden and infection severity. This project investigated if, among participants in CHMI studies at UMB-CVD, unvaccinated males versus females from the infectivity control group experience different time to detectable malaria blood stage infection to better inform the design of the CHMI trials. Data from the UMB-CVD CHMI trials from 1971 to present were reviewed and analyzed. Initial analyses of 42 participants show no difference in time to detectable blood stream infection via PCR; however, a significant difference was found between males and females (10.2 vs 11.2 days, p-value 0.0290) for time to parasite positivity via thick blood smear. Further research will be done to expand the sample size to characterize this relationship. If confirmed, future CHMI studies will need to account for sex differences among participants.

This research was supported in part by the **M4I: Maryland Infection, Immunization, Intervention, and Impact Training Program**, University of Maryland School of Medicine Office of Student Research.

P4.03 Identifying Parasite Targets of Whole-Organism Vaccination through Whole Genome Sieve Analysis, Using Plasmodium falciparum Isolates from Trials of Sporozoite-based Vaccines

Presenter: Jack Mason
Mentor(s): Joana Carneiro da Silva, PhD
Institute for Genome Sciences

Plasmodium falciparum (Pf), the most virulent agent of human malaria, is responsible for the majority of malaria-related deaths. Although two subunit vaccines against Pf are currently employed, which use the parasite protein PfCSP as an immunogen, both demonstrate efficacies below the World Health Organization's proposed 90%. This suggests that new protective targets should be identified and incorporated into the next generation of vaccines against Pf. Whole Genome Sieve Analysis (SA_{WG}), using infections collected from vaccinee and placebo recipients from field efficacy trials of a whole organism-based vaccine, has recently been proposed as a novel approach to identify the pathogenic targets of a protective immune response. SA_{WG} has now been applied to samples collected during two malaria vaccine field trials, conducted in Mali and Burkina Faso, using whole Pf sporozoites (PfSPZ) as immunogens, resulting in the identification of twelve putative Pf protection targets across both SA_{WG} studies. Here a new SA_{WG} study will be conducted on samples from a third PfSPZ vaccine efficacy trial originating in Gabon. Since its Pf population is genetically distinct from those found in Mali and Burkina Faso, this new study will reveal the robustness of the previously identified twelve targets across Pf populations. As protective antigens are ideal vaccine immunogens, the identification of overlapping vaccine targets across these countries would support their inclusion in future malaria subunit vaccines. First, samples within the Gabon study approaching inclusion eligibility via quality control statistics were sent for resequencing. Afterward, all samples were contrasted against the 3D7 *P. falciparum* reference genome, constituting the SNP calling phase, and quality control statistics were once again generated. Then, joint SNP calling was used to genotype multiple samples and generate vcf files, which were filtered and annotated to identify the location of the identified SNPs across the genome. Next, Wright's fixation index, F_{ST}, will be used to determine whether significant differences exist between the Pf genomes from vaccinee and control infections. The list of significantly differentiated sites will be used to identify the protein-coding loci to which they map, which will then be compared with the twelve previously identified in the Mali and Burkina Faso analyses.

This research was supported in part by the Maryland Infection, Immunization, Intervention, and Impact Training Program (M4I), University of Maryland School of Medicine Office of Student Research.

P4.04 Cross-reactivity among *Plasmodium falciparum* strains

Presenter: Mary Mae Robinson [1]
Mentor(s): Andrea Berry, MD, MS, FPIDS[1]
[1]Center for Vaccine Development and Global Health, University of Maryland School of Medicine, Baltimore, MD

Malaria persists as a threat to the lives and well-being of children and adults across the planet. Among the efforts aimed towards improving the outcomes of this infection is the development of a vaccine that covers the diversity of the most prevalent malaria-causing parasite *Plasmodium falciparum* (Pf). This project aims to explore the antigenic diversity of Pf with the goal of defining serogroups—or regions of cross-reactivity among strains. Through a series of repeated Controlled Human Malaria Infections over the course of two years, 8 malaria-naïve participants were challenged with the NF54 strain of Pf up to 4 times. Seroreactivity of the elicited antibody response was measured throughout each infection via fluorescence intensity of peptide microarrays containing 287 NF54 proteins and their corresponding variants present in other strains. Two immunodominant epitopes were identified for each NF54 protein by selecting two distinct regions with the highest reactivity and a significant increase between pre- and post-infection states. The amino acid sequences of these epitopes were used to search the corresponding variant protein sequences, noting the closest match. I hypothesized that the seroreactivity ratio of any given variant to the NF54 epitope would reflect the amino acid divergence between sequences, with changes in polarity or charge decreasing the ratio and conservative changes driving the ratio towards 1. Reactivity ratios among close matches will be assessed for amino acid substitutions, with focus on ratios significantly less than 1, which suggest a strain-specific response, and ratios equal to 1, which lead to a definition for serogroup phenotypes.

This research was supported in part by Maryland Infection, Immunization, Intervention, and Impact Training Program (M4I), University of Maryland School of Medicine Office of Student Research.

P4.05 Contribution of Antigen-Specific and Bystander T Cell Responses to Disease Tolerance After a Malaria Infection

Presenter: Maxwell Heath[1,2]
Mentor(s): Nevil Singh, PhD[1]
Co-Author(s): Katrina Gorga[1]
[1]Department of Microbiology and Immunology, University of Maryland School of Medicine, Baltimore, MD
[2]Supporting Undergraduate Members in Microbiology and Immunology Research (SUMMIR) Program

Humans are typically routinely exposed to multiple pathogens, which alters different aspects of their immune systems. How these alterations affect their ability to deal with a new infection is not known. Importantly, laboratory mice used in biomedical research are typically kept in clean "specific pathogen free (SPF)" accommodations, which means that they are not exposed to such constant threats. In previous studies, our lab has started to tackle this issue using sequential infections with flu and malaria. They found that malaria infection altered the number of flu-specific CD8+ cells in the lungs even though these are very different pathogens. After a sequential Flu-Malaria experience, they also found that mice had altered responses to a third pathogen – Bordetella pertussis, which causes whooping cough. The flu and malaria experienced mice had similar Bordetella infectious loads as SPF mice, but they lost less weight during Bordetella infection, indicating that prior infections can increase future unrelated disease tolerance. My project examines the cellular mechanisms of this process, by focusing on T cells. Since malaria is a common infection in many parts of the world, how it changes T cells and affects disease tolerance could be important to consider in terms of vaccine efficacy and other immune treatments. We hypothesize that malaria infection alters both antigen-specific and bystander T cells to generate a disease tolerance phenotype. In order to study this, we generated a malarial parasite model that transgenically expresses a T cell antigen. I used labelled T cells transferred to mice, followed by Plasmodium challenge to examine how antigen-specific and non-specific responses affect T cell phenotypes. I found that both kinds of T cell responses occur during an infection. I am currently evaluating key molecules identified in disease tolerant T cells, to establish if they change in antigen-specific or non-specific fashion.

Supporting Undergraduate Members in Microbiology and Immunology Research (SUMMIR) Program Investigator support to conduct this research was provided by NIH/NIAID R25AI181751-01.

P4.06 Shifting immune profile of clinical pertussis isolates.

Presenter: Allison Fitzgerald[1]
Mentor(s): Ciaran Skerry, PHD[2]
Co-Authors: Sasha Cardozo[2], David Rickert[2], Riley Himmelberger[2]
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Whooping cough is a highly contagious disease that affects approximately 170,000 people worldwide and is characterized by coughing fits, breathing issues and respiratory-based symptoms. Pertussis incidence continues to rise, despite widespread vaccination, due to waning and sub-optimal immunity induced by current vaccines. To optimize future vaccination strategies, it is essential to have a complete understanding of host-pathogen interactions during *B. pertussis* infection. Currently, B. pertussis laboratory strain, Tohama 1 contains a NOD1 activating peptidoglycan (PGN) and is positive for the adhesion protein pertactin (PRN). However, there are increasing reports of whooping cough caused by pertactin negative strains of pertussis and recent reports suggest an evolving NOD activating profile for *B. pertussis* clinical isolates. To determine the immune stimulating profile of recent clinical isolates, we isolated culture supernatants from logarithmically growing cultures and assessed the ability of shed PGNs to stimulate NOD1 or NOD2 reporter cell lines. We hypothesized that recent clinical isolates, known to express altered levels of pertussis virulence factor pertussis toxin (PtxP1, PtxP2, PtxP3) would have altered immune activating profiles from the established laboratory strain Tohama 1 (TOH1). The strains that were tested included: PtxP3 1917 *B. pertussis*, PtxP1 1920 *B. pertussis*, PtxP1 1834 *B. pertussis*, Tohama 1 *B. pertussis* (+) and 86NY *B. pertussis* (-). The cell assays aimed to distinguish a difference between the NOD1 and NOD2 activating potential of these strains. The reporter assays completed confirmed the initial hypothesis in which the *B. pertussis* clinical isolates demonstrated NOD2 biased response unlike the primary laboratory strain of *B. pertussis* Tohama I. Understanding the nature of the antigens present and immune responses stimulated by circulating clinical isolates will be essential for the development of novel vaccine candidates.

Investigator support to conduct this research was provided by NIH/NIAID R25AI28752-02. We gratefully acknowledge the support and contributions of the Microbiology and Immunology department, School of Medicine (SOM) UMB.

P4.07 Impact Of mtDNA Thresholds on Single Cell RNA-seq Analysis Outcomes in *Bordatella Pertussis*

Presenter: Alyssa Reddy[1]
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Bordetella pertussis (*B. pertussis*) is the causative agent of whooping cough, a respiratory illness characterized by severe coughing and airway inflammation. Despite widespread vaccination, disease incidence is rising, highlighting the need for improved therapies. To better understand the nature of host-pathogen interactions and identify therapeutic targets or barriers to successful vaccination, we performed single-cell RNA (scRNA-seq) sequencing on *B. pertussis* infected lung tissue. The data generated from scRNA-seq is powerful for the dissection of cellular responses during infection. However, in existing pipelines for scRNA-seq analysis, quality control steps often rely on arbitrary thresholds, particularly for mitochondrial DNA (mtDNA) content, which is commonly capped at 5% to disregard dying or stressed cells. This 5% threshold level can be useful in specific contexts, but during infection, may unintentionally exclude biologically relevant cell populations. We hypothesize this cut off may exclude cells with high levels of cellular stress, proliferation, and metabolic activity due to infection that may naturally elevate mtDNA content. Preliminary analysis of B. pertussis-infected and uninfected scRNA-seq datasets using differential gene expression and data visualization with R package, Seurat, demonstrates that applying the standard 5% mtDNA cutoff substantially alters both the total number of cells retained and the diversity of cell types identified. Increasing the mtDNA cutoff from 5% to 7% led to identification of eleven alveolar fibroblast type 1 cells (AF1s) that were previously undetected. Our findings highlight the risk of excluding relevant cell populations in disease settings and suggest that manual evaluation of quality control limits with scRNA-seq data may expand the scope of cells identified and analyzed.

Investigator support to conduct this research was provided by NIH/NIAID R25AI282752-02 and R01AI167947.

P4.08 Mapping Mindfulness to Reduce Psycho-social Suffering for Adolescents and Young Adults with Cancer

Presenter: Christina Lee[1]
Mentor(s): Kim Mooney-Doyle PhD, RN, CPNP-AC, FAAN[2]; Jessica Thompkins BSN, RN, CPN, PhD Candidate[3]

Background: Each year, nearly 90,000 adolescents and young adults (AYA) receive life-altering cancer diagnoses, experiencing substantial psychosocial challenges related to disease, treatment, and perceived life threat. Additionally, AYA and their families frequently encounter barriers such as financial instability and limited healthcare access, further exacerbated by negative social determinants of health (SDoH). These factors highlight the critical need for accessible, cost-effective psychosocial interventions. Thus, mindfulness-based strategies represent an underexplored, promising approach that offers flexibility, affordability, and potential relief from psychosocial suffering among AYA.

Methods: Employing an intervention mapping approach, we conducted a scoping review to examine the effectiveness of mindfulness interventions' effectiveness on psychosocial suffering in AYA undergoing cancer treatment. Following the JBI methodological framework, our team systematically searched various databases utilizing keywords focused on mindfulness therapies, AYA, and cancer diagnoses. Particular attention was given to identifying the core components of effective mindfulness interventions.

Results: Twenty-four articles from 2010 to 2025 met inclusion criteria. Preliminary findings indicated that most studies were pilot feasibility trials with qualitative and quantitative designs, incorporating various modalities including technology-based interventions and movement therapies. Intervention mapping enabled identification of key intervention components necessary for designing comprehensive and holistic mindfulness-based programs tailored to AYA with cancer.

Conclusion: Addressing these identified gaps, future interventions should explicitly integrate family-based components, consider SDoH, and utilize hybrid delivery methods to enhance accessibility and adherence. Through intervention mapping, researchers can build upon existing literature, emerging evidence, and lived experiences of AYA with cancer to develop acceptable, feasible, and effective mindfulness-based interventions.

UM Scholars/MPower: This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

P4.09 Estrogen Modulation of Inflammatory Cytokine Responses in 3D Human Trophoblast Organoids

Presenter: Abigail Li[1]
Mentor(s): Irina Burd, MD, PhD[1]; Jun Lei, MD, PhD[1]
Co-Author(s): Anguo Liu, PhD[1]
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Estrogen plays an important role in maintaining immune tolerance and regulating inflammation at the maternal-fetal interface during pregnancy. Disruptions in estrogen signaling—due to factors such as maternal stress, infection, diet, or environmental exposures—may sensitize the placenta to inflammatory triggers, contributing to adverse outcomes like preeclampsia and preterm birth. Although estrogen's immune effects are context-dependent, its specific role in placental inflammation remains unclear. We hypothesize that estrogen deficiency lowers the threshold for inflammatory activation, resulting in an exaggerated cytokine response, whereas intact estrogen signaling confers a potential protective role during pregnancy.

To test this hypothesis, we are establishing 3D trophoblast organoids derived from frozen, isolated cytotrophoblasts and from fresh villous tissue collected from third-trimester human placentas. Organoids will be validated for trophoblast identity using immunofluorescence staining for markers: cytokeratin 7, a pan-trophoblast marker; EGFR, which stains villous cytotrophoblasts and the surface of syncytiotrophoblasts; and GATA3, TFAP2A, and TFAP2C, which are trophoblast-enriched transcription factors. Trophoblast function will be evaluated using an ELISA to measure hCG. Once validated, experimental groups will include: (1) control, (2) IL-1β (an endogenous pro-inflammatory cytokine) stimulation to model an inflammatory challenge, and (3) estrogen inhibition plus IL-1β stimulation to assess the impact of suppressed estrogen signaling. Cytokine release into culture media will then be quantified using ELISA, and intracellular cytokine production will be assessed by immunohistochemistry.

Findings from this study will help clarify estrogen's role in modulating placental inflammatory responses and may provide insights into how hormonal disruptions contribute to inflammation-driven pregnancy complications and adverse long-term fetal outcomes.

This research was supported in part by the M4I: Maryland Infection, Immunization, Intervention, and Impact Training Program.

P4.10 Understanding Disparities in Prenatal Diagnosis of Congenital Heart Disease

Presenter: Claire Wegner[1]
Mentor(s): Alicia Chaves, MD[2]
[1]University of Maryland School of Medicine, Baltimore, MD
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Early prenatal diagnosis of congenital heart disease (CHD) remains a significant contributor to infant health outcomes. It has been shown that Latino ethnicity and non-English language preference are associated with lower odds of prenatal diagnosis and later gestational age at diagnosis. There is still much to understand about what community and socioeconomic factors affect the likelihood of timely prenatal diagnosis of CHD. This study aims to identify how the prenatal course differs between patients who received a prenatal diagnosis and those who received a late or postnatal diagnosis. We are conducting a retrospective cohort study of infants who underwent cardiac surgery for CHD within 30 days of birth at the University of Maryland Medical Center between 2011 and 2025 (N=214). Data collection includes timing of prenatal care initiation, location and type of provider administering the anatomy ultrasound scan, geographic distance from tertiary care centers, maternal immigration status, and timing of insurance acquisition. Preliminary results show differences by race with 90% of Asian, 81% of Black, 73% of White, and 58% of Other race mothers receiving a prenatal diagnosis. A prenatal diagnosis was made in 77% of non-Hispanic mothers compared to 55% of Hispanic mothers (p=0.006), and 77% of mothers who prefer English language compared to 55% of mothers who prefer non-English language (p=0.008). Further analysis is ongoing and will explore associations with additional maternal factors to better understand barriers to equitable CHD diagnosis.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

P4.11 Retrospective Chart Review of Infant & Mother Dyads seen in Special Parent and Infant Care and Enrichment (SPICE) Clinic

Presenter: Nikki Emamian[1]
Mentor(s): Matthew Grant, MD[1]
Co-Author(s): Claire Eberhardt, LCSW-C[1]
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The risk of maternal-to-child-transmission (MTCT) of HIV varies based on factors such as timing of maternal diagnosis, access to care, treatment adherence, and sustained viral suppression. At the University of Maryland School of Medicine, pregnant individuals living with HIV are enrolled in case management services through the Special Parent and Infant Enrichment (SPICE) clinic, providing support to enhance adherence, education, advocacy, and emotional support throughout pregnancy and postpartum. We hypothesize that support from the case management program through the SPICE Clinic during pregnancy will impact their viral suppression, and therefore lower risk of MTCT. Secondarily, we anticipate that prenatal case management through the SPICE clinic improves infant appointment adherence as compared to infants born to mothers who are first enrolled in the case management program during the postpartum stage. A retrospective cohort analysis of infant-mother dyads seen at the SPICE Clinic at University of Maryland School of Medicine between January 2015 and December 2024 is currently underway, with an anticipated total enrollment of 350 dyads (total 700 persons). Thus far, encounters have been reviewed between January 2021 to December 2024. Planned next steps include additional data collection, cleaning, and analysis including both descriptive analysis and chi-squared analyses to determine if enrollment during the prenatal period differentially affects maternal viral suppression, and therefore risk stratification in the newborn infant, as well as appointment adherence in the newborn period.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

P4.12 Evaluating Post-operative Pain Management with NSAIDs in Pediatric Orthopedic Patients

Presenter: Ethan Yang, BA
Mentor: Joshua M. Abzug, MD
Other Co-Authors: Jalen Tom, BS; Abel Lindley, BS; Paul Bernhard, BA
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Introduction
Postoperative pain management in pediatric orthopedics is critical, particularly with the ongoing opioid crisis. NSAIDs have emerged as a promising alternative to opioids, though their efficacy as a postoperative analgesic in pediatric populations remains uncertain. The purpose of this study is to assess the frequency of follow-up call-backs and emergency department (ED) visits in pediatric patients prescribed only NSAIDs after orthopedic procedures.

Methods
A retrospective chart review was performed to identify all pediatric patients (≤17 years) treated at the University of Maryland Medical System over a 10-year period. Inclusion criteria include patients who were prescribed only NSAIDs postoperatively. Data collected will include patient demographics, surgical details, provider type, NSAID type, and incidence of postoperative call-backs or ED visits. Simple statistical analysis was performed. Data was de-identified and analyzed securely.

Results
A total of 1,791 pediatric patients met the inclusion criteria. The mean age at surgery was 9.77 years (SD 6.84) with a male predominance (52.3%). Most injuries were fractures, including supracondylar (12.33%), distal radius (11.22%), and phalangeal (13.73%) fractures. Open surgical procedures were performed in a slight majority of cases (50.28%), and intraoperative nerve blocks or local anesthesia were rarely given (1.79%). NSAIDs were prescribed at discharge in 83.5% of cases, most commonly ibuprofen or naproxen. The incidence of postoperative call-backs for pain management within two weeks was low (2.3%), as well as emergency department visits due to uncontrolled pain (1.8%). Postoperative pain-related concerns were managed with reassurance or prescription refills. Overall complication rates were minimal (1.2%). The average follow-up duration was 4.8 weeks (SD 2.1).

Discussion
This study demonstrates that NSAIDs are a well-tolerated and effective postoperative pain management option in pediatric orthopedic patients. The low rates of pain-related callbacks, emergency visits, and complications support the safety and efficacy of NSAID-only treatment regimens. These findings support the existing literature suggesting the use of NSAIDs as a primary modality in pediatric postoperative pain management and the reduction of opioid use.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

P4.13 Investigating Use of IV Lidocaine in Perioperative Pain Control following Colorectal Surgery

Presenter: Rebecca Li
Mentor(s): Megan G. Anders, MD, MS
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This study aimed to refine lidocaine infusion practices to reduce opioid consumption in adult patients following colorectal surgery. Opioid-sparing strategies are critical in colorectal surgery to reduce postoperative complications and enhance recovery. Intravenous lidocaine (IVL) has emerged as a potential adjunct for minimizing postoperative opioid use, but current literature provides conflicting evidence regarding IVL effectiveness. We conducted a retrospective cohort study of patients who underwent elective colorectal surgery at UMMC between March 2020 and May 2025. Patients were grouped by IVL duration: no IVL, <12 hours, and ≥12 hours. Postoperative opioid use was measured as total morphine milligram equivalents (MMEs) administered within the first 24 or 48 hours postoperatively. Linear regression models adjusted for age, preoperative opioid use, and procedure type. Among 868 patients, 428 (49.3%) received no IVL, 214 (24.7%) received IVL <12 hours, and 226 (26.0%) received IVL ≥12 hours. Patients receiving ≥12h IVL had significantly lower mean MMEs over the first 48 hours postoperatively (73 mg) compared to no IVL (88.8 mg; p=0.031). IVL <12h was not significantly associated with a reduction in mean MME use at either 24 or 48 hours postoperatively compared to no IVL. Adjusted analysis showed a 21.9 mg reduction in MME for the ≥12h group compared to no IVL (95% CI: -42.4 to -1.3 mg; p=0.037). Prolonged IVL infusions (≥12 hours) were associated with reduced postoperative opioid use. These findings support incorporating extended IVL into multimodal analgesia protocols for colorectal surgery.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

P4.14 An Observational Study of Physiologic Changes During Prolonged Breath Holds in Bronchoscopic Lung Biopsies

Presenter: Lauren Colliver[1]
Mentor(s): Megan Anders, MD, MS[1]
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Image guided bronchoscopic biopsy is a minimally invasive technique performed under general anesthesia to diagnose suspicious lung nodules. Partnership between anesthesia providers and interventional pulmonologists can maximize procedure success. Ventilation protocols that optimize real-time cone beam CT (CBCT) and reduce atelectasis improve diagnostic accuracy of biopsies. A clinical practice guideline modeled after the Lung Navigation Ventilation Protocol (LNVP) published by Bhadra et al. (2022) was introduced at the University of Maryland Medical Center. The clinical practice guideline and LNVP use high positive end expiratory pressure (PEEP) and prolonged apneic breath holds to reduce atelectasis and minimize lesion movement during biopsy. There has been minimal research conducted on the physiologic parameters during prolonged breath holds. We collected intraprocedural data (heart rate, blood pressure, end tidal carbon dioxide, and oxygen saturation) before, during, and after prolonged breath holds to determine the physiologic effects. We also documented smoking status and intraprocedural vasopressor administration to determine the influence of these factors on the observed physiologic parameters. To date, 12 participants have been enrolled. Preliminary results suggest a positive correlation between the length of the breath hold and the end tidal CO2 immediately after the breath hold. Previous or current smokers appear to have higher end tidal CO2 after breath holds. Blood pressure increases during and after the breath holds, likely due to the administration of vasopressors before and during breath holds. This study aims to improve understanding of physiologic changes during prolonged breath holds to standardize techniques and protocols for bronchoscopic lung biopsy.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

P4.15 Investigating the Relationship Between Cortical Inhibition and Cognitive Control in Schizophrenia Using TMS-Measured SICI and the Stop-Signal Task

Presenter: Eugene Bosworth[1]
Mentor: Stephanie Hare, PhD[1]
Co-Author(s): Robert Buchanan, MD[1], James Gold, PhD[1]
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Schizophrenia (Sz) is a debilitating psychiatric disorder characterized by cognitive impairments that affect working memory, attention, and executive functioning. These cognitive functions depend on a coordinated balance between excitatory/inhibitory (E/I) neural mechanisms. It has been theorized that dysfunction of this E/I balance plays an important causal role in the pathophysiology of Sz, as many studies report increased cortical excitability in people with the disorder. Cortical excitability can be easily studied in the motor pathways using paired-pulse transcranial magnetic stimulation (TMS) to measure short-interval intracortical inhibition (SICI) – an electromyography (EMG) recorded biomarker. Higher SICI scores, which indicate reduced cortical inhibition, have been shown to be associated with a diagnosis of Sz when compared to healthy controls. Even though abnormal SICI provides direct evidence of altered E/I balance in the motor system, there has been a lack of research in the relationship between SICI and cognitive impairments in Sz. This study aims to examine the association between SICI and cognitive inhibitory control, measured by the Stop-Signal Task (SST), in individuals with Sz. To date, we have collected preliminary data from 12 individuals with Sz and three healthy controls. Further participant recruitment is ongoing, with the goal of evaluating whether altered SICI correlates with longer Stop-Signal Reaction Times (SSRT) and poorer cognitive control in the Sz group relative to controls. Findings may inform the utility of SICI as a biomarker for cognitive dysfunction and guide neuromodulatory interventions aimed at restoring E/I balance in Sz.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research and by grant 1K01MH133116-01A1.

P4.16 Evaluating the Role of Conserved miRNAs Across Species in the Optic Nerve Laminar Region (ONLR) that Promote Retinal Ganglion Cell Survival and Regrowth

Presenter: Yehyun (Abby) Kim[1]
Mentor(s): Steven Bernstein, M.D., Ph.D.[1]
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The optic nerve (ON) is comprised primarily of axons of retinal ganglion cell (RGC) neurons that connect the eye and brain. Single cell sequencing (scRNA seq) of the optic nerve head (ONH) revealed 10 cell types. ONH-Neural progenitor cells (ONLR-NPCs) secrete a variety of neuroprotective molecules including growth factors, extracellular vesicle (EV)-associated proteins, and microRNAs (miRNAs) that control inflammation, RGC stress and growth. We identified the miRNAs secreted in extracellular vesicles by primary ONH cells in rats and nonhuman primates using both proteomic and miRNA sequencing. We wanted to identify miRNAs that are supportive of ON and RGC survival, and their functions.

Preliminary analysis of human and rat ONH miRNAs revealed 175 conserved miRNAs that are likely required for normal optic nerve function across species. We focused on the 70 miRNAs within the top 100 highest expression levels from both human and rat data. We used both primary literature and databases (miRBase and miRDB) to evaluate predicted or validated target genes and pathways of each miRNA species. miRNAs were classified into roughly 5 groups: 1. Inflammation suppression, 2. Myelination enhancement or suppression, 3. Suppression of axonal degeneration, 4. Suppression of oxidative stress, and 5. Caspase3 and other apoptosis/ferroptosis suppression. We are currently evaluating the effects of a selection subgroup of these miRNA molecules in vitro, on rat retinal ganglion cells. Our work will provide a first look at the ability of individual NPC components to form a new class of treatment for chronic optic nerve-related diseases.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

P4.17 Cortical interneuron development in Polyhydramnios, Megalencephaly, and Symptomatic Epilepsy

Presenter: Meghna Pandey[1]
Mentor(s): Whitney Parker, MD, PhD[1]
Co-Author(s): Ria Parikh[1,2], Asmaa Hijazi[1], Thach-Vu H. Nguyen[1], Philip H. Iffland II, PhD[2], Peter B. Crino, MD, PhD[2], Whitney E. Parker, MD, PhD[1]
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Polyhydramnios, Megalencephaly, and Symptomatic Epilepsy (PMSE) is a rare genetic neurological disorder that results in severe intractable epilepsy, megalencephaly, craniofacial dysmorphism, and global developmental delay. This disorder is caused by a loss-of-function mutation in the *LYK5/STRADA* gene, which encodes the STRADA protein, a pseudokinase that activates liver kinase B1 (LKB1) toward inhibition of the mechanistic target of rapamycin (mTOR) pathway, in response to low cellular energy levels. The LKB1-mTOR pathway is heavily involved in regulating cytoskeletal dynamics necessary for developmental processes in neurons, including the migration of neural progenitor cells. While STRADA loss has been well characterized in the context of excitatory cortical neuron development, its impact on inhibitory interneurons (INs), which likely contributes to its role in epilepsy, remains largely unexplored. Our study investigates the role of STRADA in cortical IN development and lamination by comparing the expression of mature GABAergic INs, including major subtypes, and inhibitory neural progenitors in both mouse models and human tissue. Cortical INs originate in the ganglionic eminence (GE), which develops into mature subcortical regions (basal ganglia, thalamus). We compared expression across cortical and subcortical regions between wildtype (WT), heterozygous (Het) and STRADA knockout (KO) mouse tissue, and between human PMSE patient tissue (female, 7 months old) and age-matched human control tissue. Findings of this investigation will contribute to the development of targeted therapies for PMSE and related disorders.

This research was supported in part by the Program for Research Initiated by Students and Mentors (PRISM), University of Maryland School of Medicine Office of Student Research.

P4.18 The Impact of Depression on Antiretroviral Therapy Adherence in People Living with HIV: The Moderating Role of PTSD

Presenter: Nicole Fortune Hernandez[1]
Mentor(s): Jessica Magidson[2], Noah Triplett[2], Abigail Hines[2]
[1]MPOWER Program, University of Maryland School of Medicine, Baltimore, MD
[2]CESAR and GMAP, University of Maryland College Park, College Park, MD

Researchers have identified a link between depression and poor medication adherence among individuals living with HIV. A 2014 meta-analysis involving 12,243 individuals found that those treated for depression were 83% more likely to adhere to their medication regimens. Similarly, studies have shown an association between trauma exposure and low medication adherence. In this cross-sectional study, we examined the relationship between depression and antiretroviral therapy (ART) adherence amongst individuals living with HIV and a history of substance use disorder (SUD) in Cape Town, South Africa. The analysis draws on baseline data from the ongoing Khanya project which is a peer-delivered, stepped-care intervention designed to improve ART adherence and reduce substance use in primary care settings. Participant eligibility was determined at baseline using urinalysis, clinical history, and various psychosocial assessments, including the PHQ-9 and PCL-5, which measure depression and PTSD symptomatology, respectively. This analysis includes all 160 participants enrolled in Khanya and investigates whether PTSD symptomatology moderates the relationship between depression and ART adherence. A linear regression model was used to assess this association by comparing individuals with low versus moderate-to-high PTSD symptoms. Results indicated that the relationship of interest was not statistically significant. This may be due to the limited sample and the restricted generalizability of the findings, as participants in the study met criteria for low ART adherence at baseline. Further research is needed to understand how PTSD symptomatology may influence the effect of depression on ART adherence. Insights from additional research could inform the development of integrated interventions.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

P4.19 The Continuum of Care in Hospitalized Patients with Opioid or Stimulant Use Disorder and Infectious Complications of Drug Use – Treatment as Usual, Addiction/ID Integrated Clinic (CHOICE-STAR Study) & Effects on Patient Antibiotic Completion and Outpatient Utilization

Presenter: Isabel Veloso[1]
Mentor(s): Sarah Kattakuzhy MD MPH; Edward Traver, MD[1]
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A serious consequence of the continued rise in injection drug use among Americans is an increase in serious injection-related bacterial and fungal infections (SIRI). Due to the complex nature of treating infections in the setting of comorbid substance use it is imperative to develop comprehensive and effective services to target treatment of patients who use injection drugs and reduce adverse outcomes. The CHOICE-STAR study aims to elucidate how to best reduce infectious disease-related rehospitalization of patients with SIRI by comparing treatment as usual (TAU) to an integrated infectious disease and substance use disorder clinic intervention (IC). The goal of the IC is to increase access to infectious disease and substance use disorder treatment after hospital discharge by providing collocated and coordinated services. Patients are enrolled at the time of hospitalization and randomized to TAU or IC for 6 months, with follow-up assessments on Day 14 and Months 1, 3, 6, 9, and 12 after hospital discharge. The focus of this current sub-analysis is to determine if participation in the IC arm affects rates of antibiotic completion, medication for opioid use disorder (MOUD) uptake, and outpatient provider visits, as analyzed by SAS Fisher exact test. This preliminary analysis will provide insight into the challenges injection drug users face to access care outside the hospital and the benefits of care coordination starting at hospitalization.

This research was supported in part by the M4I: Maryland Infection, Immunization, Intervention, and Impact Training Program, University of Maryland School of Medicine Office of Student Research.

P4.20 Impact of Health Equity Notes on Maryland Department of Health's Policy Positions in the 2025 Maryland General Assembly Session

Presenter: Urvi Chowdhury[1]
Mentor(s): Sarah Case-Herron, JD[1]; Meghan Lynch, MPP[1]
Co-Author(s): Sarah Case-Herron, JD[1]; Meghan Lynch, MPP[1]
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In the 2025 Maryland General Assembly Session, the Maryland Department of Health (MDH) piloted "health equity notes" in its bill review process. Understanding how and when equity notes shape health policy is key to evaluating their effectiveness and future role in state governance.

We analyzed all MDH-submitted health equity notes from the 2025 Session and compared departmental voting patterns across positive, negative, or mixed impact equity notes as well as the strength of equity framing. We hypothesized that both the presence and strength of the health equity notes would increase the likelihood that MDH departments take definitive voting positions rather than defaulting to neutrality.

Our analysis showed that only a small percentage of bills included a health equity note, but most submitted notes were strongly developed. Positive impact health equity notes were most common, yet most MDH departments voted neutrally. Negative impact notes were more strongly associated with opposition, while mixed impact notes led predominantly to neutral positions. Low response rate and widespread neutrality in the presence of equity notes highlights the need for an improved adoption of equity-driven policy analysis at MDH.

Qualitative debriefs will be conducted with legislative liaisons to inform improvements for the 2026 General Assembly Session. A memorandum and accompanying presentation outlining recommendations will be submitted to the Maryland Secretary of Health.

This research was supported in part by the University of Maryland Scholars Program, an initiative of the University of Maryland: MPowering the State.

P4.21 The CATALYST Program: Teacher Researchers Bringing Biomedical and Cancer Research to the Classroom

Presenter: Madeline Large[1,2,3]
Mentor(s): Dr. Saumen Sarkar[2,3,4,5], Dr. Bret Hassel [1,3,4.], Michael Wagner[2,7]
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The immune system functions in the defense from exogenous agents such as viruses as well as from diseases originating from host cells such as cancer. Therefore, the same immune effectors can serve dual functions in antiviral and tumor-suppressor activities. One such effector is OAS1 that is classically known to function through the activation of viral RNA degradation and has recently been shown to influence autophagy. Autophagy is a pathway by which cellular components are recycled that promotes the replication of picornaviruses as well as tumor cell proliferation. We hypothesize that OAS1 modulation of autophagy mediates its antiviral and tumor suppressor functions. To test this hypothesis, we restored OAS1-deficient cancer cells with functional protein or a version that contains a naturally occurring mutation. Ongoing analysis of viral replication, cell proliferation and autophagy in these cell lines will provide insights into the role of OAS1 in host defense from picornaviruses and cancer. This study is conducted by middle school (MS) teachers who gain experience in laboratory research with the goal of developing novel curriculum material that will educate students about biomedical research and inspire their interest in STEM subjects. The broad concepts and technical approaches of this work will be adapted into accessible research experiences for middle school students to explore the fields of microbiology and immunology and their importance to cancer research.

The research reported in this presentation was supported by the National Cancer Institute of the National Institutes of Health under the Catalyzing Cancer Research among Urban Underrepresented Minority Youths and Teachers (CATALYST) Program with Award Number R25CA274166 to Drs. C. Adebamowo, B. Hassel and S. Adebamowo, and the P30 Cancer Center Support Grant under Award Number P30CA134274 to Dr. T. K. Owonikoko. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

P4.22 Tracking the Pulse of Survivorship: HRV as a Public Health Indicator of Physiological Stress

Presenter: Lakesha Johnson[1,2]
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Heart rate variability (HRV) is a non-invasive biomarker reflecting autonomic nervous system function and physiological resilience. In older adults—who comprise approximately 60% of U.S. cancer survivors—many live with cancer as a chronic illness, often experiencing persistent fatigue and autonomic dysregulation. Traditional symptom self-reports may miss these physiological changes, highlighting the need for objective tools like HRV to enhance survivorship care. To assess the relationship between treatment and physiological responsiveness, we recruited older adults living with cancer as a chronic illness. Electrocardiograms (ECGs) were collected while participants lay still for five minutes at both baseline (N = 20) and 12 weeks (N = 9). Kubios software was used to detect R-wave peaks and extract HRV parameters, including SDNN, mean heart rate, and autonomic indices. Patient-reported fatigue (BFI, FACIT-F) and chronic illness burden scores were also collected. Linear regression and correlation analyses were used to examine changes and relationships over time. Although correlations between HRV and age, BMI, or time since diagnosis were not statistically significant, inverse trends were observed—those who were older, heavier, or living longer with cancer tended to show lower HRV. Nearly 40% of participants had critically low SDNN at baseline. Strong and statistically significant inverse correlations were observed between SNS Index and both SDNN ($r = –0.74$, $p < .001$) and PNS Index ($r = –0.83$, $p < .001$), indicating autonomic imbalance under sympathetic stress. An HRV-focused program offers early detection, personalized intervention, and trackable feedback, shifting care from averages to the individual.

This project was funded by the University of Maryland Claude D. Pepper Older Americans Independence Center (UM-OAIC) Pilot program, P30AG028747.

P4.23 Congenital CMV infection among infants born to mothers with and without HIV infection in Malawi.

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Mentor(s): Miriam Laufer, MD[1]
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Over 1.5 million infants are born to women infected with HIV. Even with the distribution of antiretroviral therapy (ART) in HIV-endemic regions, *in utero* exposure to HIV is associated with adverse outcomes even in the absence of infection in the infant. One such risk is congenital cytomegalovirus (cCMV), a common herpes infection affecting infants globally. cCMV is thought to be more common in infants born in sub-Saharan Africa, but little information exists. In addition, infants born to women diagnosed with HIV may be at an increased risk of contracting cCMV. cCMV is associated with abnormalities at birth, and it may also detrimentally impact neurocognitive development. We investigated the relationship between maternal HIV infection and cCMV infection, and its impact on child development by analyzing urine samples collected at birth from infants born to mothers with and without HIV infection in Malawi. We followed the infants for two years to assess neurocognitive development. DNA was extracted from the urine samples, and we detected CMV using GeneProof Cytomegalovirus PCR kit. Out of 1054 samples, we found 34 (3.4%) samples were positive for CMV. Further results and analyses will be provided.

This research was supported by R33HD103066 and R01HD100235, Macalester College, and the Pediatric Infectious Diseases Society.

P4.24 Gene Set Analysis Identifies Immune Dysregulation Signatures of Prostate Cancer Recurrence

Presenter: Zumar Meher[1]
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Prostate cancer is responsible for over 35,000 deaths per year in the United States alone. The treatment options for localized cases include radical prostatectomy or radiation therapy. But, even after treatment, ~70% of patients experience a rise in blood prostate-specific antigen (PSA) levels, a highly sensitive biomarker for potential recurrence. The time until a patient experiences this post-treatment rise in PSA - defined as time to biochemical recurrence (BCR)—is an important marker as it provides insight into both tumor “aggression” and the extent of metastatic microseeding. As such, time to BCR predicts poor prognosis. We hope to use time to biochemical recurrence to investigate transcriptomic signatures pertaining to disease aggressiveness and the process of tumor extravasation. The transcriptomic data of 74 patients in the TCGA Prostate Adenocarcinoma dataset who experienced biochemical recurrence post radical prostatectomy were studied. Patients were stratified into quartiles based on “Days to BCR” and their gene expression profiles were analyzed using Geneset Variation Analysis, K-means clustering, Principal Component Analysis (PCA), and Over-representation analysis (ORA), and overall survival was assessed for patients with and without genomic and/or transcriptomic alterations. K-means clustering identified a cluster of interest based on a linear expression pattern that distinguished early from late recurrence. PCA suggested that epithelial splicing regulatory protein 1 (ESRP1) – a regulator of epithelial-mesenchymal transition – is significantly associated with early recurrence in a subset of patients. ORA of shared gene sets highlighted immune cell signatures related to tumor-associated macrophages (TAMs), dendritic cells (DCs), and natural killer (NK) cells, all of which were linked to immune dysregulation. This dysregulation may compromise immune surveillance and promote increased susceptibility to a tumor microenvironment (TME). Building on this further, we will examine single cell data on independent cohorts of radical prostatectomy specimens to further understand the role of immune dysregulation in prostate cancer.

This research was supported in part by the University of Maryland Greenebaum Comprehensive Cancer Center.

P4.25 The Localization of Granzyme B in Corneal Tissue of Ocular Graft versus Host Disease Murine Models and its Significance on the Pathological Pathway

Presenter: Katie Lowe[1]
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Ocular graft versus host disease (oGVHD) is a complication that occurs in 50% of patients who undergo an allogeneic hematopoietic stem cells transplant (HSCT). Patients often experience severe inflammatory dry eye disease resulting in blurred vision, corneal epithelial cell loss, conjunctival fibrosis, and in the most severe cases corneal perforation. Granzyme B (GZMB), a pro-apoptotic serine protease, enters target cells through perforin-1, a cell-membrane protein, and results in apoptosis of the target cell. GZMB also has perforin-1 independent effects as it degrades the extracellular matrix, resulting in fibrosis and pro-inflammatory cytokine release. To further investigate the role of GZMB in OGVHD development, murine models of oGVHD utilized the following donor cells from mice deficient in (1) GZMB, (2) perforin-1, and (3) both GZMB and perforin-1. The aim of this study is to use immunofluorescent staining to determine if there is a disparity between the localization of GZMB to the intracellular or extracellular space in the cornea in a mouse model of oGVHD and a deficiency in GZMB, perforin or both GZMB and perforin. Corneas from each of these groups were harvested and fixed in 4% PFA for 24 hours. Immunofluorescent staining was performed using identifier stains to distinguish the nuclei and cell membrane as well as primary and secondary antibodies to distinguish GZMB. Immunofluorescent images were taken using confocal microscopy. The results suggest that GZMB may be localized differently between the central and peripheral cornea as well as the intra- and extracellular space of the cell.

This research was supported in part by the Cigarette Restitution Fund (SBS) and the CIBMTR KL2 (SBS).

P4.26 Bringing Research to the Classroom: Targeting EGFR-Mediated Resistance to Treatment in Non-Small Cell Lung Cancer

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Lung cancer is the leading cause of cancer death in the US. Non-Small Cell Lung Cancers (NSCLCs) account for 87% of all lung cancers. The American Cancer Society estimates there will be about 226,60 new cases and 124,730 deaths in 2025. Treatments for NSCLCs include tyrosine kinase inhibitors, T-cell engagers, and cellular therapies. However, NSCLC frequently recurs after chemo and radiation, and re-irradiation often leads to acquired radioresistance. Promoting awareness, prevention, and screening through cancer-focused curricula and outreach is essential.

In this project, the teacher collaborated with researchers to develop and implement an experimental plan targeting two proteins involved in tumor growth and resistance to treatment—Epidermal Growth Factor Receptor (EGFR) and caveolin-1 (CAV1)—in three NSCLC cell lines: A549 parental, A549 radioresistant (RR), and HCC-827. After treatment with 4 Gy radiation, clonogenic survival was 55% for A549 RR, 35% for A549 parental, and 20% for HCC-827, indicating greater radiosensitivity in HCC-827 cells. Western blot analysis showed elevated phosphorylated EGFR (pTyr1068) expression only in HCC-827 cells, which may underlie this sensitivity.

To study drug resistance, CAV1 was inhibited using Methyl-Beta-Cyclodextrin (MBCD) and EGFR with Osimertinib. Combined treatment yielded 50% cell viability in HCC-827 cells versus 66% and 83% in A549 wild-type and RR cells, suggesting a synergistic role of dysregulated CAV1 and mutant EGFR in resistance.

Concepts and research techniques will be developed into classroom lessons promoting awareness, prevention, and evaluation of treatments. Hands-on activities (e.g. paper chromatography) will illustrate principles from teacher research techniques (e.g. Western Blot) and use computer-based modeling to explore the role of the cell membrane in cancer treatment.