

THE ARSENAL

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About *The Arsenal*

The Arsenal: The Undergraduate Research Journal of Augusta University (ISSN 2380-5064) is a peer-reviewed, open-access, interdisciplinary journal for undergraduate research conducted at Augusta University. This journal is a collaboration between the Center for Undergraduate Research and Scholarship (CURS), University Libraries, and the CURS student Ambassadors.

The Arsenal was launched by the undergraduate research student organization named On the Shoulder of Giants in Fall 2016. The journal represents and highlights undergraduate research of academic and scholarly value from various disciplines at Augusta University. Each article undergoes a peer-review process facilitated by the journal's Editorial Review Board and must be approved by an appointed faculty reviewer in the article's respective discipline. More information can be found at augusta.edu/arsenal-home.php.

Editorial Board

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Preface

The Center for Undergraduate Research and Scholarship (CURS) proudly presents the proceedings for the 26th annual Undergraduate Research and Fine Arts (URFA) Conference on April 17, 2026. This annual conference is supported by the CURS, the AU Chapter of Phi Kappa Phi, and the Office of Interdisciplinary Research. The proceedings consist of the program for the 26th annual conference, along with the scholarly abstract of each undergraduate presenter's original work.

This year we host 107 undergraduate student presenters majoring in Art, Biochemistry, Biology, Business Administration, Economics, Cell and Molecular Biology, Chemistry, Communications, Computer Science, Cybersecurity, Cybersecurity Engineering, Dental Hygiene, Ecology, English, Health, Society & Policy, Kinesiology, Mathematics, Neuroscience, Physics, and Psychology.

We are pleased to have 64 Faculty Mentors from the departments of Art and Design, Biochemistry and Molecular Biology, Biological Sciences, Biomedical Research, Business, Cardiology, Cellular Biology and Anatomy, Chemistry & Biochemistry, Communications, Community and Behavioral Health Sciences, English and World Languages, Gastroenterology, History, Anthropology & Philosophy, Immunology Center for Georgia, Mathematics, Medicine, Neurology, Neuroscience and Regenerative Medicine, Neurosurgery, Nursing, Oral Biology and Diagnostics, Physics & Biophysics, Physiology, and Psychological Sciences are represented at this year's conference.

We would like to express our gratitude to all the speakers, presenters, participants, and volunteers for their contributions. In particular, we would like to thank our generous sponsors for their financial support to the 26th URFA Conference. Without the support of the Provost's Office, the Vice Provost for Instruction, and the Phi Kappa Phi Honor Society, we would not be able to provide such an impactful event for our students. We hope that the proceedings and conference grant the most beneficial and fruitful experience to all those involved.

Dr. Quentin Davis, Co-Chair URFA Conference

Mrs. Elizabeth Eisner-Kelly, Co-Chair URFA Conference

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President's Welcome



It is an honor to welcome you all to Augusta University's 26th annual Undergraduate Research and Fine Arts Conference (URFA). This unique opportunity is a special chance to showcase the discoveries, innovations, and achievements of the many talented scholars pursuing their degree at AU.

The URFA conference is hosted by the Center for Undergraduate Research and Scholarship (CURS) with support from the AU Chapter of Phi Kappa Phi and the Office of Interdisciplinary Research. CURS, established in 2008, has a mission of supporting undergraduates in the pursuit of discovering new information, investigating factors of influence, and innovating research under the collaborative guidance of a faculty mentor. Since 1897, Phi Kappa Phi – the nation's oldest, largest, and most selective all-discipline honor society – has displayed an integrated approach to both academic and personal excellence.

I believe the most impactful research stems from collaboration, which is why I'm especially proud that this conference highlights partnerships that bridge all our different colleges and campuses, fostering connections and integration across research groups.

At Augusta University, our goal is to achieve excellence by staying committed to the most effective teaching and interactive, engaged learning methods. Providing access to research experiences for our undergraduate students helps us accomplish many important goals – teaching our students to become critical thinkers and problem solvers, while also enhancing the experience they have while they are with us at AU.

Through scholarly engagement, we can truly bring innovation and education to life, which has a lasting impact on students' outcomes and success.

I encourage you to engage and participate in the comprehensive and dynamic presentations developed by our students. I am deeply appreciative of the dedication and commitment of the volunteers, staff, students, and faculty whose hard work made this conference possible.

Congratulations to you all,

Russell T. Keen

President of Augusta University

Conference Agenda

Opening Ceremony	12:15 pm -12:45 pm
<i>Welcome</i>	Dr. Quentin Davis, Center for Undergraduate Research and Scholarship
<i>Opening remarks</i>	Dr. Zach Kelehear, Vice Provost for Instruction
<i>Navigating the Conference</i>	Mrs. Elizabeth Eisner-Kelly, Center for Undergraduate Research and Scholarship
Oral & Poster Sessions	1:00 pm – 5:15 pm
<i>Oral and Poster sessions</i>	See summary schedule
<i>4x4 Showdown</i>	Research Pitch Competition
Awards Ceremony & Reception	6:00 pm – 7:30 pm
<i>Conference Awards</i>	Center for Undergraduate Research and Scholarship, Phi Kappa Phi
<i>Distinctions in Research</i>	Center for Undergraduate Research and Scholarship
<i>Closing Remarks</i>	Dr. Maggy Tomova, Provost
<i>Acknowledgments</i>	Dr. Quentin Davis, Center for Undergraduate Research and Scholarship

Summary Schedule of Events

Welcome & Kickoff

12:15 – 12:45

Poster Sessions

Session A 1:00 – 1:50

Session B 2:00 – 2:50

Cellular Biology

Session 1:00 – 2:00

Neuroscience

Session 1:00 – 2:00

Public Health

Session 1:00-2:00

Neuroscience #2

Session 2:15 – 3:30

Public Health #2

Session 2:15 – 3:30

Social Sciences & Humanities

Session 2:15-3:30

Chemistry

Session 3:00 – 4:00

Medicinal Chemistry

Session 3:45 – 5:15

Physics

Session 3:45 – 5:00

Ecology

Session 3:45 – 5:15

4x4 Showdown Research Pitch Competition

Session 4:15 – 5:00

Awards Ceremony & Reception

6:00 – 7:30

Navigation

TIME	BUTLER (40) Rm 227	COFFEEHOUSE (100) Rm 235	HARDY (50) Rm 232	BALLROOM (300) Rm 155	
11:00 - 12:00				Presenter Check-in; Poster A Set up	
2:15 - 12:45				Welcome & Conference Kickoff	
12:45-1:00	Break	Break	Break	Break	
1:00 - 2:00	Neuroscience 1:00 - 2:00	Cellular Biology 1:00 - 2:00	Public Health 1:00 - 2:00	Poster Session A 1:00-1:50	
				Poster B Set Up	
2:00 - 2:15	Break	Break	Break	Poster Session B 2:00 -2:50	
2:15 – 3:30	Public Health #2 2:15-3:30	Neuroscience #2 2:15 - 3:30	Social Sciences & Humanities 2:15 - 3:30		Break (10 min)
					Chemistry 3:00-4:00
	Break	Break	Break	Break	
3:45 - 5:15	Physics 3:45-5:15	Medicinal Chemistry 3:45-5:15	Ecology 3:45-5:15	4x4 Showdown 4:15 - 5:00	
6:00 - 7:30				Closing Ceremony Awards & Reception	

1:00 – 2:00

Butler, Room 227

Neuroscience #1

Mentor: Dr. Stephen Tymanskyj

Moderator: Hope Amm

Fatimata Deyde, Neuroscience

1:00 – 1:15

CELLULAR MECHANISMS GOVERNING AXONAL TRANSPORT OF SYNAPTIC CARGO

Cassidy Martin, Neuroscience

1:15 – 1:30

INVESTIGATING THE SUBCELLULAR LOCALIZATION OF THE AXON SURVIVAL PROTEIN NMNAT-2

Elizabeth Laura, Biology

1:30 – 1:45

DETERMINING THE REGULATORY PROPERTIES OF MAP1B IN AXONAL MORPHOLOGY

Estrella Cano, Neuroscience & Psychology

1:45 – 2:00

DETERMINING THE ROLE OF NEURONAL NAVIGATORS IN AXONAL GUIDANCE AND NEURONAL CONNECTIVITY

1:00 – 2:00

Coffeehouse, Room 235

Cellular Biology

Moderator: Cammy Riemann

Diana Mejia, Biology

1:00 – 1:15

ALTERED GENE EXPRESSION IN SENESCENT PERIODONTAL LIGAMENT CELLS

Mentor: Dr. Mizuho Kittaka

Jeffrey Lin, Cell and Molecular Biology

1:15 – 1:30

THE STUDY OF CHROMOSOME VERSUS HORMONE EFFECTS IN SALT-SENSITIVE HYPERTENSION

Mentor: Dr. David L. Mattson

Gowri Vattenad, Cell and Molecular Biology

1:30 – 1:45

THE EFFECTS OF DIFFERENT STATHMIN ISOFORMS ON MICROTUBULE DYNAMICS

Mentor: Dr. Stephen Tymanskyj

Megan Rowland, Biology

1:45 – 2:00

COMPARATIVE ANALYSIS OF NORMAL AND DISEASED HUMAN SKIN

Mentor: Dr. Soma Mukhopadhyay

1:00 – 2:00

Hardy, Room 232

Public Health #1

Moderator: Tim Sadenwasser

Neah Miles, Anthropology

1:00 - 1:15

WOMEN'S HEALTH: THE LACK OF TRUST & INFORMATION

Mentor: Dr. Angela Bratton

Christy Eapen, Cell and Molecular Biology

1:15 - 1:30

HOW RACE AND COMORBID FACTORS INFLUENCED ATRIAL FIBRILLATION ABLATION OUTCOMES

Mentor: Dr. Hongyan Xu

Zahra Farooqi, Cell and Molecular Biology

1:30 – 1:45

HEALTHCARE PROVIDER PERSPECTIVES ON INTEGRATING BREASTFEEDING EDUCATION INTO SAFECARE

Mentor: Dr. Ashwini Tiwari Pandey

Alexis Mazique, Biology

1:45 - 2:00

PATIENTS' AND PHYSICIANS' PERSPECTIVES ON POLYCYSTIC OVARY SYNDROME TREATMENT

Mentor: Dr. Ellen LeMosy

2:15 – 3:30

Butler, Room 227

Public Health #2

Moderator: Thomas Weeks

Christy Eapen, Cell and Molecular Biology

2:15 – 2:30

CURRENT ATRIAL FIBRILLATION ABLATION DEMOGRAPHICS ARE NOT NATIONALLY REPRESENTATIVE

Mentor: Dr. Hongyan Xu

Dhilan Dahya, Cell and Molecular Biology

2:30 – 2:45

SYNTHESIZING FEDERAL STRUCTURAL VARIABLES AND NOBEL LAUREATE EXPERIENTIAL NARRATIVES INTO MODEL RESEARCH TRAINING PROGRAMS

Mentor: Dr. Andrew Balas

Rachel Mahend, Health Services

2:45 – 3:00

EXPLORING THE EFFICACY OF THE DENTAL THERAPY MODEL ON DENTAL DESERTS

Mentor: Dr. Lee Maynard

Sarah Buckman, Health Services

3:00 – 3:15

ORAL CANCER HEALTH AWARENESS AT AUGUSTA UNIVERSITY

Mentor: Dr. Steven Coughlin

Tumi Adaramola, Kinesiology

3:15 – 3:30

ANNUAL HEALTHCARE PARTICIPATION AND RURALITY ON COLORECTAL CANCER SCREENING UTILIZATION

Mentor: Dr. Meng-Han Tsai

2:15 - 3:30

Coffeehouse, Room 235

Neuroscience #2

Moderator: Jake Telkamp

Isaac Bloom, Neuroscience

2:15 – 2:30

JMJD1C DIRECTS OLIGODENDROCYTE MATURATION FOLLOWING DEVELOPMENTAL BRAIN INJURY

Mentor: Dr. Evan Goldstein

Jasmine Baidoo, Cell and Molecular Biology

2:30 – 2:45

OLIGODENDROCYTE HISTONE METHYLATION IN PRETERM INFANT WHITE MATTER

Mentor: Dr. Evan Goldstein

Oviya Sinthanai-Selvan, Cell and Molecular Biology

2:45 – 3:00

OLIGODENDROCYTE HISTONE TRIMETHYLATION IN PRETERM INFANT WHITE MATTER

Mentor: Dr. Evan Goldstein

Angelina Martinez, Neuroscience & Psychology

3:00 – 3:15

SOUNDS ALIGN: PHONOLOGICAL PLACEMENT EFFECTS ON LEXICAL AND SUB-LEXICAL PROCESSING

Mentor: Dr. Sara Guediche

Sireen Mohammed, Cell and Molecular Biology

3:15 – 3:30

CROSSMODAL SEMANTIC EFFECTS ON VISUAL WORD RECOGNITION

Mentor: Dr. Sara Guediche

2:15 – 3:45

Hardy, Room 232

Social Sciences & Humanities

Moderator: Erin Prentiss

Nevaeh Fowler, Neuroscience

2:15 – 2:30

“I’VE GOT OUT AT LAST”: “THE YELLOW WALLPAPER”, AIDS, AND JOYFUL REBELLION

Mentor: Dr. Amelia Hall

Maivy Huynh, Health, Society, & Policy

2:30 – 2:45

CYBERPUNK 2077: FOOD ASSOCIATION WITH SOCIOECONOMIC STATUSES

Mentor: Dr. Angela Bratton

Quintus G. Williams, Communications

2:45 – 3:00

NAVIGATING ADMINISTRATIVE SYSTEMS AND THE BLACK STUDENT EXPERIENCE

Mentor: Mrs. Bethany Welsh

Lilly Williamson, Communications

3:00 – 3:15

THE ROLE OF TIKTOK IN THE PALISADES FIRE CRISIS RESPONSE

Mentor: Dr. Carrie Reif-Stice

Raegan Johnson, Psychology

3:15 – 3:30

THE IMPACT OF SOCIAL MEDIA INFLUENCERS ON STUDENTS’ PERCEPTIONS OF SUCCESS

Mentor: Dr. Elizabeth Culatta

Sarabeth Campbell, Anthropology

3:30 – 3:45

INFLUENCE OF GREEK MYTHOLOGY AND THE HERO’S JOURNEY IN DISNEY’S HERCULES

Mentor: Dr. Jennifer Trunzo

3:00 – 4:00

Ballroom, Room 155

Chemistry

Moderator: Lee Parker

J.T. Wicker III, Cell and Molecular Biology

3:00 – 3:15

SUGAR PROTECTION OF MYOGLOBIN SECONDARY STRUCTURE FOLLOWING
PROTEIN DENATURATION

Mentor: Dr. Xiaobing Chen

Khadijah Ladoo, Biology

3:15 – 3:30

ADVANCING CANCER THERAPY: SYNTHESIS OF NRF2 INHIBITORS

Mentor: Dr. Matteo Borgini

Atoryia Adams, Cell and Molecular Biology

3:30 – 3:45

NRF2 INHIBITION TO IMPROVE CANCER PATIENTS' THERAPEUTIC OUTCOMES

Mentor: Dr. Matteo Borgini

3:45 – 5:15

Coffeehouse, Room 235

Medicinal Chemistry

Mentor: Dr. Siva Panda

Moderator: Redouane Aherrahrou

Hayden Yi, Cell and Molecular Biology

3:45 – 4:00

NATURE TO NOVEL: URSOLIC ACID SYNTHETIC HYBRIDS FOR CANCER TREATMENT

Sudhan Sivakumar, Cell and Molecular Biology

4:00 – 4:15

DESIGN AND BIOLOGICAL EVALUATION OF CURCUMIN-DERIVED HYBRIDS WITH MULTI-TARGET ACTIVITY

Anshuman Khadanga, Cell and Molecular Biology

4:15 – 4:30

ENGINEERING MULTIFUNCTIONAL HYBRID SCAFFOLDS FOR PAIN AND INFLAMMATION MANAGEMENT

FaithAnn Ferguson, Biochemistry

4:30 – 4:45

MOLECULAR HYBRIDIZATION APPROACH YIELDS PROMISING CANDIDATES FOR MELANOMA TREATMENT

Maxwell Sobel, Medicinal Chemistry

4:45 – 5:00

A GREEN ONE-POT STRATEGY FOR CONSTRUCTING BIS-BENZOTHAZOLES VIA BENZOTRIAZOLE ACTIVATION

Holden Dinkins, Medicinal Chemistry

5:00 – 5:15

BIOTINYLATED DIARYLPIPERIDINONE HYBRIDS: DUAL FUNCTION PROBES FOR TARGETED CANCER THERAPY

3:45 – 5:15

Hardy, Room 232

Ecology

Moderator: Annabella Natalini

Autumn Larson, Ecology

3:45 – 4:00

MAN-MADE CUT CLOSURE RESTORATION STUDY IN THE SATILLA RIVER ESTUARY

Mentor: Dr. Stacy Bennetts

Erin Przywara, Cell and Molecular Biology

4:00 – 4:15

TEMPORAL PREVALENCE OF KDR IN AEDES ALBOPICTUS, SOUTH RICHMOND COUNTY

Mentor: Dr. Jennifer Baltzegar

J.T. Wicker III, Cell and Molecular Biology

4:15 – 4:30

ASSESSING KNOCKDOWN RESISTANCE IN NORTHERN CSRA AEDES ALBOPICTUS MOSQUITOES

Mentor: Dr. Jennifer Baltzegar

Jake Vos, Ecology

4:30 – 4:45

EFFECTS OF POLLUTANT PRESENCE ON SOUTHEASTERN FISH COMMUNITIES

Mentor: Dr. Randal Singer

Kierra Dennison, Biology

4:45 – 5:00

INVESTIGATING GLUCOSE AVERSION IN THE GERMAN COCKROACH

Mentor: Dr. Jennifer Baltzegar

Megan Miller, Biology

5:00 – 5:15

AQUATIC MACROINVERTEBRATES AND THEIR CORRELATION WITH WATER QUALITY

Mentor: Dr. Robert Cromer

3:45 – 5:00

Butler, Room 227

Physics

Moderator: Vy Nguyen

Jagraj Parmar, Cell and Molecular Biology

3:45 – 4:00

DEVELOPMENT OF AN AUTOMATED MANUAL DEXTERITY ASSESSMENT INSTRUMENT

Mentor: Dr. Joseph Hauger

Karen Rudisill, Physics

4:00 – 4:15

SMARTGRASS: IMPLEMENTATION OF AUTOMATED SENSORS FOR CONTINUOUS GRASS MONITORING

Mentor: Dr. Genevieve Reeves

Richard Kremetz, Physics

4:15 – 4:30

A LOW-COST CALIBRATION APPARATUS TO ENSURE RAIN TIPPER ACCURACY

Mentor: Dr. Joseph Hauger

Alexis Jenkins, Mathematics

4:30 – 4:45

COLLECTIVE OSCILLATIONS OF ULTRACOLD FERMI GASES ACROSS A FESHBACH RESONANCE

Mentor: Dr. Theja DeSilva

Brooke Vos, Physics

4:45 – 5:00

FRACTIONAL QUANTUM HALL PHYSICS IN RAPIDLY ROTATING FERMIONS AND EXCITONS

Mentor: Dr. Theja DeSilva

4:00 – 5:00

Ballroom, Room 155

4x4 Showdown

Moderator: Aspasia Luster

Autumn Richards, Psychology

STRESS, RESILIENCE, DISTRESS TOLERANCE AND COLLEGE ADJUSTMENT

Mentor: Dr. Sabina Widner

Lillian Witherington, Cell and Molecular Biology

GREATER NETRIN-1 IN MALE ADPKD MICE ENHANCES CYSTOGENESIS THAN FEMALES

Mentor: Dr. Riyaz Mohamed

Amy Jacob, Cell and Molecular Biology

HEALTHSPAN EFFECTS OF DISRUPTING CIRCADIAN RHYTHMS ON *DROSOPHILA MELANOGASTER*

Mentor: Dr. Jessica Hoffman

Alana Dorgan, Pre-Nursing

IMPACT A STUDY OF MEDICINE WITHIN 18TH-CENTURY RURAL NORTHEASTERN AMERICA

Mentor: Dr. Wendy Turner



1:00 – 1:50

Ballroom, Room 155

Poster Session A

- 1. Navya Pampatwar, Cell and Molecular Biology**
MECHANOSENSITIVE PROTEOLYTIC CLEAVAGE REQUIREMENTS IN NOTCH SIGNAL TRANSDUCTION
Mentor: Dr. Paul Langridge
- 2. Amani Mouna, Cell and Molecular Biology**
PREPARATION OF NUTRACEUTICAL NANOPARTICLES USING FACILITATED SELF-ASSEMBLING TECHNOLOGY (FAST) FOR NEXT-GENERATION NUTRITIONAL SUPPLEMENTS
Mentor: Dr. Stephen Hsu
- 3. Lydia Utomwen, Kinesiology**
THE EFFECT OF TURMERIC DERIVATIVES ON PAIN-PRESSURE THRESHOLD
Mentor: Dr. Dawn Langley-Brady
- 4. Calvin Tran, Neuroscience**
THE ROLE OF MAP7D1 IN NEURON DEVELOPMENT AND INTRACELLULAR TRANSPORT
Mentor: Dr. Stephen Tymanskyj
- 5. Kelli Mitchell, Neuroscience**
TARGETING THE BTK-IDO AXIS ENHANCES ONCOLYTIC HSV IN GLIOBLASTOMA
Mentor: Dr. Bangxing Hong
- 6. Radha Garikipati, Cell and Molecular Biology**
PRECISION OF DUAL-TASK MOBILITY MEASURES TO DIFFERENTIATE FALLERS FROM NON-FALLERS
Mentor: Dr. Deborah Jehu
- 7. Leslie Turnbull, Chemistry**
THE QUANTITATIVE ANALYSIS OF MRI IN EARLY ALZHEIMER'S DISEASE DETECTION
Mentor: Dr. Christina Wilson

8. Taylor Grace Yancey, Nursing

A PROCESS FOR TAILORING SCIENTIFIC ABSTRACTS FOR PATIENT AUDIENCES
Mentor: Ms. Lauren Cafferty

9. Saja Sanadiki, Nursing

PREVENTING ADVERSE MATERNAL AND NEONATAL OUTCOMES IN
ADOLESCENT MOTHERS
Mentor: Dr. Desiree Bertrand

10. Konstantin Kurzin, Biology

HEPARIN EFFECTS IN ACUTE KIDNEY INJURY (AKI)
Mentor: Dr. Paul O'Connor

11. Aeris Xiong, Neuroscience

OLIGODENDROCYTE H3K9ME3 DYNAMICS IN MICE HOUSED IN AN ENRICHED
ENVIRONMENT
Mentor: Dr. Evan Goldstein

12. Aman Kalsi, Cell and Molecular Biology

DEVELOPMENT OF A NON-SYSTEMIC PDE4 INHIBITOR FOR IBD
Mentor: Dr. Darren Browning

13. Luke Horne, Cell and Molecular Biology

DEFINING THE INTERACTOME OF MICROTUBULE ASSOCIATED PROTEINS
Mentor: Dr. Stephen Tymanskyj

14. Julie Bishara, Cell and Molecular Biology

THE ROLE OF PTK7 RELATED TO PROSTATE CANCER
Mentor: Dr. Sharanjot Saini

15. Simran Patki, Cell and Molecular Biology

OLIGODENDROCYTE HISTONE ACETYLATION IN PRETERM WHITE MATTER
Mentor: Dr. Evan Goldstein

16. Lotem Kol, Cell and Molecular Biology & Psychology

VALIDATING SOX2-CREERT2 FOR CONDITIONAL GATA3 DELETION IN
DEVELOPING INNER EARS
Mentor: Dr. Lin Gan

17. Sriya Rajanala, Cell and Molecular Biology

COMPARATIVE ANALYSIS OF AMYLOID-B PATHOLOGY IN 5XFAD MICE USING ANTIBODIES

Mentor: Dr. Yun Lei

18. Jenny Patel, Neuroscience

ROLE OF LRRK2 IN REGULATING AXONAL GROWTH AND TRANSPORT

Mentor: Dr. Stephen Tymanskyj

19. Tina Tutuwan, Neuroscience

ANALYZING THE EFFECT OF MAP7 D3 ON NEURONAL MORPHOLOGY

Mentor: Dr. Stephen Tymanskyj

20. Laurie Conde-Rodriguez, Neuroscience

CELL-TYPE SPECIFIC LOCALIZATION OF MAP7 IN MOUSE NERVOUS TISSUE

Mentor: Dr. Stephen Tymanskyj

21. Saniya Patel, Neuroscience

PREPARATION OF STABLE TAU OLIGOMERS

Mentor: Dr. Stephen Tymanskyj

22. Roberto Barrera Jaimes, Business Administration

EXCHANGE RATE FLUCTUATIONS AND INTERNATIONAL STUDENT ENROLLMENT IN THE UNITED STATES

Mentor: Dr. Simon Medcalfe

23. Sahil Patel & Sasha Hammarlund, Cell and Molecular Biology

SEASONAL SHIFTS IN WILDLIFE ACTIVITY WITHIN SHALLOW RIPARIAN CORRIDORS

Mentor: Dr. Robert Cromer

24. Mason Leavins & Nelson Lau, Chemistry & Physics

DopTC: A LOW-COST WATER QUALITY MONITORING SYSTEM

Mentor: Dr. Joseph Hauger

25. Taylor Hardeman, Neuroscience

LONG TERM INFLAMMATORY RESPONSES TO TASTE BUD DEGENERATION

Mentor: Dr. Lynnette McCluskey

26. Adam Chadli, Accounting

CHARACTERIZATION OF A NEW MURINE GILOMA MODEL FOR ONCOLYTIC
VIROTHERAPY

Mentor: Dr. Balveen Kaur

2:00 – 2:50

Ballroom, Room 155

Poster Session B

1. Kripa Patel, Cell and Molecular Biology

ALTERNATIVELY SPLICED VEGF-A ISOFORMS DIFFERENTIALLY REGULATE ISCHEMIC ENDOTHELIAL GENETIC PROGRAMS

Mentor: Dr. Vijay Ganta

2. Avneesh Prabakar, Cell and Molecular Biology

TAMOXIFEN ATTENUATES RETINAL NEUROVASCULAR INJURY IN DIABETIC RETINOPATHY

Mentor: Dr. Modesto Rojas

3. Rinu Sabu, Biology

ROLE OF POLYAMINES IN RETINOPATHY OF PREMATURITY COMPLICATIONS

Mentor: Dr. Syed Adeel Zaidi

4. Prabhav Gundeboina, Cell and Molecular Biology

NETRIN-1 IS UPREGULATED IN AUTOSOMAL RECESSIVE POLYCYSTIC KIDNEY DISEASE

Mentor: Dr. Riyaz Mohammed

5. Alexis Jenkins, Mathematics

PERFORMANCE OF CLASSICAL AND MACHINE LEARNING SURVIVAL MODELS IN GBSG2

Mentor: Dr. Durga Kutal

6. Emily Hammons, Chemistry

THERMODYNAMICS OF REACTION ENTHALPIES FOR LIPID OXIDATION OF MONOGALACTOSYLDIACYLGLYCEROL

Mentor: Dr. Mandy Green

7. Tiffany Amoako Twum, Psychology

PARENTS PERSPECTIVES ON AFTERSCHOOL RX: A RAPID QUALITATIVE STUDY

Mentor: Dr. Lauren Von Klingraeff

8. Tanika Chevalier, Psychology

THE IMPACT OF SHIFT SCHEDULES ON NURSES' SLEEP AND HEALTH

Mentor: Dr. Lauren Von Klingraeff

9. Gavin Gnagey, Biology

STUDIES ON EXOSOMES AS TREATMENT OF MESENCHYMAL STEM CELLS

Mentor: Dr. Ali Eroglu

10. Kayla Wilder, Cell and Molecular Biology

INVASION POTENTIAL AND GENE EXPRESSION CHANGES IN NUCLEAR FACTOR-KAPPA B ALTERED MACROPHAGES IN TRIPLE-NEGATIVE BREAST CANCER

Mentor: Dr. Jennifer Bradford

11. Bansari Patel, Biology

EXPLORING THE GLUTAMATERGIC INTERACTION BETWEEN ASTROCYTES AND NEURONS: IMPLICATION FOR ALCOHOL USE DISORDER PATHOPHYSIOLOGY

Mentor: Dr. Seungwoo Kang

12. Gavin Feinberg, Cell and Molecular Biology

A BIOLUMINESCENCE ASSAY STUDYING SEQUENCE DETERMINANTS OF DHHC 5-PALMITOYLTRANSFERASE SPECIFICITY

Mentor: Dr. Nevin Lambert

13. Alaira Beecher, Biology

B-ARRESTINS AS PREDICTORS OF OVERALL SURVIVAL IN CANCER

Mentor: Dr. Georgios Kallifatidis

14. Zackary Kimel, Cell and Molecular Biology

DEVELOPING A CLINICAL-GRADE CRYOPRESERVATION METHOD FOR NATURAL KILLER CELLS

Mentor: Dr. Ali Eroglu

15. Ella Shipp, Biology

THE IMPACT OF BLOOD PRESSURE VARIABILITY ON MICROGLIA REACTIVITY

Mentor: Dr. Jessica Filosa

16. Myles Davis, Cybersecurity Engineering

COMPUTATIONAL PERFORMANCE OF JULIA-BASED DMRG ALGORITHM

Mentor: Dr. Trinanjan Datta

17. Guluna Qazi, Cell and Molecular Biology

ANALYTICAL GEOMETRIC APPROACH TO MINKOWSKI SPACE TIME DIAGRAM

Mentor: Dr. Asanka Amarasinghe

18. Adam Porter, Kinesiology

THE IMPACT OF VIRTUAL REALITY ON AFFECT-REGULATED EXERCISE

Mentor: Dr. Andrew Moore

19. Rakshna Anithadevi Sivakumar, Cell and Molecular Biology

CORRELATES OF INTRAINDIVIDUAL SLEEP VARIABILITY IN YOUNG ADULTS

Mentor: Dr. Lauren Von Klinggraeff

20. Anthony Jones, Cell and Molecular Biology

SPATIAL PREVALENCE OF PYRETHROID RESISTANCE IN AN *AEDES ALBOPICTUS* POPULATIONS

Mentor: Dr. Jennifer Baltzegar

21. Temmie Siebert, Biology

ECOD ASSAY METHOD DEVELOPMENT IN A BLACKWORM MODEL

Mentor: Dr. Faith Wiley

22. Liam Hashimoto, Psychology

EVIDENCE-BASED PARAMETERS OF A HEALTHY LIFESTYLE

Mentor: Dr. Andrew Balas

23. Briana Graham, Cell and Molecular Biology

TRANSFORMATIVE RESEARCH DISCOVERIES WITH THE GREATEST MEASURABLE PUBLIC HEALTH IMPACT

Mentor: Dr. Andrew Balas

24. Harsh Patel, Cell and Molecular Biology

CRE-LOXP SYSTEM FOR GENETIC MANIPULATION IN RETINAL NEURONS

Mentor: Dr. Lin Gan

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Mentor: Dr. Matteo Borgini

26. Kennedy Gross, Biology

THE EFFECTS OF HABITAT FRAGMENTATION ON WILDLIFE MOVEMENTS

Mentor: Dr. Robert Cromer

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Abstracts

Nrf2 Inhibition to Improve Cancer Patients' Therapeutic Outcomes

Presenter(s): Atoryia Adams and Maria Biondolillo

Author(s): Atoryia Adams, Khadijah Ladoo, Emil Ayala-Cosme

Faculty Sponsor(s): Matteo Borgini, PhD and Lindsey Davis, PhD

Affiliation(s): Department of Chemistry and Biochemistry, Augusta University

ABSTRACT

Cancer is a disease characterized by the abnormal growth of cells in the body to form tumors. Currently, there is not a definitive cure for this disease, but cancer therapeutics, along with radiation therapy, has been proven to slow and even treat cancer by killing or damaging the affected cells. However, the cells in some cancer patients are remarkably resistant to these therapies. It has been found that this resistance is due to a protein called nuclear erythroid related factor-2, Nrf2. Nrf2 is a protein involved in the KEAP1-NRF2 pathway, which is a pathway that produces antioxidant molecules. These molecules help cancerous cells combat the oxidative stress used by therapies to kill cancer cells. In cancerous cells, this pathway is overactivated which causes an accumulation of Nrf2. This information proves Nrf2 to be a hopeful target in treating cancer. The goal of our research is to develop peptides that will block the nuclear translocation of Nrf2 and inhibit its effects. Nrf2 can only produce its effects when it is translocated into the nucleus where it interacts with DNA, promoting the expression of cytoprotective and antioxidants genes. We aim to identify a peptide that blocks the complex formation between Nrf2 and a shuttle protein responsible for Nrf2 nuclear translocation. This inhibition is expected to increase cancerous cells sensitivity to radiation and pharmacological therapies and in turn lead to better treatment for cancer patients.

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Annual Healthcare Participation and Rurality on Colorectal Cancer Screening Utilization

Presenter(s): Tumi Adaramola

Author(s): Tumi Adaramola and Meng-Han Tsai

Faculty Sponsor(s): Dr. Meng-Han Tsai

Affiliation(s): Department of Chemistry and Biochemistry, Augusta University

ABSTRACT

Colorectal cancer (CRC) is the third most diagnosed form of cancer and the second leading cause of cancer mortality in the United States. Due to these alarming findings, it is important to research ways to reduce prevalence and worsen health outcomes related to CRC. Previous research has found that having up-to-date CRC screening is associated with early detection of CRC. While regular doctor visits are associated with having up-to-date CRC screening, people living in rural areas are less likely to have screening uptake. Thus, this study evaluated the interconnected relationship of annual healthcare utilization, rurality, and CRC screening behaviors in the United States. A cross-sectional analysis of 2023 National Health Interview Survey data was performed to examine the mentioned association. The primary outcome was up-to-date CRC screening, and the exposures of interest were annual checkups and rurality. Weighted multivariable logistic regression models were applied. Out of 13,695 screening-eligible respondents, the majority had up-to-date CRC screening (65.3%), resided in large metropolitan areas (54.0%), and had an annual checkup (89.6%). Those living in rural areas had the lowest CRC screening utilization (66.4%) compared to large metropolitan areas and small/medium metropolitan areas (71%) despite receiving an annual checkup ($P < 0.001$). In adjusted analysis, participants reporting an annual checkup (AOR 4.57; 95% CI, 3.9-5.4) and living in a large metropolitan area (AOR 6.91; 95% CI, 6.0-8.0) were more likely to be up to date on CRC screening. Participants who had an annual checkup had almost 5 times increased odds of CRC screening uptake than those without an annual checkup, regardless of rural status (AOR 4.57; 95% CI, 3.88-5.37). Our findings suggest that having an annual healthcare utilization is a key component of CRC screening uptake. Effective implementation of patient-provider communication and provider referral to CRC screening through yearly doctor visits may improve screening adherence.

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Parent Perspectives on Afterschool Rx: A Rapid Qualitative Study

Presenter: Tiffany Amoako Twum

Author(s): Tiffany Amoako Twum, Lauren von Klinggraeff

Faculty Sponsor: Lauren von Klinggraeff, PhD

Affiliation: Department of Community & Behavioral Health Sciences

ABSTRACT

Childhood physical inactivity is associated with an increased risk of long-term cardiovascular disease and other adverse health outcomes. After-school programs may provide an important opportunity to increase children's physical activity and support healthy behaviors. Afterschool Rx is a program that allows healthcare providers to prescribe structured afterschool programming through partnerships with community organizations. The purpose of this qualitative study was to explore parents' perceptions of the acceptability and feasibility of the Afterschool Rx program and to identify areas for improvement. Families enrolled in the program were invited via text message to participate in brief interviews. Six of twelve eligible parents completed interviews, with an average duration of about six minutes. Interviews were transcribed, de-identified, and analyzed using rapid qualitative analysis to identify key themes. Preliminary findings suggest that parents generally perceive Afterschool Rx as both feasible and acceptable. Participants highlighted easy enrollment and supportive after-school activities as key strengths of the program. However, some parents reported communication challenges and inconsistent staff engagement, suggesting opportunities for clearer expectations and improved program support. These findings provide insight into program implementation and may inform future improvements to enhance participant experience and program sustainability.

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Correlates of Intraindividual Sleep Variability in Young Adults

Presenter(s): Rakshna Anithadevi Sivakumar

Author(s): Rakshna Anithadevi Sivakumar, Ben Aderinwale, Tanika Chevalier, and Lauren von Klinggraeff

Faculty Sponsor(s): Lauren von Klinggraeff, PhD

Affiliation(s): Department of Community & Behavioral Health Sciences

ABSTRACT

Sleep research has predominantly focused on interindividual averages – the average sleep duration across a group of people. Comparatively less attention has been given to intraindividual variability (IIV)—the day-to-day fluctuations in sleep/wake patterns within an individual. Despite its potential significance for health, IIV remains understudied. The present study draws on a subset of studies from a larger scoping review to examine correlates of daily IIV in sleep/wake patterns among young adults. We conducted a systematic search of peer-reviewed empirical studies across PubMed, CINAHL Plus, PsycInfo, Scopus, and Embase between the years 2014 and 2022. Inclusion criteria for studies required participants to be between the ages of 18 and 34, conduct daily sleep assessment for at least three days, provide quantitative reporting of sleep data, and the examine the relationships between sleep variability and non-sleep variables (e.g., health outcomes, affect). Studies were excluded if they used only polysomnography (PSG) to measure sleep, as PSG requires scheduled measurement times that can influence IIV. Across the 26 studies analyzed, there were a total of 5,892 participants with a mean age of 21 years (SD 2.67 years). On average, 63.86% of participants were female. The most common sleep measurement tools utilized were wearable devices (20 studies) and daily diaries (15 studies). The average sleep measurement period across the studies was 46.4 days (median 14, range 6-730). Included studies examined a wide range of correlates of IIV in sleep, including cardiovascular outcomes, decision-making, mood, affect, stress, and academic performance. Correlates most consistently associated with greater IIV in one or more aspects of sleep/wake patterns among young adults were poorer cardiovascular health and higher stress. Sleep schedules and attitudes towards sleep as a time commitment were associated with increased sleep regularity among experimental studies. Overall, IIV in sleep appears to be linked to health outcomes in young adults. Greater intraindividual consistency in sleep may be associated with better health outcomes, particularly for stress and cardiovascular health. To gain a clearer understanding of IIV and its relationship to health among young adults will require more regular measurement and consistent reporting across the literature.

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Microwave-Assisted Solid-Phase Synthesis of Biologically Active Peptides

Presenter(s): Emil Ayala-Cosme

Author(s): Emil Ayala-Cosme

Faculty Sponsor(s): Matteo Borgini, PhD

Affiliation(s): Department of Biological Sciences

ABSTRACT

Therapeutic agents are developed to target specific molecules in the body as a means to restore their original function. Biological molecules that are therapeutically modulated can be considered “druggable”. “Undruggable” biomolecules, often have structural characteristics that causes traditional drugs to be ineffective. The Nrf2 transcription factor, involved in the cellular anti-oxidative pathway, is considered one of these undruggable targets. In the cell, Nrf2 is translocated to the nucleus by importins where it will interact to activate the transcription of antioxidant response elements also known as AREs. In cells, this antioxidant protection can help counteract effects caused by events, such as lack of sleep and malnutrition, that increase oxidative stress in cells. While beneficial in healthy cells, Nrf2’s increased expression in cancers normally found in the lungs, livers, and other smooth muscle organs, has been linked to heightened chemo-resistance and a worsened prognosis for patients. The goal of our research is to synthesize peptides that act as protein-protein interaction inhibitors that will prevent the expression of Nrf2-dependent genes. By inhibiting the expression of Nrf2-dependent genes we hope to counteract these protective elements in cancer cells that may lead to chemo-resistance and increased cancer cell proliferation. Our study attempts to develop novel approaches to modulate undruggable targets from a medicinal chemistry perspective. The microwave-assisted solid-phase peptide synthesis has been employed for the quick synthesis of various peptides. This report will discuss the details of the synthesis process along side the techniques employed for the purification of the compounds and the screening of our peptides against Nrf2-dependent chemo resistant cancer cells.

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Oligodendrocyte Histone Methylation in Preterm Infant White Matter

Presenters(s): Jasmine Baidoo

Author(s): Jasmine Baidoo, Hailey Netherton, and Evan Goldstein

Faculty Sponsor(s): Evan Goldstein, PhD

Affiliation: Department of Biological Sciences

ABSTRACT

Premature birth remains a significant healthcare challenge, with survivors experiencing a range of neurodevelopmental disabilities. They can develop intellectual disabilities, locomotor deficits, and behavioral disorders that profoundly impact their quality of life. Underdeveloped respiratory tracts in preterm infants lead to inadequate blood oxygenation and expose the brain to hypoxia. This deprivation disrupts brain white matter development, resulting in diffuse white matter injury (DWMI). In the central nervous system, there are oligodendrocyte lineage cells (OLCs) where the oligodendrocytes (OLs) are responsible for producing myelin, a crucial component of the brain's white matter. These cells wrap around axons to form a protective layer, enabling faster electrical impulses to travel efficiently throughout the brain. This process occurs primarily after birth, making it particularly vulnerable to hypoxia. Histone methylation regulates the expression of genes involved in white matter development. Since OL maturation involves extensive chromatin remodeling, the methylation status of histone 3-lysine 9 (H3K9) is important for OL development and myelin formation. The findings from our preclinical mouse model of DWMI suggest that histone modifications play a role in disease pathogenesis. This research aims to investigate whether abnormal H3K9 methylation found in our mouse model of DWMI is also present in preterm infants. To approach this study, postmortem human brain samples from the biobank of the Children's National Hospital (Washington, DC) were analyzed. The staining density of H3K9 methyl marks in the nuclei of OLCs was quantified using antibodies specific to H3K9 and OL markers and assessed using ImageJ image analysis software. Consistent with findings in the preclinical mouse model, preterm infants have more H3K9 methylation in OLs than full-term infants. Completing this study validates the relevance of the preclinical mouse model of DWMI and improves our understanding of the pathogenesis underlying preterm birth-related brain injury. This work has the potential to inform therapeutic strategies for preterm birth survivors.

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Exchange Rate Fluctuations and International Students Enrollment in the US

Presenter: Roberto Barrera Jaimes

Author(s): Roberto Barrera Jaimes and Simon Medcalfe

Faculty Sponsor: Simon Medcalfe, PhD

Affiliations: Hull College of Business

ABSTRACT

This study examines the relationship between exchange rate fluctuations and international student enrollment in the United States using country-specific time-series regressions for sixteen of the top sending countries in the world. To account for the forward-looking nature of education decisions, the analysis incorporates a two-year lag on the exchange rate, reflecting the fact that families typically respond to macroeconomic conditions well before actual enrollment occurs. All regressions control for inflation, GDP, youth population unemployment, and an economic freedom index, allowing the focus to remain on macroeconomic dynamics rather than purely demographic or institutional factors.

Preliminary results, contrary to what was expected, reveal a consistent and statistically significant positive association between exchange rate depreciation and U.S. student enrollment for most countries in the sample. This pattern is especially pronounced among emerging and middle-income economies, including Brazil, Chile, Colombia, India, Ghana, Nigeria, Pakistan, and Peru, as well as in select advanced economies such as Spain and the United Kingdom. In contrast, the exchange rate effect is weaker or statistically insignificant in countries with relatively stable currencies and strong domestic higher-education systems, such as France and Germany.

While currency depreciation increases the local-currency cost of U.S. education, the persistence of a positive relationship suggests that exchange rates may operate as a push factor rather than a deterrent. In many economies, depreciation coincides with broader macroeconomic uncertainty, inflation expectations, loss of trust in local currency, or political risk, potentially motivating households to view U.S. education as a form of risk diversification or investment in dollar-denominated human capital. GDP generally exhibits a positive and significant relationship with enrollment, indicating that economic capacity remains an important constraint, while youth unemployment and institutional measures display more heterogeneous effects across countries.

Overall, the findings suggest that exchange rate movements capture broader economic incentives for international mobility rather than simple affordability effects. Although this analysis is preliminary, the results point to meaningful cross-country differences in how macroeconomic instability shapes international student flows and motivate further robustness checks and extended empirical approaches.

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β -Arrestins as Predictors of Overall Survival in Cancer

Presenter(s): Alaira Beecher

Author(s): Alaira Beecher and Dr. Georgios Kallifatidis

Faculty Sponsor(s): Dr. Georgios Kallifatidis, PhD

Affiliation(s): Department of Biological Sciences

ABSTRACT

Cancer stem cells (CSCs) represent a subpopulation of tumor cells with stem-like properties, including self-renewal and the ability to differentiate into multiple cancer cell types. These cells are believed to drive tumor initiation, progression, metastasis, therapy resistance, and recurrence, thereby posing a major challenge to effective cancer treatment. Currently, there is no golden standard marker for identification of CSCs. Recent evidence suggests that β -arrestins (multifunctional adaptor proteins that scaffold signaling complexes and regulate intracellular pathways) regulate CSC marker expression and CSC properties and play a crucial role in therapy resistance and metastatic potential. Therefore, we analyzed in the current study the prognostic significance of β -arrestins compared to CSC marker expression in tumor samples. This study investigated whether β -arrestins (ARRB1 and/or ARRB2) can predict overall survival and disease outcomes in cancer patients, particularly due to their important role in regulating CSC properties.

The cBioPortal, a database that is used to analyze de-identified genomic and clinical data from cancer patients, was used as the form for data collection. The majority of the data collected comes from the cancer genome atlas (TCGA), a research database that maps the genomic alterations responsible for cancer. This study analyzed three common cancer types: liver hepatocellular carcinoma, lung adenocarcinoma, and bladder urothelial carcinoma. The results were compared to the prognostic significance of 7 established CSC markers (CD44, PROM1, ALDH1A1, BMI1, FUT4, EpCAM, CD90 (THY1)). Overall, it was shown that individual marker expression alone was insufficient to predict overall survival. Our findings showed that these markers yielded more meaningful and significant results when dual marker expression rather than individual markers were analyzed. In conclusion, the results of the current work highlight that co-expression of ARRB1 and ARRB2 can serve as a prognostic indicator for overall survival in certain cancers, such as hepatocellular carcinoma. This research may be helpful in identifying biomarkers that predict overall survival and outcome in cancer patients.

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The Role of PTK7 Related to Prostate Cancer

Presenter: Julie Bishara

Author(s): Julie Bishara and Sharanjot Saini

Faculty Sponsor: Sharanjot Saini, PhD

Affiliation(s): Department of Biological Sciences

ABSTRACT

Protein Tyrosine Kinase 7 (PTK7) is an important enzyme that is involved in the process of WNT signaling which directly affects the proliferation of cells, apoptosis, cell migration, in cell adhesion, cell polarity, and actin cytoskeleton reorganization. It plays a critical role in the overall development of embryonic cells and aids in the homeostatic self-renewal in tissues. Its dysregulation is associated with cancer, developmental shortcomings, and degenerative disorders. PTK7 was found to have an effect in various cancer types ranging from lung, liver, and colon cancers. Some cancer types, such as the ones previously mentioned, are associated with higher PTK7 levels which are correlated with a low prognosis and higher metastatic potential. Knockdown of PTK7 has been found to prevent proliferation and induce apoptosis in cancer cells such as liposarcoma. PTK7 is a relatively novel kinase involved in cancer research, however a surface biotinylation assay (done by Dr. Saini) showed the upregulation of PTK7 in prostate cancer cells in vitro. This project will focus on investigating usages of PTK7 to treat and target prostate cancer. Prostate cancer is one of the leading life threatening diseases in men with limited treatment options available, due to increased drug resistance in different subtypes of prostate cancer. The goal of this research project is to examine correlations between PTK7 and different prostate cancer cell types, to allow for future exploration of the possibility of engineered exosomes that specifically target cells that express PTK7. This would be done by treating exosomes, drug carrying exosomes, with PTK7 to present a PTK7 antibody on their surface. The exosome will recognize other antigens PTK7 presented in prostate cancer cells and deliver the drugs to the target, inducing cell death. Dr. Saini has previously worked with engineered exosomes targeting non-PTK7 cells by delivering drugs to the target via biomarkers on the surface of the exosome.

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JMJD1C Directs Oligodendrocyte Maturation Following Developmental Brain Injury

Presenter(s): Isaac Bloom, Dr. Evan Goldstein

Author(s): Isaac Bloom, Dr. Evan Goldstein

Faculty Sponsor(s): Dr. Evan Goldstein

Affiliation(s): Department of Biological Sciences

ABSTRACT

Over ten percent of babies are born preterm. Often, the lungs of preterm infants are not fully developed, leading to brain hypoxia. This causes white matter injury (WMI) in the brain characterized by aberrant axonal myelination. Previous research using a hypoxic mouse model of developmental WMI demonstrated that recovery in an enriched environment (EE) promotes generation of oligodendrocytes (Ols), the myelinating cells in the brain, myelination, and locomotor recovery. To explore mechanisms of this EE-induced recovery, OL-specific RNA sequencing was performed. One particularly interesting differently expressed gene is Jumanji Domain Containing 1C (JMJD1C), a histone demethylase that promotes lipogenesis, a critical process for myelination. JMJD1C is downregulated after hypoxic injury but upregulated during EE-induced recovery. We hypothesize that JMJD1C directs enrichment-induced effects on following hypoxia. To determine the role of JMJD1C in Ols after hypoxic injury, JMJD1C was conditionally knocked-out of OL precursors. Transgenic mice were housed in hypoxia (10.5% O₂) from postnatal day (P) 3 through P11. Mice recovered in either a standard cage or an enriched environment, until P45. OL dynamics were assessed in the subcortical white matter using immunohistochemistry and fluorescent confocal microscopy. Our data suggests that OL-specific knockout of JMJD1C diminishes the oligodendrogenic response present during enrichment-induced recovery from hypoxia due to an accumulation of post-mitotic pre-myelinating Ols. More work will be done to elucidate the role of JMJD1C in myelination and functional recovery. This work has the potential to identify a therapeutic target for preterm infants with white matter injury.

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Oral Cancer Health Awareness at Augusta University

Presenter(s): Sarah Buckman

Author(s): Maivy Hyunh and Sarah Buckman

Faculty Sponsor(s): Steven Coughlin, PhD, MPH, Madison Richards, DMD, Lorriane Odhiambo, PhD, MPH, Ban Majeed, PhD, Monirul Islam, MBBS, MPH, PhD

Affiliation(s): Department of Biostatistics, Data Science and Epidemiology, School of Public Health

ABSTRACT

Oral cancer has become a significant public health concern in the United States, with over 45,000 cases being recorded as of 2020, and the ages of diagnoses are becoming younger. Despite this fact, many are unaware of what oral cancer is and the risks and prevention methods, specifically among the younger generation. Early detection of oral cancer is critical, as screenings can identify lesions and sores, which monitor the risk of disease progression. Human papillomavirus is a main cause of oropharyngeal cancer, for which there are no easily identified early lesions, and require close monitoring. This, along with how the Human papillomavirus vaccines were rolled out in 2006, resulted in HPV being the most common risk factor for oral cancer. Tobacco and alcohol are the next leading risks for oral cancer. Tobacco releases harmful carcinogens, weakens the immune system, and mutates cells. Tobacco can be found in various products, including cigarettes, hookah, snuff, and betel quid. Alcohol acts as an enhancer when combined with tobacco. There is no safe amount of alcohol that one should consume regarding cancer risk, and yet alcohol use is rampant on college campuses and among young adults. Tobacco use, alcohol consumption, dietary, and sexual behavior are established risk factors for oral cancer. Although research has emphasized the risks and preventative care that should be done to treat and prevent this disease, many are unaware of oral cancer.

The campaign aims to educate students at Augusta University about oral cancer, the risks, and prevention methods. Tabling events structured around the oral cancer campaign will be conducted in March and April; the number of students spoken with and the number of flyers distributed will be recorded. This project aims to promote health education for future healthcare and professional leaders, integrating preventative methods and awareness into their practice and community.

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Influence of Greek Mythology and the Hero's Journey in Disney's Hercules

Presenter(s): Sarabeth Campbell

Author(s): Sarabeth Campbell

Faculty Sponsor(s): Jennifer Trunzo, PhD

Affiliation(s): Department of History, Anthropology, and Philosophy

ABSTRACT

In 1923, the Walt Disney Company was founded and released its first feature film, *Snow White and The Seven Dwarves*, in 1937. Since then, the Walt Disney Company has been producing movies and television shows that have been loved for generations due to their unique storytelling and art. Combining creativity with renowned myths, fairytales, and folklore, Disney movies have become enjoyable classics that not only are entertaining but also teach children valuable life lessons that stay with them into adulthood. Many Disney films also seek inspiration from the Hero's Journey, which is a formula that storytellers have used to guide their stories or create characters. This article seeks to explore the impact that mythology and the Hero's Journey have had on the storytelling used in the 1997 Disney Film, *Hercules*, through analyzing the film with careful attention to details relating to myth and the Hero's Journey. This analysis showed that, while the film borrowed from Greek mythology and the Hero's Journey, the Disney version of Hercules' story deviates from the formula once in the movie. However, although the film is adapted from the Greek myth, the movie is almost entirely different from its origin. Instead, the film borrows characters, places, and aspects from different Greek myths to create a new story. Hercules received mixed reviews after its release. While some denounced the film for not staying faithful to the myth, others celebrated the movie for its creativity. Regardless of those differing opinions, the Disney version of Hercules remains a good example of how myths and the Hero's Journey can be combined with the creativity of storytelling to make a new story. Although it is an animated film, Hercules is still enjoyed by many for its characters, creative design, storytelling, and life lessons that stay with viewers long after they've gone the distance.

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Determining the Role of Neuronal Navigators in Axonal Guidance and Neuronal Connectivity

Presenter(s): Estrella Cano

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ABSTRACT

Neurons are the fundamental units of the nervous system. They are nerve cells that send messages all over the body. Axon guidance is an essential process used during brain development that ensures neurons form accurate and functional connections. The precise wiring of the neurons underlies cognition, motor coordination, and sensory processing. This process depends on the regulation of cytoskeletal dynamics, the internal structural framework that allows neurons to grow, change shape, and transport materials within developing neurons. Disruptions in axonal guidance, when neurons are guided towards their correct target, and neuronal polarization, in which a neuron develops distinct functional and structural regions, have been associated with neurological disorders such as autism spectrum disorder (ASD), epilepsy, and schizophrenia. Recent studies have shown Neuronal Navigators (NAV_s), a family of cytoskeletal interacting proteins, have mutations that are potentially linked to neurological disorders. However, the exact function of this protein remains unknown. Among the Neuronal Navigator proteins (NAV₁, NAV₂, NAV₃), NAV₂ has been associated with axonal elongation, the lengthening of axons, and coordination of actin and microtubule networks which is a cell's internal support system works together to shape the cell. All of the NAV_s share a common AAA ATPase domain which currently has an unknown function, but it is likely critical to regulate cytoskeletal interactions through ATP-dependent conformational changes. This leads to the question of whether the AAA domain of NAV₂ is the primary regulator in neuronal polarization, axonal growth, and controlling cytoskeletal organization. To address this question, cellular and molecular techniques will be used to design and characterize NAV₂ constructs with domain-specific disruption of the AAA domain. Primers were made to isolate and amplify the AAA ATPase domain which we then tagged with green fluorescent proteins. The resulting construct was expressed in N2A cells, which are mouse neuroblastoma cells that behave like nerve cells, so they are helpful for studying how neurons grow, communicate, and for evaluating changes in cytoskeletal organization, neuronal morphology, and axon growth. I co-transfected cells with cytoskeletal markers including actin markers for growing microtubules. This experiment demonstrated that NAV₂ is associated with polymerizing microtubules. We confirmed this association in cultured embryonic rat cortical neurons. This project aims to clarify the functional role of the AAA ATPase domain in NAV₂ function to provide structural insight into how NAV₂ integrates actin and microtubule dynamics to direct neuronal growth and connectivity. Understanding this mechanism may reveal new treatment targets for neurological disorders.

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Characterization of a New Murine Glioma Model for Oncolytic Virotherapy

Presenter(s): Adam Chadli

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Faculty Sponsor(s): Balveen Kaur, PhD

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ABSTRACT

Glioblastoma (GBM) is a highly aggressive cancer with a poor prognosis due to its high resistance to widely used therapies such as chemotherapy, radiation, and surgery. Oncolytic virus (OV) therapy is a promising biotherapy for treating solid tumors that use viruses such as Oncolytic Herpes Simplex Virus-1 (oHSV) to specifically target cancer cells. Human Nectin-1 (hNectin-1) is one of the major cellular entry receptors for HSV-1, making the virus more human-specific. This species-specific nature of HSV-1 results in its poor permissiveness to the currently utilized murine GBM models, hence demanding for a syngeneic murine model that recapitulates human GBM without being too immunogenic to better understand the effects of OV therapy in immune competent mice. We hypothesize that a murine GBM model for OV therapy with human Nectin-1 (hNec1) expression, along with tumor suppressor p53 knock out and overexpression of Ras oncogene will mimic human GBM and will allow for increased oHSV infection with less immunogenicity.

The generation of this model consisted of breeding humanized mice expressing hNec1 with mice knocked out for p53. PCR was then done to screen mice based on the genes of interest that they expressed, which is how the hNec1^{+/+} x p53^{-/-} model was created. Neural Stem Cells (NSCs) were harvested from the pups with hNec1^{+/+} x p53^{-/-} and were transduced with hRas lentivirus particles to induce hRas overexpression. Western blots were conducted to ensure the expression of the proteins of interest. Colony formation assay was then conducted to determine the tumorigenic potential of the transformed NSCs. To evaluate *in vivo* tumorigenic potential, 5x10⁵ NSCs were implanted in hNec1 mice followed by Magnetic resonance imaging (MRI) 21 days post tumor implantation. The brain was harvested, and immunofluorescence staining was performed to confirm human GBM-like heterogeneity of the tumors. Next, we treated these NSCs with increasing multiplicities of infection (MOI) of oHSV to evaluate the susceptibility of these NSCs to oHSV infection over time using Incucyte live cell imaging system. Additionally, oHSV induced killing of the tumor NSCs was assessed by MTT cell viability assay. Lastly, a survival study was conducted to determine if there was a survival benefit conferred by oHSV therapy compared to untreated control. Results from this study confirm the generation of a murine GBM model with a genetic background of hNec1^{+/+}/p53^{-/-}/hRas which did not show immunogenicity, was permissive to oHSV infection, mimicked human GBM-like heterogeneity and responded to oHSV therapy *in vivo*.

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The Impact of Shift Schedules on Nurses' Sleep and Health

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ABSTRACT

Nursing is the nation's largest healthcare profession, with nearly 4.7 million registered nurses (RNs) nationwide according to the AACN (American Association of Colleges of Nursing). Nursing can be a demanding field that is often associated in poor or irregular sleep. This scoping review aimed to describe the current state of the science of the effect of shift work or night work on interindividual sleep variability (IIV) among nurses, and its potential impact on physical and mental health outcomes.

The present study draws on a subset of studies from a larger scoping review of peer-reviewed empirical studies across PubMed, CINAHL Plus, PsycINFO, Scopus, and Embase for articles published between 2014-2022. For this sub-study, studies were required to review to examine full or part time nurses with shift work schedules. Studies were included if sleep was assessed daily for at least 3 days, reported quantitative sleep data, and examined relationships between sleep variability and non-sleep variables (e.g., diagnosis, mood, functioning). Studies were excluded if they used only polysomnography (PSG) to measure sleep, as PSG requires scheduled sleep that can alter free-living sleep patterns.

Six studies (1,367 participants) met the inclusion criteria. Five out of six studies were observational and measured sleep using an accelerometer/activity tracker. Sleep was measured for an average of 7.5 days (9.5 SD). The participants were majorly female (94.7%), and three out of six studies reported race/ethnicity. Studies primarily focused on day shift vs night shift or rotating shifts and the relationships between sleep IIV and acute health outcomes. Nurses working night shifts and rotating shifts had higher IIV for sleep duration, midpoint, and rest-activity rhythm relative to nurses working day shifts. Increased IIV in sleep was associated with higher immune cell expression, depression, anxiety, restlessness, and lower executive function.

Our findings showed that irregular sleep schedules from rotating and night shift work negatively affect the physical and mental health of nurses. Given that the wellbeing of nursing staff can impact patient outcomes, night shifts and rotating shifts may inadvertently contribute to poorer patient care. Investing in strategies to support stable sleep patterns among nurses is likely to promote health for both the nursing workforce and patients.

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Cell-Type Specific Localization of MAP7 in Mouse Nervous Tissue

Presenter(s): Laurie Conde-Rodriguez

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Faculty Sponsor(s): Stephen Tymanskyj, PhD

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ABSTRACT

As the primary functional cells of the nervous system (NS), neurons mediate electrical signaling across afferent and efferent pathways in both the central and peripheral NS. In neurons, there are several processes that must occur for a successful neural transmission. Microtubules are a key component of the neuronal cytoskeleton, contributing to axonal growth, synapse development, and intracellular transport. These processes are regulated by microtubule-associated proteins (MAPs), which influence microtubule stability and interactions with motor proteins. This study focuses on the MAP7 family of microtubule-associated proteins. The MAP7 family, which comprises MAP7 D1, D2, and D3, is crucial for cell motility and neural development, by regulating kinesin-1 motor proteins. Regulation ensures that cargo within the cell reaches its destination efficiently. MAP7 is known to be in the axonal branch points, such as dendrites, and the cell body within a neuron. MAP7's location on these points is pivotal for preventing axon depolymerization and promoting axon branching alongside maturation. In Nervous Tissue (NT), the MAP7 family can be expressed across several cells, namely astrocytes, oligodendrocytes, and neurons. This raises the question pertaining to the location of MAP7 in NT by cell type. To locate the areas of NT with the highest concentration of MAP7, brain and spinal cord was acquired from mouse CNS and was labeled with a fluorescent tagged MAP7 then observed on a Confocal Microscope. Cell density then be analyzed to find areas of NT with the highest expression of MAP7. Confocal microscopy is expected to reveal stronger MAP7 fluorescence intensity in neuronal cell bodies and processes compared to glial cells. MAP7 proteins are expected to be highly expressed in neuronal populations that primarily depend on structural remodeling and microtubule-based transport. Glial cells that support and stabilize the nervous system may also express MAP7. Knowing how MAP7 proteins are distributed among various cell types may help us understand how they function in the nervous system's structural organization, cellular transport, and neural development. Future work will analyze the disparity in concentration of MAP7-expressing cells when injury is present.

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The Architecture of Excellence: Synthesizing Federal Structural Variables and Nobel Laureate Experiential Narratives into Model Research Training Programs

Presenter: Dhilan Dahya

Author: Dhilan Dahya

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ABSTRACT

Biomedical research training lacks a standardized definition of the "optimal" environment, leading to high variability in trainee outcomes and career retention. This study aims to synthesize the structural components of federal training mechanisms with the qualitative formative experiences of Nobel Laureates to define a high-performance model for scientific development. A database of 27 NIH and private research training mechanisms was analyzed across nine variables, including funding, duration, and mentorship structure. Simultaneously, qualitative content analysis was performed on the official interviews and autobiographical accounts of 27 Nobel Laureates in Physiology or Medicine from 2000 to 2024. These two datasets were then integrated to create evidence-based model programs for the undergraduate, doctoral, and postdoctoral levels. Of the 27 programs analyzed, 7 focused on the undergraduate level, 11 on graduate training, and 9 on postdoctoral development. Analysis revealed a tiered pipeline with research effort requirements escalating from $\geq 75\%$ for undergraduate programs to 100% for postdoctoral awards, with total training durations ranging from 10 weeks to 8 years. Mandatory curricula across all NIH-funded mechanisms included Responsible Conduct of Research (RCR), while all early-career mechanisms required formal leadership training. Qualitative analysis of the Nobel Laureate narratives identified specific recurring factors: the vast majority of laureates cited "intellectual autonomy" or the "freedom to fail," while a substantial majority emphasized "methodological rigor" and "technical perseverance." Additionally, a significant portion of the accounts specifically noted the value of "interdisciplinary mentorship" or "mentors who defended unconventional research." The findings suggest that the most successful training environments intentionally integrate structural rigor with high-risk intellectual freedom and institutional advocacy. These synthesized model programs provide a blueprint for institutions to move beyond minimum compliance toward the "highest combination of factors" necessary for cultivating independent scientific leaders.

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Computational Performance of Julia-Based DMRG Algorithm

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ABSTRACT

Developing energy-efficient technology is a crucial challenge of the modern era. Contemporary devices rely on three classes of quantum materials: metals (copper), semiconductors (silicon), and magnets (iron). Broadly speaking, these materials allow the transfer, manipulation, and storage of digital information. Identifying the chemical compounds (naturally occurring or laboratory-synthesized) with the potential of enabling energy efficient technology is an area of ongoing research. To answer this scientific question, which is tied to serious economic impact, material scientists and engineers mimic the functionality of quantum systems in a computer simulation in search of high-performance materials. Through these simulations, we attempt to understand the laws governing atoms and molecules from a computational perspective. However, these analyses require substantial computational resources, incurring high operational costs. In response, physicists must devise computationally efficient algorithms (called numerical instruction) to investigate these material platforms economically. One such numerical algorithm, Density Matrix Renormalization Group (DMRG), is the focus of our work. Its purpose is to investigate the energy of the system, one portion at a time, while performing simulations required to computationally measure the physical properties (magnetic strength) of the quantum material. DMRG is particularly useful for studying materials with magnetic properties, especially chains of atoms which possess individual magnetic moments (also called a spin chain). The primary aim of our research is to assess the computational efficiency in implementing an algorithm such as DMRG. In our work, we compared Julia (a recently developed free and open-source scientific computing language) with MATLAB (a best-in-class commercially licensed scientific computing software). Benchmarking, carried out utilizing the Augusta University High Performance Computing cluster, showed that unoptimized Julia code was slower than the equivalent MATLAB implementation by a factor of ten. However, after applying computer memory optimization techniques the Julia code performance improved significantly, though still slower than the MATLAB version. In future, we plan to optimize the code even further to attempt MATLAB level efficiency.

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Investigating Glucose Aversion in the German Cockroach

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Faculty Sponsor(s): Jennifer Baltzegar PhD

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ABSTRACT

The German cockroach, *Blattella germanica*, poses numerous concerns for health as they vector various pathogens and produce asthma-causing allergens. Glucose aversion (GA) evolved rapidly due to human-induced selection pressure caused by exposure to insecticide baits containing glucose as an attractant. This raises concern in maintaining control over the regulation of the population as these insecticides have been an established method to maintain that control over their population. If this trait continues to thrive in the species, it could have several consequences, including but not limited to jeopardizing health. The GA phenotype is inherited in a dominant Mendelian pattern, which suggests that one gene is likely responsible for this trait. Our study aims to pinpoint this gene by conducting PCRs targeting gustatory receptors. We hypothesize that a gustatory receptor (GR) is the responsible gene because gustation (taste perception) is likely involved in taste regulation. To accomplish this task, we will compare DNA sequences of different cockroach strains. These strains are the wildtype German cockroach (WT BG), the glucose averse German cockroach (GA BG), the wildtype Asian cockroach (WT BA), *Blattella asahinai*, and finally the cockroach strain BAGA, which carries the glucose averse trait from *Blattella germanica* which has been introgressed into *Blattella asahinai*. We will compare the DNA sequences of all four strands by using a combination of primers and degenerate primers which would amplify our targeted genes of interest. We expect the primers to aid in analyzing and potentially targeting the GA trait if it is in fact caused by one of the gustatory receptors.

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Cellular Mechanisms Governing Axonal Transport of Synaptic Cargo

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Faculty Sponsor(s): Stephen Tymanskyj, PhD

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ABSTRACT

The growth cones of neurons enable target recognition and pathway finding of the axon, which is also found to have a significant influence on the directional migration of cargo at axon branch junctions. Neuronal cargos consist of molecules, proteins, or organelles that are synthesized in the soma, or cell body, and transported throughout the neuron to maintain neuronal function and survival. In most cases, supplies that are needed in a particular area of the cell are not produced locally. As a result, the cargo can be transported both anterograde and retrograde by ATP-dependent motor proteins like Kinesins and Dynein, attaching to cargo-filled vesicles on a microtubule track. Unlike many other cells, neurons can be characterized by their long, specialized processes, such as axons, which can extend great distances to reach their target locations, which, in humans, can span up to over a meter in length. This study aims to consider cargo distribution analysis (vesicle number), neuronal morphology (neurite length), & growth cone classification to generate a comprehensive idea of the relationship between neuronal structure and function intracellularly. Ultimately, this can be applied to the overarching idea that synaptic function is a process highly regulated and conserved, though not fully understood. To accomplish this, a photoconversion assay was used where rat embryonic sensory neurons or N2A neuroblastoma cells were transfected with a Lysosomal Associated Membrane Protein (LAMP-EOS4) and a near-Infrared Fluorescent Protein (iRFP) plasmid to serve as markers for cargo and cell morphology, respectively. While imaging, the soma is identified and isolated for photoconversion from green to red at 400 nm for roughly 10 seconds, followed by a 15-minute incubation period, where the photoconverted cargo is allowed to travel towards the distal axon terminals and growth cones. From there, the entire neuron is imaged under three main channels: an iRFP channel for morphology, a green channel for unconverted vesicles, and a red channel for converted vesicles. It has been determined from the data analyzed that there is in fact a positive correlation between the length of the branch of interest and the number of converted LAMP vesicles. Additionally, the characteristics of vesicle distribution in relation to varying distal and proximal branches from the soma were quantified. Ultimately, these results are being applied to improve the techniques of optogenetics and targeted cargo transport event imaging.

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Biotinylated Diarylpiperidinone Hybrids: Dual Function Probes for Targeted Cancer Therapy

Presenter(s): Holden Dinkins

Author(s): Holden Dinkins

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Affiliation(s): Department of Chemistry and Biochemistry, Augusta University

ABSTRACT

Cancer remains a leading cause of mortality in the United States, with heart disease the only condition responsible for more deaths. Breast and colon cancers are among the most prevalent, collectively expected to account for over 474, 474,000 cases and 95, 95,500 deaths in 2025. The complexity of these diseases is partly attributable to tumor heterogeneity and aggressive progression, which complicate management. Novel pharmacological agents offer therapeutic strategies for these diverse cellular populations, particularly when combined with other treatment modalities. Natural products offer promising avenues for enhancing drug development by modifying lead compounds with favorable pharmacokinetic profiles. One notable example is curcumin, a naturally occurring polyphenol extracted from the rhizome of turmeric. While widely recognized for its anti-inflammatory properties, curcumin also exhibits anticancer effects. However, its clinical application is limited by poor bioavailability and a lack of target specificity. Our research group has advanced the structural properties of curcumin, yielding a series of molecules based on a diarylpiperidinone scaffold. These derivatives demonstrate improved compactness, bioavailability, and aqueous solubility compared to curcumin. A critical area of investigation is elucidating the molecular targets of this scaffold, which can be explored through biotinylation, a process that attaches biotin to the scaffold. The resultant biotin moiety enables fluorescent tracking, facilitating identification of a conjugate's binding partners and, consequently, its molecular targets. Furthermore, the conjugates exhibit inherent anticancer properties. Given that breast and colon cancer cells express various biotin transporters, biotin acts as a targeting agent that enhances the selectivity of the compounds toward cancerous cells, thereby promoting cell death. In a synergistic manner, the diarylpiperidinone scaffold interacts with cancer cells, contributing to their eradication. Following synthesis via molecular hybridization and spectral characterization, eight biotin- diarylpiperidinone conjugates have been evaluated against breast and colon cancer cell lines, as well as normal epithelial cell lines. The results indicate that cancer cell death occurred at a significantly higher rate compared to normal cell death. Currently, these conjugates are undergoing animal testing to validate their efficacy.

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A Study of Medicine Within 18th-Century Rural Northeastern America

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Faculty Sponsor(s): Wendy Turner, PhD

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ABSTRACT

This paper seeks to understand the ways in which settlers in 18th-century Northeastern rural colonies took healthcare for themselves and their communities into their own hands. Life in the colonies was, uncertain, at best, as the colonists adapted to unfamiliar, occasionally hostile, environments, using their learned knowledge of medications from previous experiences in their homelands, and the expertise of physicians trained in Europe to survive. Integrating a variety of secondary sources and personal accounts from the time period, this paper explores the historical medical practices of the rural regions, within the Northeastern sector of the colonies, using qualitative data from medical members of these communities during this time to create a clearer picture of what healthcare was like for early settlers. The results of this study demonstrate that the 18th-century Northeastern colonies centered around sustaining the communities' health by protecting its members as part of recognizing their value within such a small society. The results also demonstrate the reliance on apothecaries and self-made "physicians" within the colonies who provided medical care when the community needed them most. This paper exists to give a greater insight into the similarities between modern healthcare, and the methods used care for others in the 18th-century. Within isolated communities in the colonies, individualistic societies became common to care for one another and preserve their resources, both medical and societal. We can see similar behavior in the modern day, with things like healthcare, food and other necessities, it is expected that people take their care into their own hands, rarely seeking the help of their community. The fear of medicine is another similarity between the past and present, with inoculation being a controversial choice in communities, just as vaccinations carry a stigma to some today. To a modern audience, health management in these rural 18th century communities will look more like our contemporary healthcare than they might first have considered. Things have changed, of course, but the point of caring for others has remained the same. Keeping the whole community healthy has always been a priority amongst humanity, no matter the location or period.

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How Race and Comorbid Factors Influenced Atrial Fibrillation Ablation Outcomes

Presenter(s): Christy Eapen

Author(s): Christy Eapen, Kalp Patel, Niraj Sharma

Faculty Sponsor: Hongyan Xu, PhD

Affiliation(s): Department of Biostatistics, Data Science, and Epidemiology, School of Public Health, Northside Hospital Gwinnett

ABSTRACT

Atrial fibrillation ablation has become an increasingly common procedure, with persistent disparities present. This study analyses how race and comorbid conditions influenced atrial fibrillation ablation usage and safety. Data was extracted from the National Inpatient Sample (NIS) from 2012-2022 and analyzed using SAS Analytics. Conditions were coded using the International Classification of Diseases (ICD); 2015 was not included due to the ICD-9 to ICD-10 transition. Native American groups were excluded from analysis due to small population size which limited statistical significance. ICD-9 codes included 427.31, 427.32, 427.5, 427.41, 427.42, 37.34, 37.35, and 37.36. ICD-10 codes included I48.0, I48.1, I48.11, I48.19, I48.91, I46.0, I46.1, and I47.8. Comorbidities were calculated as a sum of individual conditions: Obesity, Diabetes, Heart Failure, Renal Failure, Peripheral Vascular Disease, Hypertension, Coronary Artery Disease, and Obstructive Sleep Apnea. Across 69,147 ablated patients from 2012-2022, there was a 0.6-0.9 % increase in average comorbid levels. African American patients also had the highest average comorbidity score (2.46). Although the number of comorbid patients undergoing ablation have gone up over the past decade, the procedure has become relatively more efficient, with complication-related cardiac arrest decreasing over the past decade from an average of 134 individuals in 2012 to 85 individuals in 2022. While overall cardiac arrest trends remain optimistic, African American males showed the largest percent increase (+116.7%) in ablation-related cardiac arrest cases from 2012-2022. Caucasians took the largest share of ablations over time, from 76.7% in 2012 to 78.3% in 2022. Men underwent 62.3% of ablations in 2012, and the count has since gone down to 59.7% in 2022. These results are thought-provoking regarding the notable rise in cardiac arrest cases in African American patients, implying persistent racial disparities. This could in part be linked to greater co-morbidities in African American patients. More randomized studies are needed for further explanation. Limitations are present: data is observational and not randomized. Surrogate ICD markers were used for atrial fibrillation ablation. Reliability depends on NIS coding accuracy.

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Current Atrial Fibrillation Ablation Demographics are not Nationally Representative

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ABSTRACT

Ablation is a standard therapy in managing Atrial Fibrillation (AF). This study examines ablations by race and US region. Data was extracted from the National Inpatient Sample (NIS) from 2012-2022 and analyzed using SAS Analytics. 2015 was not included due to the ICD-9 to ICD-10 transition. Census data was taken for 2010 and 2020 for those aged 65+. Native Americans were excluded from analysis due to small population size. ICD-9 codes included 427.31, 37.34, 37.35, and 37.36. ICD-10 codes included I48.0, I48.1, I48.11, I48.19, I48.91, and 02583ZZ. In 2012, the race distribution of 12,477 ablated patients was 77% White, 8% African American, 4% Hispanic, and 1% Asian. The 2010 US population was 82% White, 9% African American, 3% Asian, and 5% Hispanic. In 2022, the distribution of 6,881 ablated patients was 78% White, 10% African American, 5% Hispanic, and 2% Asian. The 2020 US population was 77% White, 9% African American, 3% Asian, and 8% Hispanic. Ablations grew by +9% in the Midwest and -10% in the South from 2012-2022. The 2020 Census found the Midwest population grew by 2% and the South by 10%. Results indicate underrepresentation of ablated Hispanics and Asians across the study period, and disproportionate representation of ablations in the Midwest and South. Ablations were found to be disproportionately performed among White patients. Structural barriers such as access to care, referral pathways, and insurance coverage may amplify these disparities and call for targeted interventions. Representation by race and region had minimal changes over the past decade, indicating slow diffusion of access beyond traditional centers and a significant overall imbalance. Centers with ablation-performing capacity beyond traditional centers and stronger equity initiatives are called to mitigate these disparities. Overall, population changes could be due to multiple mitigating factors. Randomized studies are needed for further explanation. Limitations are present: data is observational and not randomized. Data was extracted for 2012-2022 (excluding 2015), but US Census data was taken for 2010 and 2020. Surrogate ICD markers were used for AF ablation. Reliability depends on NIS coding accuracy.

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Healthcare Provider Perspectives on Integrating Breastfeeding Education into SafeCare

Presenter(s): Zahra Farooqi, Jhaanvi Panchal

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Faculty Sponsor(s): Ashwini Tiwari Pandey, PhD, MPH

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ABSTRACT

Breastfeeding initiation and duration rates in Georgia and the U.S. remain disproportionately lower, particularly among Black mothers compared to White and Hispanic groups. These disparities underscore inequity in maternal health and women's health, as breastfeeding is linked to recovery after childbirth, improved psychosocial health, and a decrease in risk of cardiovascular disease and various cancers. While community peer education programs are effective in increasing breastfeeding among Black mothers, the positive effects of these programs are limited by a lack of reach to vulnerable families. One way to improve access to health education programs is through integration within widely disseminated and complementary behavioral programs for comparable populations. Systematic braiding of both a parenting program and breastfeeding program at the development and curriculum levels has the potential to meet the more complex needs of families who are at risk for maltreatment. This project serves to examine user-centered feedback from health care providers that will guide the development of a program integrating breastfeeding education into the SafeCare parenting program, an existing evidence-based parenting program for families with children (ages 0–5) at risk for or reported for maltreatment. Interviews were conducted among 11 health care providers who have experience working with pregnant individuals to obtain their opinions on how the program would be structured and what the curriculum would include. Specific areas of inquiry included what the goals of a combined breastfeeding-parenting program should be, who the target population should be, logistics of program delivery, and potential barriers to participation. Data analysis is still underway, but results from rapid content analysis of interview content will be included in the final product and indicate the specific elements and themes that healthcare providers believe should guide the development of the combined Breastfeeding/Parenting curriculum targeted toward at-risk families. This work will be important to reducing disparities in breastfeeding among vulnerable mothers.

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A Bioluminescence Assay Studying Sequence Determinants of DHHC S-Palmitoyltransferase Specificity

Presenter(s): Gavin L. Feinberg

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ABSTRACT

S-Palmitoylation is a reversible post-translational covalent modification in which a 16 carbon palmitic acid is added to a cysteine residue on either the N-terminus (amino) or C-terminus (carboxylic) of a target protein via a thioester bond. This modification is carried out by a class of proteins known as palmitoyltransferases (PATs) all of which contain a highly conserved Asp-His-His-Cys (DHHC) catalytic motif. Palmitoyltransferase activity is an integral part of cellular regulation for membrane association, trafficking, stability, and signaling. Because of the vital role palmitoylation plays in cell signaling, alterations to PAT function may contribute to cellular disruptions in diseased states. There are hundreds of palmitoylated proteins and 23 human DHHC PAT enzymes. There is not a consensus sequence motif for palmitoylation, and it is not known how the amino acid sequence surrounding the modified cysteine residue contributes to enzyme-protein substrate specificity. To begin to address this question we have adapted the SWISS-KASH assay to study modification of specific amino acid sequences by different DHHC enzymes using bioluminescence resonance energy transfer (BRET). Sequences with known palmitoylation sites were fused to Nanoluciferase (Nluc) and expressed in HEK 293 cells together with DHHC enzymes and the BRET acceptor Venus, both of which were directed to the outer nuclear membrane (ONM) by fusion to a KASH domain. Palmitoylation of Nluc-peptide fusions by a given DHHC-KASH resulted in localization of the Nluc-peptide substrate to the ONM which was detected by bystander BRET. Our results show overall weak enzyme-substrate specificity; most DHHCs could localize most peptide substrates to some extent, with DHHC7 and DHHC17 often being the most efficient. However, the most efficient DHHC enzyme differed for different Nluc-peptide substrates, indicating a degree of specificity. These results demonstrate a tractable in-cell assay for studying DHHC enzyme function, and set the stage for future studies to determine how peptide sequences determine preferential palmitoylation by specific DHHC enzymes.

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Molecular Hybridization Approach Yields Promising Candidates for Melanoma Treatment

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ABSTRACT

Cancer is a major global health challenge, and the limitations of current chemotherapeutic agents' drug resistance, systemic toxicity, and the restricted effectiveness of single-target drugs underscore the urgent need for innovative therapeutic approaches. This study presents the design and synthesis of a new series of twelve hybrid molecules that integrate three pharmacologically significant components: a curcumin-mimic scaffold (3,5-diarylidene-4-piperidone), ibuprofen, and natural or unnatural amino acid linkers. Using a molecular hybridization strategy, all conjugates were synthesized, purified, and structurally characterized. Biological evaluations against three human cancer cell lines, A431 (skin), HCT116 (colon), and MCF7 (breast), revealed that several hybrids exhibited potent antiproliferative activity, with some surpassing the efficacy of the clinically used drug 5-fluorouracil. Notably, the fluoro-substituted analog incorporating glycine as its linker emerged as the most promising candidate, demonstrating consistent sub-micromolar inhibitory activity across all evaluated models. Mechanistic investigations indicated that this potent analog induces G1-phase cell-cycle arrest and promotes apoptosis, thereby disrupting pathways associated with cancer cell proliferation. Computational docking and molecular dynamics simulations further illustrated strong and stable interactions between the hybrid molecules and the MDM2 protein, a key negative regulator of the tumor suppressor p53. Enzymatic assays confirmed significant inhibition of MDM2 and subsequent activation of p53, corroborating the proposed multi-target mechanism of action. To evaluate translational potential, the lead compound was assessed in a murine melanoma model. In vivo studies demonstrated a considerable reduction in tumor growth and enhanced survival rates compared with cisplatin. Histological analyses revealed decreased tumor cellularity, extensive necrosis, and increased collagen deposition in treated tumors, all of which align with strong antitumor activity. Computational ADME predictions indicated high gastrointestinal absorption and low toxicity, suggesting favorable drug-like properties. Overall, this investigation shows that rationally designed ibuprofen-piperidone hybrid molecules, particularly the most potent fluorinated analog, represent promising candidates for next-generation multi-target anticancer agents. These findings provide a robust foundation for the continued preclinical development of hybrid therapeutics that enhance anticancer efficacy while minimizing systemic toxicity.

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“I’ve Got Out at Last”: “The Yellow Wallpaper”, AIDS, and Joyful Rebellion

Presenter: Nevaeh Fowler

Author(s): Nevaeh Fowler

Faculty Sponsor(s): Amelia Hall, PhD

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ABSTRACT

My paper harnesses the fictional story of “The Yellow Wallpaper” to help us better understand real-life responses to the AIDS crisis. Bringing these two narratives into conversation with one another reveals how systems of oppression worsen suffering in people society deems disposable, while simultaneously stimulating unconventional and joyous forms of rebellion as a response. To make these claims, I first employ the methodology of close-reading to construct a comparative analysis of the language of illness in “The Yellow Wallpaper” alongside the language used to discuss illness in newspaper articles and medical journals during the AIDS crisis. Second, I establish how this language results in similarly marginalized groups whose medical issues are deemed unworthy of societal concern—married women suffering from postpartum depression in 1890s America, on the one hand, and gay men suffering from AIDS in 1980s America, on the other. I then investigate how, in each case, recommendations for alleviating illness often result in the erasure of one’s identity. For the narrator of “The Yellow Wallpaper,” recommendations not to write or leave the house results in the erasure of her agency and personhood. For gay men during the AIDS epidemic, many recommendations to prevent the disease centered around erasing queer identity rather than providing medically accurate information on how the disease spreads. Finally, I demonstrate how these circumstances give rise to unusual and exuberant expressions of rebellion. The narrator of “The Yellow Wallpaper,” through gleefully crawling around on the floor, is able to reclaim her agency and identity in defiance of both her illness and her husband’s harmful treatment of it. Gay men similarly, through pride parades, celebrated their identities and refused to be defined by illness or stigma. In both narratives, joy functions as resistance, challenging systems that deny care, visibility, and agency. Bringing these two narratives into conversation with one another ultimately demonstrates that joyful rebellion, even in unconventional forms, can be a powerful and necessary act of survival in the face of systemic neglect.

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Precision of Dual-Task Mobility Measures to Differentiate Fallers from Non-fallers

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Faculty Sponsor(s): Deborah A Jehu, PhD

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ABSTRACT

Falls are a leading cause of injury among older adults, associated with declines in mobility due to decreased muscle strength, sensorimotor coordination, and executive function. Dual tasking is defined as the simultaneous performance of two tasks. Although the degree of sensitivity of dual-task gait evaluations in identifying abnormalities is still unknown, they may be more sensitive than single-task assessments. This study aimed to investigate whether dual-task gait assessments can better differentiate fallers from non-fallers than single-task assessments. Eighty-five participants aged 65 years or older (79.81 ± 8.25 years) were recruited from assisted living facilities (63.5%), nursing homes (2.4%), and the community (34.1%). Participants completed a health questionnaire to obtain demographic information, such as age, race, sex (17.19% female fallers, 12.42% female non-fallers), ethnicity, height, weight, education level, and fall history. Participants completed the Montreal Cognitive Assessment (24.48 ± 3.00 points). Inclusion criteria required that participants stand unassisted for 30 seconds, self-report gait impairments, and demonstrate English proficiency. Exclusion criteria included cognitive impairment, high physical activity, a MOCA score ≤ 18 points, or musculoskeletal/neurological conditions. Participants completed mobility tasks such as the Timed Up and Go (TUG) test. Participants were instructed to rise from a chair, walk 3 meters forward, turn, walk back, and sit. The dual-task version included counting backward by 3s while walking. Participants completed an 8-meter gait speed test at normal and fast paces. In the dual-task version, participants listed words starting with “S” and “A”. Mobility was measured using APDM inertial sensors, with data analyzed via Mobility Lab 2 software. We conducted separate repeated measures analyses of variance for Group (faller/non-faller) x Condition (single-task/dual-task) x Measure (TUG or gait parameters). Follow-up independent samples t-tests were performed on significant interactions. There was a significant Group x Condition x Measure for TUG parameters [$F_{(3,222)}=3.15, p=0.03, \eta^2=0.04$]. There was a trend for smaller single-task turn angle [$t_{(82)}=1.60, p=0.06$], single-task turn velocity [$t_{(82)}=1.50, p=0.07$], and dual-task turn angle [$t_{(82)}=1.50, p=0.07$] among fallers compared to non-fallers. There was no significant Group x Condition x Measure for gait parameters [$F_{(5,400)}=1.46, p=0.20, \eta^2=0.02$]. Dual-tasking was not more sensitive to differentiate fallers from non-fallers than single-tasking. However, TUG duration, a common clinical metric, failed to distinguish fallers from non-fallers, but precise inertial sensor measurements detected subtle differences. Our results support the use of objective measures of mobility to detect impairments over common clinical measures among older adults with poor mobility.

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Studies on Exosomes as Treatment of Mesenchymal Stem Cells

Presenter(s): Gavin Gnagey

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Faculty Sponsor (s): Ali Erolgu, Ph.D., D.V.M., Roni Bollag, MD, PH.D., Maleah Winkler, Ph.D.

Affiliation(s): Department of Neuroscience and Regenerative Medicine

ABSTRACT

Mesenchymal stem cells (MSCs) can differentiate into several cell types, repair damaged tissues, modulate immune response, and thus are essential for tissue homeostasis. Aging compromises the functionality of MSCs, reducing their regenerative capacity and limiting their potential in stem cell therapies. Hence, it is critical to restore the functionality of MSCs for an optimal outcome of autologous stem cell therapies. This study investigates the rejuvenating effects of exosomes derived from human umbilical cord blood (UCB). Previous experimentation involving heterochronic parabiosis, the surgical joining blood circulations of a young and an old mouse revealed that blood-borne factors from the young mouse are capable of reversing aging-associated health decline in the old one (Katsimpardi et al., 2014). Many blood-borne growth and signaling factors are known to be secreted and transported in exosomes as bioactive cargos (Amirzadeh Gougheri et al., 2023) (Kumar et al., 2020). Exosomes are membrane-bound extracellular vesicles (EVs) in size of 40 to 160 nanometers and carry bioactive molecules, including proteins, lipids, nucleic acids, and metabolites, that mediate intercellular communication and influence cellular function (Kalluri, R. & LeBleu, 2020). Notably, umbilical cord blood (UCB) is rich in growth factors and cytokines with known regenerative and anti-inflammatory properties (Rheaume, M.E, et al., 2022) (Ehrhart, J., 2t al., 2018) making it a promising source of therapeutic exosomes. It is the hypothesis of this project that exosomes derived from UCB express factors that are capable of rejuvenating aged MSCs. This hypothesis was tested by accomplishing the following aim: characterizing the molecular signatures of exosomes derived from UCB and adult peripheral blood (APB) and comparing data to observe differences in molecular cargo and regenerative capabilities. By leveraging the regenerative potential of UCB-derived exosomes, this study aims to establish grounds for a novel therapeutic approach to counteract age-related MSC dysfunction. Findings of this research are expected to significantly contribute to the field of regenerative medicine by offering a novel approach to enhance the effectiveness of stem cell-based therapies for aging populations. This study is currently in progress, as experiments are still underway.

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Transformative Research Discoveries with the Greatest Measurable Public Health Impact

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Faculty Sponsor(s): Andrew E. Balas, PhD, MD

Affiliation(s): Department of Public Health

ABSTRACT

Since 1990, advances in medical science have significantly reduced global mortality and disease burden through the development and implementation of effective healthcare, clinical, and preventive interventions. These advances improved treatment outcomes, increased life expectancy, and reduced the spread of infectious and chronic diseases worldwide. However, identifying which discoveries have produced the greatest impact requires evaluating measurable public health outcomes rather than focusing solely on scientific novelty. Examining these breakthroughs through population-level indicators such as mortality reduction, disease incidence, and improvements in survival rates provides a clearer understanding of their real-world significance. By analyzing the outcomes associated with major medical interventions implemented since 1990, researchers can better assess which scientific discoveries have most effectively improved global public health. This study employed a secondary data analysis of thirty major medical interventions introduced from 1990 to the present. Interventions were categorized into three primary groups: healthcare system procedures, clinical procedures, and preventive interventions. Data was collected from peer-reviewed scientific literature, epidemiological studies, and global health reports accessed primarily through Google Scholar and academic databases. Each intervention was evaluated based on measurable outcomes such as reductions in disease-specific mortality, changes in disease incidence, and improvements in survival or treatment effectiveness. Artificial intelligence tools, including ChatGPT and Google Gemini, were used to assist with organizing sources, categorizing interventions, and synthesizing relevant findings across literature. Across the interventions reviewed, disease-specific mortality reductions ranged from approximately 20% to over 90%, depending on the condition and type of intervention implemented. Preventive interventions, including vaccines and public health screening programs, were associated with reductions in disease incidence and transmission rates of up to 95% in certain populations. Clinical procedures and therapeutic innovations contributed to significant improvements in long-term survival and disease management, collectively preventing tens of millions of deaths worldwide. Healthcare system-level procedures also played a critical role improving access to treatment and enabling large-scale implementation of medical innovations across diverse populations. These findings demonstrate that the most impactful advances in medical science are characterized by measurable reductions in population-level disease burden and mortality. Categorizing interventions based on their functional role and public health outcomes provides a useful framework for identifying discoveries that have meaningfully improved global health. Understanding the real-world impact of these advances help guide future medical research priorities and inform public health strategies aimed at reducing disease burden worldwide.

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Transformative Effects of Habitat Fragmentation on Wildlife Movements

Presenter(s): Kennedy Gross

Author(s): Kennedy Gross

Faculty Sponsor(s): Robert Cromer, PhD

Affiliation(s): Department of Biological Sciences

ABSTRACT

This study aims to determine the effectiveness and need for culverts under roadways for wildlife movement. The development of roadways is a major cause of habitat fragmentation which in turn puts wildlife in danger. Wildlife travel for many reasons and do not understand the threat of roadways. This ultimately leads to vehicular wildlife collisions and deaths. Therefore, infrastructure is needed to preserve wildlife populations, a common type being culverts. Culverts provide a cost-effective and safe path for animals to travel and avoid the risk of roadways. This study focuses on wildlife movement through culverts across the Central Savannah River Area (CSRA). Cameras were placed at six various locations and photos were collected over a span of several months. A total of 28277 photos were collected and of those 1354 contained identifiable wildlife individuals of many species. It was found that many factors influence the usage of culverts by various species including water level, culvert size, time of year and day, and the surrounding environment. The information collected can be used to make recommendations to the Georgia Department of Transportation about how to improve infrastructure to reduce wildlife collisions and demonstrate how important it is to have methods for wildlife to safely cross roadways.

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Netrin-1 is Upregulated in Autosomal Recessive Polycystic Kidney Disease

Presenter(s): Prabhav Gundeboina

Author(s): Prabhav Gundeboina, Lillian Witherington, Jennifer C Sullivan , and Riyaz Mohamed

Faculty Sponsor(s): Jennifer C Sullivan, PhD

Affiliation(s): Department of Physiology

ABSTRACT

Polycystic kidney disease (PKD) is a genetic kidney disorder that is found in the perinatal period, early childhood or adulthood and is associated with high morbidity and mortality. Despite extensive study of PKD, the molecular mechanisms driving cyst growth remain undefined. Hallmark features of PKD include excessive epithelial cell proliferation and fluid-filled cyst formation, leading to decreases in kidney function. We previously demonstrated that netrin-1, a laminin-related secreted protein, is upregulated in rodent model of autosomal dominant form of PKD (ADPKD) and contributes to cyst growth. However, the role of netrin-1 in autosomal recessive PKD (ARPKD) has not been investigated. We hypothesize that netrin-1 is upregulated in ARPKD and correlates with disease severity. To test this hypothesis, male and female wild-type (WT) and *PKHD1* mutant polycystic kidney (PCK) rats, a model of ARPKD, were euthanized at 12 weeks of age, and blood and kidney tissues were collected for histological, biochemical, and molecular analyses. Renal function was assessed by blood urea nitrogen (BUN), cell proliferation by ki67 staining, and cyst burden was quantified using cystic index. Circulating and renal netrin-1 levels were measured by ELISA and immunostaining, and renal expression of netrin-1 and its receptors UNc5a, UNc5b, and DCC were evaluated by Western blot analysis. Renal ($P<0.001$) and circulating ($P<0.01$) levels of netrin-1 were greater in PCK rats compared with WT controls and were accompanied by elevated BUN ($P<0.0001$), increased epithelial cell proliferation ($P<0.001$), and enhanced cyst growth ($P<0.0001$). Moreover, there is a significant positive correlation observed between BUN and renal netrin-1 levels ($r=0.72$; $P<0.01$) in PCK rats. In addition, renal expressions of UNc5a ($P<0.01$) and UNc5b ($P<0.001$) were significantly upregulated, whereas DCC expression was markedly reduced ($P<0.001$) in PCK rat kidneys compared to WT. Immunostaining of human ARPKD kidney tissue showed strong netrin-1 expression mainly in cyst-lining epithelial cells relative to normal kidney underscoring clinical relevance of our study. In conclusion, netrin-1 and its receptors UNc5a and UNc5b are upregulated in both male and female ARPKD rats and in human ARPKD kidneys, suggesting a potential role for netrin-1 signaling in cyst growth and disease progression in ARPKD. Future studies will aim to elucidate the role of upregulated netrin-1 in cyst growth and disease progression in ARPKD.

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Thermodynamics of Reaction Enthalpies for Lipid Oxidation of Monogalactosyldiacylglycerol

Presenter: Emily Hammons

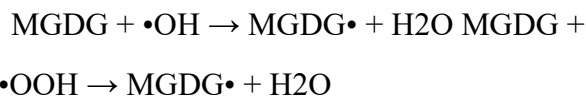
Author(s): Emily Hammons, Mandy Green

Faculty Sponsor: Mandy Green, PhD

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ABSTRACT

Monogalactosyldiacylglycerol (MGDG) is the most abundant polar lipid in nature and aids in the development of the chloroplast. Within the chloroplast, there is a specialized set of internal membranes called the thylakoid membrane which contains chlorophyll. The thylakoid membrane is found within the chloroplast and the cytoplasm of cyanobacteria. The thylakoid membrane contains protein complexes that aid in converting solar energy into chemical energy through photosynthesis. MGDG plays a crucial role in maintaining proper structure of the thylakoid membrane, which is essential for photosynthesis. MGDG also stabilizes and forms light harvesting protein complex II. During photosynthesis electron leakage from the light harvesting complexes in the thylakoid membrane generate reactive oxygen species (ROS). To model the oxidation of MGDG the reaction enthalpies of hydrogen abstraction by hydroxyl radical are determined. To determine the reaction enthalpies for MGDG, the optimized geometries are computed with *ab initio* methods. Reaction enthalpy is determined as a difference in the energies of the sum of products minus the reactants. All reactants, products, and intermediates geometries and energies are computed via quantum computational methods at RHF/UHF method and a Pople 6-31G* basis. Fragment molecular orbital theory (FMO) is also utilized in the quantum calculations to improve calculation time. Hydrogen abstraction reaction enthalpies are found and compared in the sugar head and in the tails of MGDG. The thermodynamics offers insights into the mechanistic role of MGDG under oxidative stress during photosynthesis.



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Long-Term Inflammatory Responses to Taste Bud Degeneration

Presenter(s): Taylor Hardeman

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Faculty Sponsor(s): Lynnette McCluskey, PhD

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ABSTRACT

Traumatic injury of the chorda tympani (CT) nerve causes degeneration of the anterior taste buds. Loss of taste can occur from head injury or middle ear surgery that impairs the CT nerve. Tumor necrosis factor 1 (*Tnfr1*) has been previously known to play an important role in taste bud regeneration. *Tnfr1* works to signal for cell repair, cell growth, or cell deconstruction in the case of infection or damage. Normal taste function is shown to recover within 3 weeks. However, long-term immune response activity remains unresolved. We tested taste bud regeneration in mice lacking *Tnfr1* at day 60. Unilateral CT sectioning was performed on wild-type and *Tnfr1*KO mice to detect CD45+ responses. Placebo surgery was administered to wild-type and *Tnfr1*KO mice as the control for surgical treatment. Mice were humanely euthanized before tongue collection. CD45+ serves as the marker for immune cells. Anterior taste buds were labeled with fluorophore-conjugated antibodies. Taste buds were analyzed 60 days post sectioning and post-placebo surgery using an immunofluorescence microscope. We found that there is a significantly increased number of CD45+ immune cells in *Tnfr1*KO post-injury, but not in wild-type. Additionally, CD45+ immune cells are significantly increased in *Tnfr1*KO compared to wild-type after placebo surgery. However, there was no significant difference between the surgical treatment in either strain. *Tnfr1*KO suggests immune responses may be overcompensating for the lack of *Tnfr1*. This is the first indication of prolonged immune activity. Significant long-term presence of immune cells in mice lacking *Tnfr1* indicates further research of immune cell type and mechanism to be conducted. Understanding the importance of *Tnfr1* and prolonged injury analysis is essential to taste bud regeneration.

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Evidence-based Parameters of a Healthy Lifestyle

Presenter(s): Liam Hashimoto

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ABSTRACT

The global rise in lifestyle diseases, conditions linked to unhealthy lifestyle choices, indicates that there is still a disconnect between knowledge of what constitutes a healthy lifestyle and the factors that contribute to these levels of unhealthiness. The purpose of this study is to identify and synthesize statistically supported lifestyle factors associated with experience-based health outcomes. A systematic review and knowledge extraction were conducted to collect and synthesize actionable factors. The eligibility criteria were that the factors required a measurable change in the likelihood of a specific harm with a minimum threshold of 5% and supported by at least two independent sources with experimental sample sizes of ≥ 100 and affecting a population $\geq 1,000$. The estimated effects of the factors were standardized to frequency-based outcomes and were organized into a structured table framework. A current total of 28 factors were collected that met the criteria. The most common data sources were censuses and public health journals. Categories include behavioral, environmental, and dietary factors that have had a substantial effect on the risk of lifestyle diseases. The three most influential factors, starting with the highest impact, were Body Mass Index and the risk of type-II diabetes, smoking consumption, lung cancer, and alcohol consumption and the risk of cancer mortality. The findings provided evidence that multiple overlapping factors heavily impact specific risks including some that are still less known by the public. Some of the lesser-known risks include not completing a high school degree which increasing the risk of atherosclerotic cardiovascular disease death and high omega-6/omega-3 ratio increasing the risk of cardiovascular disease. Many of the high-impact factors are components of people's daily lives. These habit-forming factors are what can cause the most long-term harm while also being, if changes are achieved, the opportunity for the greatest potential benefits.

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Defining the Interactome of Microtubule Associated Proteins

Presenter(s): Luke Horne

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Faculty Sponsor(s): Stephen Tymanskyj, PhD

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ABSTRACT

One key component of the neuronal cytoskeleton are microtubules which are responsible for the cell's structure and acting as tracks for intracellular transport. Regulation of these microtubules is accomplished by microtubule associated proteins (MAPs) which are needed for normal cell function and survival. MAPs can be divided into two categories including motor maps, which are responsible for intracellular transport, and nonmotor MAPs, which have various functions, such as regulating microtubule polymerization, depolymerization, and bundling. Further, MAPs are often spatially restricted within the neuron, a feature that is likely critical to their localized functions. A mutation in any MAP typically has a detrimental outcome. For example, the nonmotor MAP spastin is a microtubule severing ATPase, typically responsible for depolymerization of microtubules. In the case of spastin, a mutation in the *SPAST* gene can cause autosomal dominant hereditary spastic paraplegia (AD-HSP) leading to degeneration in long motor neurons in the spinal cord by disrupting microtubule dynamics, axonal transport, and cellular structures like the endoplasmic reticulum. Ultimately, AD-HSP causes progressive leg stiffness, difficulty walking, and poor bladder control. A key piece of missing information is how this MAP interacts with other proteins to mediate their functions. Until recently, addressing this question has been very inefficient. In this study, we will use the TurboID (TID) system to identify proteins that are mediated by the spastin MAP. The TID system will generate constructs that express spastin coupled to the TID biotin ligase. Then, we will transfect this system into neurons where a wild type or mutant spastin will continue to mediate their typical interactions. When biotin is added to our media, the TurboID portion of the construct will covalently link biotin to each protein that spastin interacts with. As wild type and mutant spastin-TID constructs interact with the proteome, each protein whose function is mediated by spastin will be linked with a unique biotin tag that can be examined with mass spectrometry. When comparing wild-type and mutant interactions, it will become apparent which protein interactions are important in inducing or preventing AD-HSP. Information from this study could be key in understanding which protein targets could prevent or reverse the effects of AD-HSP.

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Cyberpunk 2077: Food Association with Socioeconomic Statuses

Presenter(s): Maivy Huynh

Author (s): Maivy Huynh

Faculty Sponsor(s): Dr. Angela Bratton

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ABSTRACT

Cyberpunk 2077 is a dystopian video game in which a technologically advanced civilization impacts food accessibility across different socioeconomic statuses. *Cyberpunk 2077* takes place in Night City, a futuristic Los Angeles dominated by corporations, that revolutionized the food industry after corporate wars created a famine and pollution problem. Corporations such as *AllFoods* created scientific food interventions manufacturing meat tubes, human-kibble, and other synthetic food supplies. The quality and accessibility to these interventions varied among groups with differing socioeconomic statuses (SES), with lower SES communities relying on processed foods and elites having reserves for fresh, organic foods. Similarly, in real life, stratified systems categorize communities based on their prestige, restricting or allowing access to certain goods. Upper class communities can access fresh foods and products. However, lower classes may have food insecurity or food deserts in which the only cheap and accessible intake of food is fast-food chains (Holt-Giménez and Wang 2011). *Cyberpunk 2077* gives a warning to real life that the status that people hold limits accessibility on what foods may be produced.

Participant-observation of over twenty hours was conducted in the *Cyberpunk 2077* game to understand differing socioeconomic statuses and their accessibility to foods. Observations of the advertisements and buildings (fast food outlets, bars, etc.) were recorded to understand the food economy and the products being offered to players from one of the three life paths they chose before starting the game (Nomad, Street Kid, and Corpo). The Nomad path is considered lower class, as the Street Kid aligns with middle class, and Corpo path is associated with upper class. Public perception from other characters within the game reflects off the players' path either leading to prejudice, alienation, or authority. The qualitative data collected was analyzed by coding them into specific themes regarding the life paths, limitations and accessibility to the game's food economy.

Video games, usually fictional, can reflect reality, discussing potential outcomes of the socioenvironmental situations in modern times and converting them into what they could be (Yiannakoulis 2022). The stratified system in real life and in the game interlap with what is accessible to certain communities. *Cyberpunk 2077* is not just "another" game, but it is a game that engages real-world problems and depicts them as what could be in the future.

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Healthspan Effects of Disrupting Circadian Rhythm on *Drosophila melanogaster*

Presenter(s): Amy Jacob

Author(s): Amy Jacob, Jessica Hoffman

Faculty Sponsor(s): Jessica Hoffman, PhD

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ABSTRACT

Circadian rhythms, which internally regulate biological and alertness cycles in humans and other animals, have been disrupted with modern work schedules and use of devices. Light disruption can lead to circadian disorders and potentially affect healthspan and lifespan. Here we use the laboratory model organism, *Drosophila melanogaster* to model the effects of disrupting circadian rhythms. Previous studies in *Drosophila* have looked at the effects of light disruption on lifespan. However, few have analyzed healthspan, and most studies favored male animals. Complete darkness is associated with significantly longer lifespan in flies, with increased light intensity leading to markers of neurodegeneration. As organisms age, reactive oxidation species (ROS) increase, leading to heightened oxidative stress, and lesser life and healthspan. ROS levels are increased by light exposure in *Caenorhabditis elegans*, but effects in *Drosophila* are unknown. In this study, four genotypes of *Drosophila melanogaster* were used, and each was placed under 3 light cycles: 24 hr-dark, 24 hr-Light, and 12-hr-light/dark to determine light modulation effects on lifespan and healthspan. Healthspan was measured via climbing assays and oxidative stress assays. We found a clear trend of flies having shorter lifespans, and reduced health in complete light, though there was sex and genotype dependent variation. The flies survived the longest under 12 hr light-dark condition in every genotype except the control yellow-white (yw), which had a longer lifespan under complete darkness. However, both conditions had very similar lifespans. Females lived on average longer than their male counterparts, matching the literature, and yw and yw sphingosine-kinase 2 (ywsk2, a mutant) had the longest lifespans for most trials in all the treatments. There was no difference in performance between treatments in the climbing assay at 30 days of age, but at 45 days, there was a marked difference in performance, with complete light performing the worst, and complete darkness showing the best climbing ability. Flies under complete darkness survived the longest under oxidative stress, followed by flies on control lighting, then complete light. Females survived 10-15 hours longer than males, and the mutant ywsk2 survived the longest under all conditions. Findings from this study further our understanding of the effect of light cues on *Drosophila* healthspan, as well as photooxidative stress effects on *Drosophila*. Light exposure, whether modified or aligned with the *Drosophila* circadian rhythm, decreases longevity and healthspan, and longer light exposure was associated with faster mortality under oxidative stress conditions.

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Collective Oscillations of Ultracold Fermi Gases Across a Feshbach Resonance

Presenter(s): Alexis Jenkins

Author(s): Alexis Jenkins, Asanka Amarasinghe, Theja De Silva

Faculty Sponsor(s): Asanka Amarasinghe, Theja De Silva

Affiliation(s): Department of Physics and Biophysics, Augusta University

ABSTRACT

Ultracold atomic gasses are gasses that are at an extremely low temperature, nearing absolute zero. To achieve this low temperature, advanced laser-cooling techniques are used to provide an exceptional platform for studying strongly interacting quantum matter with tunable interactions. Recently, experimental advances have been done to investigate the collective oscillation frequencies. In the experiments that were conducted they used an axially symmetric trap. An axially symmetric trap is when the atomic clouds shape is equal in two directions and different in the third one. We use this to measure the collective oscillation frequency which is when the entire atomic cloud moves together. We will look at two different types of oscillations, monopole and quadrupole mode, of harmonically trapped Fermi gasses across a Feshbach resonance. Feshbach resonance is what allows you to transition from the Bose-Einstein Condensation (BEC) side to Bardeen-Cooper-Schrieffer (BSC) side, which depends on the interaction parameter. We first use hydrodynamic equations with a density dependent polytropic index to obtain analytical results for collective mode frequencies. For our results we found that the equations that we derived from the hydrodynamic theory were very similar to the results that were found experimentally. The slight difference in the two could be a result of the factors that were not taken into account in our equations, such as damping and temperature. As we cannot reach absolute zero with our current experimental setups. Second, we employ a sum-rule based approach combined with mean-field theory to calculate the monopole and quadrupole mode for single-band Fermi systems. This approach gives us two different moments to calculate that are based on quantum mechanical equations. Once we have found the different moments, we can use our results to compare with our hydrodynamic theory equations that were previously found and the experimental results. Our results reveal the dependence of collective oscillations on interaction strength and provide theoretical predictions directly applicable to ongoing and future experiments probing the dynamics of strongly correlated Fermi gases.

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Performance of Classical and Machine Learning Survival Models in GBSG2

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Faculty Sponsor(s): Durga H. Kutal, PhD

Affiliation: Department of Mathematics

ABSTRACT

Survival analysis is a statistical framework used to analyze time-to-event data, where the primary interest lies in estimating the time until a specific event occurs, such as death, disease recurrence, or treatment failure. A distinguishing characteristic of survival data is the presence of censored observations, which occur when the exact event time is unknown due to loss to follow-up or the termination of the study before the event occurs. Appropriate statistical techniques are therefore required to properly analyze such incomplete observations while preserving the integrity of time-to-event information. In this study, we analyze the German Breast Cancer Study Group dataset (GBSG2) to evaluate survival outcomes and compare the predictive performance of traditional statistical models and modern machine learning approaches. The dataset includes 686 breast cancer patients, among whom 387 experienced the event of interest, providing a suitable dataset for evaluating survival modeling techniques. We first conducted an exploratory survival analysis using the Kaplan–Meier estimator, a nonparametric method used to estimate survival probabilities over time in the presence of censored observations. Kaplan–Meier survival curves were constructed to examine differences in survival across several clinical subgroups, including menopausal status (pre-menopausal vs. post-menopausal), hormonal therapy status, and tumor grade. Results indicated no statistically significant difference in survival between pre-menopausal and post-menopausal patients. In contrast, hormonal therapy status and tumor grade showed statistically significant differences in survival, suggesting that these clinical factors may play an important role in influencing patient survival outcomes. We implemented several survival modeling approaches, including the Cox proportional hazards model, survival decision tree, random survival forest (RSF), and DeepSurv, a neural network–based extension of the Cox model. These models represent traditional statistical approaches and modern machine learning techniques designed to capture complex nonlinear relationships in survival data. Model performance was evaluated using time-dependent receiver operating characteristic (ROC) curves and the area under the ROC curve (AUC), which quantify the discriminative ability of each model to correctly distinguish between patients with different survival outcomes. Predictive accuracy was assessed at 3-year, 5-year, and 7-year time horizons. The Cox proportional hazards model produced AUC values of 0.743, 0.750, and 0.794, respectively. The survival decision tree achieved AUC values of 0.725, 0.705, and 0.713, while the DeepSurv model yielded 0.708, 0.677, and 0.751. The random survival forest model demonstrated the strongest predictive performance, with AUC values of 0.915, 0.817, and 0.863 across the three time points. Overall, the results suggest that machine learning–based survival models, particularly random survival forests, can provide improved predictive accuracy compared with traditional statistical models. These findings highlight potential advantages of nontraditional approaches in analyzing complex biomedical survival data and improving prognostic modeling in breast cancer research.

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The Impact of Social Media Influencers on Students' Perceptions of Success

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Faculty Sponsor(s): Dr. Elizabeth Culatta, Dr. Raquel V. Oliveira, Dr. Tim Sadenwasser

Affiliation(s): Pamplin College of Arts, Humanities, and Social Sciences

ABSTRACT

Social media has become a central part of daily life for young emerging adults (18-25 years old), shaping not only how they communicate but also how they define success, achievement, and self-worth. Platforms such as Instagram and TikTok frequently present highly curated images of academic excellence, financial independence, and influencer lifestyles, which can influence how emerging adults evaluate their own progress. Grounded in social comparison theory, this study examines how social media influencers shape college students' perceptions of success and well-being. The central research question explores how exposure to influencer content relates to students' definitions of success, engagement in upward social comparison, and emotional responses such as motivation, confidence, and self-esteem.

To explore this question, this study uses a scoping review to closely examine recent research on social media, influencer culture, and college students' perceptions of success. Peer-reviewed articles were gathered from academic databases using search terms related to social media use, influencers, social comparison, self-esteem, and student well-being. Studies were selected based on their direct relevance to social media and influencer content, a focus on college students or emerging adults, and publication within the past five years to reflect current social media trends. To maintain consistency, only studies conducted in the United States were included. The selected literature was reviewed to identify common themes, patterns, and theoretical approaches, particularly in how students define success and emotionally respond to online comparison.

Findings across the literature suggest that frequent exposure to influencer content is associated with higher levels of upward social comparison and more externalized definitions of success, such as financial status, lifestyle aesthetics, and social visibility. While some students report feeling inspired or motivated by influencer content, many studies also document increased pressure, self-doubt, and feelings of inadequacy when comparing themselves to idealized online portrayals. Students with higher social comparison orientation appear more vulnerable to these negative emotional outcomes, whereas those who emphasize intrinsic goals and personal growth tend to experience more neutral or positive effects.

Overall, this research highlights the complex role social media influencers play in shaping how college students define and pursue success. The findings contribute to a growing body of literature on digital culture and mental well-being and underscore the importance of media literacy and critical engagement with online content. Understanding these dynamics can inform educational interventions that help students navigate social media in healthier and more self-directed ways.

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Spatial Prevalence of Pyrethroid Resistance in an *Aedes albopictus* Population

Presenter(s): Anthony Jones

Author(s): Anthony Jones, Erin Przywara, JT Wicker

Faculty Sponsor(s): Jennifer Baltzegar, PhD

Affiliation(s): Department of Biological Sciences, Augusta University

ABSTRACT

Aedes albopictus (Asian tiger mosquito) is an invasive species that has spread globally, including to the Southeastern United States, and is a vector of arboviruses such as dengue fever (DENV), chikungunya (CHIV), and Zika (ZIKV) as well as being a vector for the zoonic parasite *Dirofilaria immitis*, which causes dog heartworm disease. Mechanistic modeling predicts that with continuing climate change as many as one billion additional people will be at risk of new exposure to such mosquito caused diseases by 2080, worst case scenario. Regions which were previously thermally unsuitable for transmission are predicted to initiate and retain suitability, suggesting that bloodborne arboviruses such as DENV have a high chance of becoming seasonally endemic to the Southeastern United States. As a nuisance species for pets and humans alike, measures such as independent spraying of pyrethroid insecticides have been implemented to reduce the number of mosquitoes in the local Central Savannah River Area (CSRA). Consequently, insecticide resistance in *Ae. albopictus* is emerging as a potential challenge for long-term management of this species. Pyrethroid resistance, known as knockdown resistance (*kdr*), is caused by mutations in the voltage-gated sodium channel (*vssc*) gene; mutations which have been shown to reduce the mosquito's susceptibility to pyrethroids. With continued use of pyrethroid insecticides, *kdr* mutation allele frequencies are expected to increase, as suggested by previous studies. Here, we report the first records of *kdr* alleles the Richmond County, Georgia population of *Ae. albopictus* mosquitoes. Using allele-specific polymerase chain reaction (AS-PCR), we genotyped 635 samples of *Ae. albopictus* from 15 sites in Richmond County, Georgia at the F1534S *kdr* locus. We then mapped *kdr* allele frequencies across the county using open-source geospatial visualization software. This study establishes a baseline of resistance prevalence that future studies will use to improve vector management and preserve the efficacy of pyrethroid insecticides.

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Development of a Non-Systemic PDE4 Inhibitor for IBD

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ABSTRACT

Inflammatory Bowel Disease (IBD), which includes Crohn's disease and ulcerative colitis, results from inflammation of the gut lining that often causes debilitating pain and diarrhea in up to 3.1 million patients in the United States. There is an urgent need for new IBD drugs because current treatments are often inadequate, and in severe cases, total colectomy is the only relief for patients. More recently, a novel anti-inflammatory drug (apremilast) was developed to treat psoriatic arthritis. Apremilast inhibits phosphodiesterase 4 (PDE4) in myeloid leukocytes, leading to increased cyclic adenosine monophosphate (cAMP) levels and subsequent suppression of proinflammatory cytokine production. This drug has a poor side effect profile that includes headaches, suicidal ideation, and diarrhea, largely due to its systemic delivery and inhibition of all PDE4 isoforms. The goal of this project is to develop an improved PDE4 inhibitor for IBD patients that is non-systemic and is specific for myeloid leukocyte PDE4. It was recently reported that a sildenafil analog (PDE5 inhibitor) containing a carboxylic acid group targets the gut epithelium without systemic delivery. A similar approach was utilized to design an apremilast analog containing a carboxylic acid group (carboxy-apremilast, car-apr). In silico analysis (SwissADME) predicts that car-apr will exhibit reduced GI absorption (non-systemic) and will be less susceptible to metabolic inactivation compared to apremilast. Analysis of published scRNAseq studies was carried out to determine which PDE4 isoforms are expressed in the intestinal epithelium and in myeloid leukocytes. Independent datasets identified PDE4B as the primary isoform in myeloid leukocytes, whereas PDE4C is enriched in the intestinal epithelium. Our study has designed an apremilast analog that is predicted to be more effective than the parent compound for treating IBD and with a lower side effect profile. We have also identified PDE4B as the preferred pharmacological target. Future work will focus on pharmacological studies in vitro and in cell lines, as well as molecular refinement to optimize PDE4B target specificity. The next steps will include preclinical pharmacokinetic and efficacy studies in rodent IBD models and ultimately highlight this approach for development into a novel drug to treat IBD patients.

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Engineering Multifunctional Hybrid Scaffolds for Pain and Inflammation Management

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ABSTRACT

Nonsteroidal anti-inflammatory drugs (NSAIDs) are integral to managing pain and inflammation; however, their clinical effectiveness is often limited by gastrointestinal toxicity and insufficient selectivity for cyclooxygenase-2 (COX-2). This research proposes a molecular hybridization strategy that combines three biologically favored scaffolds, namely isatin, rhodanine, and phthalimide, into a cohesive class of novel conjugates designed to enhance anti-inflammatory and analgesic efficacy while minimizing adverse effects. A diverse library of hybrid molecules was synthesized using an optimized Knoevenagel condensation protocol, followed by rigorous structural confirmation through comprehensive spectroscopic characterization. The biological efficacy of the synthesized hybrids was evaluated *in vivo* using a carrageenan-induced paw edema model and in analgesic assays employing the peripheral writhing and central hot-plate methods. Several candidates exhibited significant biological activity, with one hybrid displaying notable promise across all assessments. This lead molecule produced anti-inflammatory effects comparable to or exceeding those of standard reference drugs and demonstrated enhanced central analgesic activity. Importantly, when assessed for ulcerogenic potential, a substantial drawback associated with traditional NSAIDs, the lead hybrid produced significantly fewer gastric lesions than the reference drug, indicating a markedly improved safety profile. To elucidate the molecular basis for its superior performance, the lead candidate was investigated in COX-1/COX-2 enzyme inhibition assays and exhibited a pronounced preference for COX-2, consistent with the observed anti-inflammatory outcomes. Furthermore, the compound effectively reduced levels of pro-inflammatory cytokines, including IL-6 and TNF- α . Computational studies, including molecular docking, molecular dynamics simulations, and 2D quantitative structure-activity relationship (QSAR) modeling, further confirmed strong and stable binding interactions with COX-2 and identified key structural characteristics that contribute to its enhanced biological activity. Collectively, this study underscores the potential of molecular hybridization as a robust strategy for designing next-generation therapeutics. The lead hybrid molecule exemplifies a compelling combination of efficacy, selectivity, and safety, positioning it as a valuable prototype for the future development of safer and more effective anti-inflammatory and analgesic agents.

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Developing a Clinical-Grade Cryopreservation Method for Natural Killer Cells

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Faculty Sponsor(s): Ali Eroglu, Ph.D., D.V.M., Blaire Zeiders Ph.D., Zoya Kurago Ph.D., DDS.

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ABSTRACT

Adoptive cell therapy (ACT) refers to a form of treatment relying on the transfusion of immune cells into patients to eliminate various cancers and infectious diseases. Natural killer (NK) and chimeric antigen receptor (CAR) T cells are common types of immune cells used in ACTs. Recent findings suggest that NK cells derived from umbilical cord blood (UCB) represent a sustainable approach for allogeneic ACTs. Safe and reliable cryopreservation methods are needed to realize the potential therapeutic applications of immune cells fully. However, currently used cryopreservation methods typically employ undefined components and relatively high concentrations (~10%) of permeating cryoprotective agents (CPAs), such as dimethyl sulfoxide (DMSO) and glycerol, which are toxic to mammalian cells and induce multiple side effects. Thus, developing a chemically defined method that is animal product-free and safe for use in clinical treatments is essential.

This study aims to establish a basis for a clinical-grade method for cryopreserving therapeutic NK cells from human UCB. This will be achieved by adding a low concentration of a less-toxic intracellular CPA (ethylene glycol, EG) with higher concentrations of non-toxic extracellular saccharides/polymers, such as trehalose, dextran, and polyvinyl alcohol (PVA). These carbohydrates and PVA act as non-permeating CPAs and inhibitors of ice crystal growth. By using a lower concentration of less-toxic permeating CPAs, along with non-toxic extracellular CPAs, the UCB-derived NK cells can be properly stored for future uses in the treatment of patients with cancer and immunodeficient diseases. Human umbilical cord blood samples from Augusta University Medical Center will be collected, and the UCB-derived NK cells will be isolated to achieve the aims of (1) enhancing the osmotic tolerance of the NK cells and (2) improving the sub-zero cold tolerance of NK cells. This study is in progress, as experiments are still underway data has been obtained for exposure to PBS experiments and for -20 and -80 exposure experiments.

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Validating Sox2-CreERT2 for Conditional Gata3 Deletion in Developing Inner Ears

Presenter(s): Lotem Kol

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ABSTRACT

The transcription factor Sox2 marks progenitor populations that give rise to the sensory regions of the developing inner ear, whereas GATA3 is required for the proper differentiation and maintenance of sensory epithelial cells. In this study, an inducible Cre-loxP system was developed and utilized to trace the lineage of Sox2-expressing progenitors and conditionally delete GATA3 within these progenitors in the embryonic mouse inner ear. A Sox2-IRES-CreER^{T2} mouse line was crossed with a Cre-dependent reporter to validate temporal and spatial recombination prior to the conditional deletion of GATA3. Reporter expression at embryonic day (E) 11.5 demonstrated efficient labeling of Sox2-lineage cells within developing sensory regions following a single dose using immunofluorescent imaging. Analysis of both heterozygous and homozygous Cre genotypes demonstrated that the Cre genotype alone did not affect recombination efficiency or cause phenotypic changes under identical tamoxifen conditions. To assess the conditional deletion of GATA3, Sox2-CreER^{T2} mice were then crossed with GATA3^{lox/lox} mice and administered tamoxifen to induce recombination. A standard tamoxifen dose (3 mg/40 g body weight) was compared to a higher dose (4 mg/40 g body weight) at matched developmental time points to check for GATA3 expression. Standard dosing resulted in incomplete recombination and preserved GATA3 expression, while increased tamoxifen dosing produced more efficient recombination, reduced nuclear GATA3 expression in Sox2-lineage cells, and a more pronounced disruption of sensory epithelial organization. These findings demonstrate that the Sox2-CreER^{T2} driver efficiently mediates Gata3 excision in a time-dependent manner, validating the fidelity of this conditional knockout model. Consequently, this system provides a robust genetic platform for dissecting the distinct temporal requirements of Gata3 during critical developmental stages.

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A Low-Cost Calibration Apparatus to Ensure Rain Tipper Accuracy

Presenter(s): Richard Kremenz

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ABSTRACT

Tipping bucket rain gauges (TBRs) are commonly used for rainfall measurement. Rainfall measurements is an important task particularly in urban environment, due to the causal relationship between rainfall and flooding events. These devices consist of a funnel that collects rainfall and channels it into a small bucket. As rainfall accumulates in the bucket its weight eventually causes the mechanism to tip and empty the bucket. The tip is counted and the emptied bucket returns to its original position to repeat the cycle. To collect useful data a calibration must be performed to determine the bucket volume which, given the funnel collection area, can be directly related to the rainfall depth. Two methods of calibration are commonly used: static and dynamic. For static calibration, the volume of water required to cause the bucket to tip is measured and assumed independent of rainfall rate. Dynamic calibration measures the effective tipping volume at varying rainfall rates. This is necessary because the delay of the tipper repositioning itself after each tip causes a gap in data. Previous studies have shown that rainfall measured using static calibration can be underestimated by upwards of 30% due to this effect.

We have developed a low-cost, user-friendly, dynamic calibration system which uses a bank of three peristaltic pumps controlled by a Raspberry Pi to mimic varying rainfall rates. Our calibration protocol measures the tip rate for fifty pump rates, varying from 3 mL/hour up to 150 mL/hour which is equivalent to a rainfall rate of 15 in/hr for the TBRs investigated in our project.

We have tested our system using commercially available TBRs. A full calibration can be completed in 3 hours. We find that TRBs using a static calibration overestimate rainfall by 5% at low rainfall rates, but underestimate rainfall by 5% at higher rainfall rates when compared to dynamic calibration achievable using our system.

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Heparin Effects in Acute Kidney Injury (AKI)

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ABSTRACT

Acute kidney injury (AKI) is a major health concern which presents as a sudden reduction of kidney function. Ischemic AKI is an AKI subtype that is caused by a reduction of blood flow to the kidney for an extended period of time. In ischemic AKI, injury is thought to be a result of hypoxia, however, clinical trials targeting hypoxia have failed to improve outcomes in patients. Rather than from hypoxia, our data suggests that tubular injury in ischemic AKI may result from toxic damage to the tubular epithelia of the kidney. We think this injury occurs secondary to red blood cell (RBC) trapping. Red blood trapping, or the is the accumulation of RBC in the kidney medullary vasculature, occurs due to obstruction of the veins that drain the renal medulla. The accumulation of blood in the medullary circulation causes an increase in pressure within these vessels, leading to the extravasation of blood plasma and toxic proteins from degraded trapped RBCs into the surrounding tissues causing toxic injury to the surrounding tubular epithelia. The cause of venous obstruction in the kidney following a period of ischemia remains unclear. We hypothesized that this venous blockage results from blood stasis in the veins during ischemia that results in fibrin clots that then block the flow of blood through the renal veins upon reperfusion of the kidney. To test this hypothesis, we pre-treated rats with high doses of heparin to prevent fibrin formation before the ischemic period. We then clamp the renal artery of anesthetized rats for 45 minutes before removing the clamps and allowing reperfusion. Rather than preventing the obstruction and preventing kidney injury, heparin pre-treatment appeared to worsen kidney injury. The veins of animals treated with heparin remained obstructed and the weight of the kidneys in these groups was greater than that found in controls. These data indicate that post-ischemic venous obstruction of the kidney veins is not mediated by the formation of fibrin clots. We speculate that the increase of the kidney weight may have been due to heparin increasing permeability of the vasculature, leading to greater extravasation of blood proteins. To test this, we are quantifying the extravasation of blood proteins in heparin treated and control rats using Evans blue dye to label the blood proteins. Understanding how post-ischemic venous obstruction occurs in the kidney is crucial in determining future treatment for AKI.

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Advancing Cancer Therapy: Synthesis of Nrf2 Inhibitors

Presenter(s): Khadijah Ladoo

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Faculty Sponsor(s): Lindsey O. Davis, PhD and Matteo Borgini, PhD

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ABSTRACT

Chemotherapeutic resistance remains a major challenge in the treatment of cancers such as lung, esophageal, and head and neck malignancies. In healthy cells, nuclear factor erythroid 2–related factor 2 (Nrf2) functions as a cytoprotective transcription factor that regulates the cellular response to oxidative stress. In cancer cells, however, Nrf2 can become overactivated, leading to increased expression of antioxidants and radical scavengers that counteract the effects of several chemotherapeutic drugs. This allows tumor cells to survive treatment and contributes to the development of drug resistance. Despite its relevance in cancer biology, there are currently no known Nrf2 inhibitors available in clinical practice.

This project aims to develop peptide-based inhibitors to improve the efficacy of current cancer treatments in resistant tumors. Directly targeting Nrf2 is challenging due to its lack of a well-defined binding pocket. This challenge is addressed by synthesizing peptides designed to disrupt the nuclear localization of Nrf2 by blocking its interaction with importin- α . By preventing nuclear entry, Nrf2 is unable to activate antioxidant gene expression that protects cancer cells from chemotherapy.

To pursue this approach, a series of peptides designed to mimic the nuclear localization sequence of Nrf2 were synthesized to competitively inhibit its interaction with importin- α . Peptides were prepared using microwave-assisted solid-phase peptide synthesis (MW-SPPS) on Rink amide resin. This method accelerates the process by using microwave energy to reduce reaction times from hours to minutes. Amino acids were sequentially added to the growing peptide chain through repeated cycles of coupling and deprotection reactions. After synthesis, the peptide was cleaved from the resin.

The crude peptide products were then purified through reverse-phase flash chromatography followed by high-performance liquid chromatography (HPLC) under established solvent gradients. The identity and purity of the final peptides were confirmed using mass spectrometry and HPLC analysis. The purified peptides are currently being evaluated by collaborators in cancer cell models to determine whether they can inhibit Nrf2 activity and improve the effectiveness of chemotherapeutic drugs. This project provides a potential new strategy for targeting transcription factors and overcoming chemotherapeutic resistance in cancer.

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Man-made Cut Closure Restoration Study in the Satilla River Estuary

Presenter(s): Autumn Larson and Emilie Barenghien

Author(s): Autumn Larson, Emilie Barenghien, and Stacy Bennetts

Faculty Sponsor(s): Stacy Bennetts, PhD

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ABSTRACT

Salt marshes are a key foundation to the coastal ecosystems of Georgia. The marsh flora, mostly comprised of *Spartina alterniflora*, “smooth cordgrass”, provide vital resources for fishes, crustaceans, and birds with a variety of ecological and economic benefits. Healthy estuaries protect productive coastlines and support diverse ecosystems through the provision of migratory routes and nursery areas for many ecologically and economically important fish and invertebrate species. The Satilla Project investigates the water quality and biodiversity in the Satilla River estuary following the closures of logging cuts, which has changed water and sediment flow. Plant and fish species diversity and growth parameter data has been collected since June 2014 at all locations: Todd Creek (reference), Parsons Creek, Piney Bluff Node, and Noyes Cut. Students sample every other month in the marsh and river at four sites, one being a reference. A boat is taken out to each of the four sites, along with one that is dedicated to measuring water quality at several points in the river and soil samples. Each site-specific boat deploys a gill net, which sits along the riverbed from the edge of the marsh going out to the middle of the channel. While the net “soaks” for two hours, boats run a trawl for ten minutes and students conduct marsh transects. Transects are conducted by taking samples in a line using a standard sized hula hoop. Number of each species, along with species density and height are recorded per five quadrants. From gill nets and trawling, fish diversity and length are recorded/site. Along with valuable field experience for students, the data provides information on changes in marsh health as the estuarine cuts are closed. Pre-closure results indicated that *S. alterniflora* was significantly taller at Noyes Cut than the other sites, whereas Todd Creek had a denser *S. alterniflora* population. Parsons Creek contained the greatest species richness, including some succulents. Cuts have greatly impacted Piney Bluff Node, resulting in a monoculture of *S. alterniflora* that was significantly lower in both height and density compared to those in nearby Noyes Cut. These negative impacts were most likely due to the altered tidal flow created by cuts in the estuary. With recent closures to Dynamite and Old River Run Cuts, there have been some initial changes, such as *S. alterniflora* densities, among collection locations, which may suggest salinity and water flow have changed in a short period of time. The observed changes in these plant communities are expected as natural tidal flow returns to the estuarine system.

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Determining the Regulatory Properties of MAP1B in Axonal Morphology

Presenter: Elizabeth Laura

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Faculty Sponsor: Stephen Tymanskyj, PhD

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ABSTRACT

Neurons are the functional unit of the nervous system and are therefore important for electrical signaling in both afferent and efferent pathways. Microtubules play an integral role in neuronal function, in terms of intracellular transport, cytoskeletal structure, axonal growth, and synapse development. Microtubule-associated proteins (MAPS) can regulate microtubule properties and other associated proteins, such as actin, dynein, and kinesin. My protein of interest is MAP1B.

Knockdown or knockout of MAP1B *in vitro* results in an increase of neurites (axonal projections) in neurons. This is likely because MAP1B acts as an inhibitory regulator in terms of neuronal branching. MAP1B codes for a polypeptide with two discrete proteins, a heavy chain (HC) and a light chain (LC1), both having been found to have specific domains to bind actin (ABD) or microtubules (MBD). The heavy chain also contains a microtubule-assembly domain (MTA) that has been seen to support microtubule stabilization. The regulatory properties of these domains in terms of branching have yet to be seen. This poses the question of which domains-or specific combination of domains- regulate MAP1B's branching inhibition.

To address this question, primers were created to amplify and isolate individual domains, which were then cloned and tagged with fluorescent proteins. These were then expressed in neuroglioblastomas (Neuro-2A line) and cortical neurons. They were labeled with markers for neuronal morphology and visualized with confocal microscopy.

The next steps are to combine these findings with a CRISPR knockdown of MAP1B to elucidate the mechanisms of the domains by reintroducing them independently, as well as in stepwise combination, to determine interactions between domains. Moving forward, I am also looking to determine the effects of phosphorylation on the protein and its associated domains using nulls and mimics to increase or discourage branching.

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DOPTC: A Low-Cost Water Quality Monitoring System

Presenter(s): Mason Leavins, Nelson Lau

Author(s): Mason Leavins, Emma Herndon, Nelson Lau, Victoria Makowski, Wes Byne, Oscar Flite, and Joseph Hauger

Faculty Sponsor(s): Joseph Hauger, PhD

Affiliation(s): Department of Physics and Biophysics, Department of Biological Sciences, Department of Chemistry and Biochemistry, City of Augusta Utilities Department

ABSTRACT

Water chemistry is an effective way to measure water system health, just as medical standards are used to gauge a human's health. However, water chemistry instruments are generally expensive and proprietary to the manufacturer, resulting in sparse deployments throughout a region, with use restricted to trained field technicians. This often leads to infrequent and geographically sparse data collection, resulting in a small portion of the system representing the whole. To increase system comprehension, a low-cost sensor suite, DOPTC, was developed that can be deployed for continuous monitoring in multiple configurations to accommodate various environmental conditions.

Last year, the developed prototype returned values within 0.7% of calibration standards versus the 4.9% average of commercial sensors and was integrated with an autonomous surface vehicle. The design methodology is based on a common set of processing, communication, and sensor technologies with environmentally differentiated configurations. Data is transmitted using Particle's cellular communication and viewed remotely on a dashboard. Atlas Scientific and DFRobot sensors are used due to their electronic hardware compatibility and robust design. Compared to commercial sensors, DOPTC is a fraction of the cost with a significant increase in customization and deployment options.

Over the past year, DOPTC was deployed several times and collected data remotely, with the housing, component selection, circuit design, and code being improved with each prototype. Data is reported in real-time to a dashboard accessible to anyone with the link, no matter where they are in the world. These improvements have elevated the overall ease of use and the longevity of a deployed sensor, with additional sensors being added to increase the capabilities of DOPTC.

Additional DOPTC deployments throughout a single water system reduce reliance on manual data collection, in turn reducing human risk and improving consistency of readings at any time of day, regardless of weather. The reported data can be used to remotely identify the presence of pollutants or phytoplankton communities and weather events, substantially improving our understanding of the overall system. Future plans include additional deployments in Lake Olmstead, the Augusta Canal, and along the Satilla River.

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The Study of Chromosome Versus Hormone Effects in Salt-Sensitive Hypertension

Presenter(s): Jeffrey Lin

Author(s): Jeffrey Lin, Samuel D. Walton, Mark A. Vanden Avond, Mary Cherian-Shaw, Maya L. Mcfadden, Ayomikun Akin-David, Vincent R. Harley, Melinda R. Dwinell, Arthur P. Arnold, Aron M. Geurts, Justine M. Abais-Battad, David L. Mattson

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ABSTRACT

The Dahl Salt-Sensitive (SS) rat is a widely used model of hypertension that mimics key features of human salt-sensitivity. Previous studies demonstrated that male SS have exaggerated high salt (HS)-induced hypertension and renal damage compared to females. These sex differences may result from the combined effects of gonadal hormones and sex chromosome composition; however, the individual contributions to the SS phenotype remain unclear. The sex-determining region Y (SRY) gene encodes for the SRY protein, and it has been determined that the *Sry1*, *Sry4a*, and *Sry3c* genes in rats are essential for inducing male gonadal development and sex hormone production. To investigate the roles of sex chromosomes and the male gonadal hormones in SS hypertension, a transgene containing these Sry genes was inserted into an autosome of the Dahl SS rat. This approach generated three experimental groups: XY males, XX females, and XX^{SryTg} gonadal males (n=8-10/group). We hypothesized that this model can differentiate and separate the roles of hormones and sex chromosome dosage in the hypertensive phenotypes of the SS rat. At 10 weeks of age, rats were placed on an 8.0% NaCl HS diet for two weeks. Blood pressure, body weight, kidney weight, heart weight, and kidney histopathology were recorded and analyzed. At 7 weeks of age, XX females exhibited significantly lower body weight than XY males and XX^{SryTg} gonadal males (XX=167.4±2.6g, XY=211.0±6.7g, XX^{SryTg}=211.3±4.7g). No differences in mean arterial pressure (MAP) were observed at baseline, but following two weeks of HS, XX females displayed slightly lower MAP than XY males and significantly lower MAP than XX^{SryTg} gonadal males (XX=151.0±3.2mmHg, XY=155.8±6.3mmHg, XX^{SryTg}=163.7±4.7mmHg). XX females also showed reduced kidney and heart weights compared to the XY males and XX^{SryTg} gonadal males (kidney: XX=1.1±0.1g, XY=1.69±0.2g, XX^{SryTg}=1.8±0.2g; heart: XX=1.0±0.03g, XY=1.3±0.1g, XX^{SryTg}=1.3±0.1g). Medullary protein casting as well as CD3⁺ T-cell staining, as indices of kidney damage and inflammation, were also reduced in XX females when compared to the XY males and XX^{SryTg} gonadal males (% protein cast: XX=1.6±0.6, XY=4.2±0.5, XX^{SryTg}=4.0±0.5; % CD3⁺ staining: XX=0.15±0.02, XY=0.19±0.03, XX^{SryTg}=0.23±0.02). Overall, these results indicate that male gonadal hormones importantly contribute to elevated blood pressure, body and organ size, renal injury, and renal inflammation, and play a greater role than sex chromosome dosage in promoting salt-induced hypertension and renal end-organ damage in the Dahl SS rat.

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Exploring the Efficacy of the Dental Therapy Model on Dental Deserts

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Sponsor(s): Lee Anna Maynard, PhD

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ABSTRACT

Dental therapists are highly skilled healthcare professionals whose role falls between that of a dentist, who has received a doctorate in dental medicine or surgery, and a dental hygienist, who has either an associate or bachelor's degree in dental hygiene. They practice exclusively in areas with severe shortages of dental care providers. As a result, state legislators have welcomed them with open arms. In clinics, however, they are not commonly found despite there being millions of Americans who live in areas with patient-to-dentist ratios that exceed five thousand to one.

This lack of access to oral health care leads to critical dental disease, worsened overall health, and higher costs for patients. Fortunately, the implementation of dental therapists has been proven to increase the use of preventative oral health care, increase profits for dental practices, and improve workplace efficiency. In my analysis of the implementation of dental therapists, I draw a comparison to mid-level providers in traditional healthcare environments to better illustrate their role in the workplace. Moreover, I include data on dental therapists' clinical competency to demonstrate the feasibility of their role. I also include surveys conducted on dentists and dental hygienists to gauge their attitudes towards dental therapists and their compatibility with the existing dental workforce. The objective of my research is to highlight the effectiveness of the dental therapy care model in addressing regional disparities in dental care.

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Investigating the Subcellular Localization of the Axon Survival Protein NMNAT-2

Presenter(s): Cassidy Martin

Author(s): Cassidy Martin and Stephen Tymanskyj, PhD

Faculty Sponsor(s): Stephen Tymanskyj, PhD

Affiliation(s): Augusta University, Department of Biological Sciences

ABSTRACT

Neurons are highly specialized cells with long axons that rely on coordinated intracellular transport and protein regulation to maintain structural integrity and survive cellular stress. When neurons become injured, such as in spinal cord injury or neurodegenerative disorders, axonal transport becomes disrupted, contributing to neuronal degeneration and death. Nicotinamide mononucleotide adenylyltransferase-2 (NMNAT-2) is an axon survival protein that prevents axon degeneration by supporting cellular metabolism. NMNAT-2 functions as an enzyme that catalyzes the conversion of nicotinamide mononucleotide (NMN), a metabolic intermediate generated during NAD⁺ recycling pathways, into nicotinamide adenine dinucleotide (NAD⁺), a coenzyme required for energy production and neuronal viability. Although NMNAT-2 is known to be essential for axon maintenance, the mechanisms that regulate its intracellular transport and localization within neurons, especially during injury, are unclear. Understanding where NMNAT-2 localizes within neurons provides important context for its role in axon maintenance and neuronal survival. This study examines where NMNAT-2 localizes inside neurons and which subcellular components it associates with.

Fluorescently tagged NMNAT-2 constructs are used to visualize protein distribution relative to organelle markers using live-cell confocal microscopy. Neurons are co-transfected with NMNAT-2 constructs and fluorescent markers that label intracellular structures, such as lysosomes and mitochondria. Colocalization analysis is then performed to quantify the degree of spatial overlap between NMNAT-2 and these organelles, allowing evaluation of potential associations with specific intracellular compartments.

In addition to examining the localization of full-length NMNAT-2, domain-specific NMNAT-2 constructs are generated to investigate how individual regions of the protein contribute to subcellular targeting. Comparing localization patterns among constructs containing different NMNAT-2 domains allows evaluation of whether specific regions of the protein influence its association with lysosomes, mitochondria, or other intracellular structures.

It is predicted that NMNAT-2 will exhibit measurable colocalization with organelles involved in intracellular trafficking and metabolic regulation, and that altering particular domains of the protein may modify these localization patterns. By combining colocalization analysis with investigation of domain-specific NMNAT-2 constructs, this study offers insight into how NMNAT-2 localization within neurons relates to axon maintenance and neuronal survival.

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Sounds Align: Phonological Placement Effects on Lexical and Sub-Lexical Processing

Presenter: Angelina Martinez

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Faculty Sponsor(s): Dr. Sara Guediche

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ABSTRACT

Verbal communication is one of the most important means of human interaction for sharing ideas in society. Through this process, the brain relies heavily on the ability to accurately recognize spoken words. However, most real-world environments are noisy, making listening more challenging for the average person. As a result, the brain must rely on partial sound cues to activate and identify words. The process becomes more complex when words share similar sounds (phonological overlap), as multiple word candidates may be activated simultaneously, either early in the word (onset overlap) or later in the word (offset overlap). Since spoken words unfold over time, the position of phonological overlap between words, either at the beginning (onset) or end (offset) in the word, may differentially affect the timing and outcome of lexical processing. The goal of this study is to examine differences in how the position of phonological overlap affects word and sound recognition, measured by accuracy and speed. Participants will hear a prime-target stimulus pair and make either a lexical or a phonological decision on the target stimulus. The target will be preceded by a prime that either shares onset or offset sounds or is unrelated. Half the participants will hear a clear target (Group 1), and half will hear a target that has overlapping noise on the overlapping segment (Group 2). Thus far, data from 26 participants have been collected in Group 1. For the response times, the results show a significant main effect of phonological overlap. Follow-up t-tests show that the offset condition resulted in significantly faster response time compared to the unrelated and onset conditions, replicating prior work. These findings may reflect the fact that offset overlap serves as a useful cue to support recognition once earlier lexical competitors have been narrowed down. Data collection for Group 2 is still underway. Based on the data, we predict that the facilitatory effect of offset overlap may be due to pre-activation of the correct sounds and may therefore be exaggerated in the noise condition where the correct sound in the target is less clear. Overall, this study clarifies how the position of phonological similarity among words influences spoken word recognition, as a function of a listening environment.

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Patients' and Physicians' Perspectives on Polycystic Ovary Syndrome Treatment

Presenter(s): Alexis Mazique¹

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ABSTRACT

Polycystic Ovary Syndrome (PCOS) is a common yet underdiagnosed endocrine disorder affecting approximately 8–13% of women of reproductive age worldwide. Characterized by hyperandrogenism, anovulation, and polycystic ovarian morphology, and is associated with reproductive, metabolic, and psychological complications that negatively impact quality of life. Despite its prevalence and long-term health implications, there is no standardized treatment protocol for PCOS, and current management strategies vary considerably across clinical practice. Existing treatment approaches—including lifestyle modification, hormonal contraceptives, antiandrogen therapy, insulin-sensitizing agents, and nutritional supplementation—often demonstrate variable effectiveness and may not adequately address patient-reported outcomes.

The purpose of this study is to evaluate the effectiveness of commonly recommended treatments for PCOS by examining both healthcare providers' prescribing practices and patients' lived experiences. This study utilizes a two-phase design consisting of a comprehensive review of the literature followed by the distribution via social media of distinct, anonymous structured surveys to healthcare providers and to patients with PCOS. Survey questions assess treatment utilization, perceived effectiveness, symptom management, and gaps between clinical recommendations and patient outcomes. Thirty-two completed patient surveys have been analyzed so far; no health care providers completed a survey and possible reasons for this outcome will be discussed.

Findings from the patient surveys indicate that oral contraceptives are the most commonly prescribed and utilized treatment, reported by the majority of patient respondents. Although many patients perceived oral contraceptives as effective for short-term symptom control, only 52% of patients recalled discussion of alternative or complementary therapies such as exercise and dietary changes. A substantial proportion of respondents reported persistent symptoms, including fatigue, weight management difficulty, anxiety, and mood disturbances despite ongoing treatment. These results suggest that current management may emphasize symptom regulation while insufficiently addressing long-term physical and psychological well-being.

Overall, variability in treatment approaches and inconsistencies between provider recommendations (from literature and as reported by patients) and patient experiences highlight the need for improved communication and more individualized, patient-centered PCOS care.

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Altered Gene Expression in Senescent Periodontal Ligament Cells

Presenter(s): Diana Mejia

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ABSTRACT

Periodontal disease affects older individuals, yet the mechanism underlying its connection to age-related bone loss remains unclear. This study investigated whether cellular senescence (known to play a key role in biological processes) alters the expression of bone regulatory genes in periodontal ligament (PDL) cells and whether it is a potential mechanism for periodontal bone deterioration. Human PDL cells were cultured to develop non-senescent (passage 6) and senescent (passage 13) groups. In the experimentation process, RNA was isolated using the phase separation method and then purified via alcohol precipitation. Reverse transcription was performed to convert RNA into cDNA, which was then used for PCR analysis to assess the expression of bone regulatory genes Osteoprotegerin (OPG), a negative regulator of osteoclast induction; Receptor Activator of Nuclear Factor Ligand (RANKL), an inducer of osteoclastic bone resorption; and Sclerostin (SOST), a Wnt inhibitor and negative regulator of bone formation. A comparison of band intensities using Student's t-test was conducted to determine whether a significant difference exists between senescent and non-senescent cells. Expression levels of bone regulatory genes were normalized to GAPDH, a reference gene. Data normality for each regulatory gene was assessed using the Shapiro-Wilk test and QQ plots. Analysis revealed a significant elevation of SOST expression in senescent PDL cells ($p = 0.0173$) with mean \pm standard deviation values of 0.0602 ± 0.0377 for control and 0.448 ± 0.259 for senescence. RANKL expression showed a $p = 0.58075$, well above the threshold for a significant difference, along with values of 0.538 ± 0.313 for control and 0.684 ± 0.281 for senescence. OPG expression approached significance with $p = 0.0525$ and values of 0.740 ± 0.0878 for control and 1.03 ± 0.158 for senescence. Analysis of the RANKL/OPG ratio remained stable ($p = 0.963$), with averages of 0.715 (control) and 0.700 (senescence). We found that cellular senescence induced the expression of SOST, a bone negative regulator, in PDL cells, which may play a role in age-associated periodontal bone loss.

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Women's Health: The Lack of Trust & Information

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ABSTRACT

Chronic reproductive health conditions such as Polycystic Ovarian Syndrome (PCOS) and endometriosis still significantly need more studies on the conditions despite their widespread prevalence and long-term impacts on women's health. Existing research suggests that the lack of adequate medical attention towards these conditions contributes not only to physical and mental challenges, but also the social and emotional burdens of those affected by these conditions. This study explored the experiences of women diagnosed with PCOS and endometriosis, with particular focus on the patients' perceptions of their trust in their doctors. Because researchers needed to conduct more studies to develop effective treatments, women often endured many unsuccessful interventions. These women were also at risk for reduced fertility, painful periods, painful intercourse and much more. This research also helps understand the relationship of trust between these women and their doctors. With IRB approval data was collected through qualitative interviews and online content analysis. Interview participants included eleven women aged 18 and older who had received a diagnosis of PCOS or endometriosis. Participants completed a semi-structured interview regarding their condition, healthcare experiences, and levels of trust in medical professionals. Additionally, post from three Facebook support groups were analyzed. The groups consisted of a private endometriosis page, public endometriosis page, and a public PCOS page. Findings indicated that individuals with PCOS and endometriosis were generally dissatisfied and concerned about their quality of medical care. Themes identified from both interviews and online support group posts aligned with prior scholarship that emphasized systemic gaps in women's reproductive healthcare and their mistrust in doctors. These results highlight the urgent need for improved medical awareness, stronger patient-provider communication, and expanded research addressing the lived experiences of women managing chronic reproductive health conditions.

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Aquatic Macroinvertebrates and Their Correlation to Water Quality

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ABSTRACT

Macroinvertebrates are biological indicators of water quality due to their varied tolerance to stressors. Different species found in an environment will indicate changes in environmental conditions, including pollution. This study evaluates the health of the ecosystem at Reed Creek, located in Augusta, Georgia, by comparing the present macroinvertebrates to the local community composition throughout the seasons. Two sampling sites along the same stream were selected to represent differing infrastructures surrounding the creek. Site 1 was located under a highway and behind a fast-food restaurant, while Site 2 was located within a residential area with higher vegetation and reduced traffic.

Sampling was done seasonally during spring, summer, and fall using a combination of Hester-Dendy traps for long term data and D-nets for immediate data collection. Then organisms were classified into sensitive, somewhat sensitive, and tolerant taxa using Georgia Adopt-A-Stream guidelines. Additionally, data was collected on water quality, including dissolved oxygen, nitrogen, and phosphorus levels in the creek to identify if there was sufficient, depleted, or excess nutrient levels. This was combined with pH, turbidity, temperature, and overall observations of water clarity, color, and odor of the creek at the time of data collection. Together, the data was used to support the macroinvertebrates found and provide a comprehensive assessment of ecosystem health.

Results indicated that nutrient levels at both sites remained stable throughout the seasons. Dissolved oxygen levels were sufficient to support aquatic life but not excessive indicating low levels of pollution. Expected seasonal variation was seen during the study with changes to temperature and water flow. Turbidity values remained low, other than a temporary change at Site 1 during summer following storm runoff. Additionally, sensitive macroinvertebrate taxa were present at both sites in all seasons, supporting the conclusion that Reed Creek can sustain a healthy aquatic ecosystem. However, Site 2 consistently demonstrated greater biodiversity and a higher abundance of sensitive taxa compared to Site 1. These differences in the sites are likely due to their surroundings. Site 2 had additional vegetation and reduced urban influences enhancing habitat quality.

Overall, both sites were classified as healthy, with no immediate need for restoration. This study establishes a baseline for long-term ecological monitoring and highlights the importance of monitoring both biological and chemical within local ecosystems to evaluate the impact of human influences.

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Targeting the BTK-IDO Axis Enhances Oncolytic HSV in Glioblastoma

Presenter(s): Kelli Mitchell

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Faculty Sponsor(s): Konstantina Kyritsi, PhD, Dr. Bangxing Hong, PhD

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ABSTRACT

Glioblastomas are malignant and extreme tumors, due to the random mutation on how promptly it can propagate. Glioblastomas or GBMs are in the central nervous system (CNS), which includes our brain, spinal cord, and neurons. GBMs have demonstrated a limited responsiveness to different immunotherapies, which is attributable to a profoundly immunosuppressive tumor microenvironment (TME). An inhibitor that disrupts signals in the TME, is Bruton tyrosine kinase (BTK). BTK is a member of B-cell signaling, which influences the regulation for oncogenic signaling and is present in myeloid and neuronal cells, with a low survival rate. BTK's inhibitor Ibrutinib, is a potent inhibitor that has clinical satisfactory results with Indoleamine 2,3-dioxygenase (IDO) axis to display enhancement of an anti-tumor immune response. However, patients with GBM are difficult to treat due to the uniqueness of the tumor. GBM has the highest probability of spreading throughout healthy tissue in the brain. Certain markers like oncolytic viruses (OVs), overcome barriers of tumor destruction and have the outcome of priming tumor apoptosis (Jackson et al., 2025). A vector is crucial to target cancer cells in response to the inhibition of tumor growth. oHSV or known as oncolytic herpes simplex virus one illustrates effectiveness against tumor cells. Our hypothesis highlights the use of combination therapy with a BTK inhibitor and oHSV will increase tumor lysis in GBM12 and GBM28 human cells *in vitro* and *vivo*. Furthermore, the combination of oHSV, BTK inhibitor, and IDO inhibitor with increase anti-tumor response in 005 model. Different concentrations of BTK inhibitor Ibrutinib (2.5, 5, 10, 12.5 ug/mL) were monitored in a time lapse for 48 hours in human glioblastoma cells. In the human intracranial GBM12 model in *vivo* (005 model) mice, the mice had to undergo an intratumoral (i.u.) and an intraperitoneal (i.p.) injection of Ibrutinib. Immunofluorescence staining aided in expression with tumor cell death, oHSV infection, and macrophage infiltration. Apoptotic areas increased in the combination of both oHSV and BTKi (BTK-Inhibitor). However, in human GBM28 cells, adding ibrutinib (2.5ug/mL) 16 hours after oHSV infection, does not show any clinical significance on how well the virus spreads in GBM28 tumor cells, which proves BTK inhibition does not interfere with established viral infection. Our data proved significantly with the combination of oHSV and BTK inhibitor after 16-hour post-infection treatment, establishing tumor lysis activity.

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Crossmodal Semantic Effects on Visual Word Recognition

Presenter(s): Sireen Mohammed

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ABSTRACT

The significance of this research lies in finding the manipulations that will provide the most assistance in visual word recognition to improve reading skill as it is a near-necessity. The goal of this experiment is to help determine the effect of a predictable sentence context on visual word recognition, depending on the modality (visual vs. auditory) of the context. Past research has indicated that predictable sentence contexts facilitate word recognition and are especially beneficial when visual words are less intelligible. However, readers who need the most contextual support may benefit more from a context that is provided through spoken language (Clark, Guediche, Lallier, 2022). Clark et al. (2022) showed that an auditory context can mitigate the negative effects of crowding on visual word recognition. Nevertheless, little is known about the differences between the neural systems supporting visual vs. auditory sentence context effects on visual word recognition. The current study uses functional magnetic resonance imaging (fMRI) to investigate this question. To this end, we manipulate the modality of the sentence context (auditory/visual), the predictability of the sentence context (predictable/neutral), and the intelligibility of the word target (crowded/uncrowded), in a lexical decision task. Participants will either hear sentences through MRI-compatible headphones or read them on a screen. These will be followed by a final sentence target, which will either be a real word or a nonword. Participants will be asked to make a lexical decision by pressing a button if they believe the target is a real word and another if it is a nonword. Accuracy and reaction time measures will be collected. Sentence predictability was determined through an online experiment using a sentence rating task, in one group of participants. In addition, quantitative measures of predictability were obtained using a sentence completion task. We used these results to select the sentences for the fMRI study. In our discussion of this study, we will describe the full fMRI protocol and progress on data collection.

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Preparation of Nutraceutical Nanoparticles Using Facilitated Self-Assembling Technology (FAST) for Next-Generation Nutritional Supplements

Presenter(s): Amani Mouna, Angelena Jacob

Author(s): Amani Mouna, Angelena Jacob, Stephen Hsu

Faculty Sponsor(s): Stephen Hsu, PhD

Affiliation(s): Department of Biological Sciences

ABSTRACT

Many nutraceutical compounds, including curcumin, resveratrol, lycopene, lutein, and coenzyme Q10, are widely recognized for their antioxidant and anti-inflammatory benefits. These compounds are commonly used in dietary supplements and functional foods. However, many of them do not dissolve well in water and are not easily absorbed by the body, which limits their effectiveness. Traditional approaches to improve their solubility often involve chemical modification, fat-based encapsulation, or the use of surfactants and organic solvents. These methods can increase manufacturing complexity and cost and may raise regulatory concerns for food applications.

In this research project, we explored a new approach called Facilitated Self-Assembling Technology (FAST) to produce nutraceutical nanoparticles using food-compatible materials. FAST allows certain molecules to spontaneously organize into nanoparticles, when placed in a suitable environment. These nanoparticles can disperse evenly in water and may improve the stability and usability of nutraceutical compounds.

In this study, the FAST process produced stable nanoparticles with a negative surface charge that helped prevent them from clumping together in solution. Particle size distribution and stability were evaluated, including testing under simulated gastric conditions that mimic the environment of the human stomach.

To examine safety, we conducted XTT cell viability assays to determine whether the nanoparticles affected cell survival. The results showed that the nanoparticles were biocompatible and did not reduce cell viability compared with untreated control samples. In addition, fluorescence imaging using Cy5-labeled EC16 hybrid nanoparticles showed that the nanoparticles could interact with cell surfaces without causing observable toxicity.

Overall, the FAST platform offers a promising and environmentally friendly strategy for producing nutraceutical nanoparticles. Compared with traditional approaches, FAST avoids the use of harsh chemicals, requires less energy during production, and uses materials that are compatible with food safety standards. This technology may provide new opportunities for developing next-generation nutritional supplements and functional beverages with improved stability and potential effectiveness.

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Mechanosensitive Proteolytic Cleavage Requirements in Notch Signal Transduction

Presenter(s): Navya Pampatwar

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ABSTRACT

Notch signaling is an evolutionarily conserved juxtacrine pathway essential for cell fate specification, differentiation, and tissue homeostasis across metazoans. In most organisms, Notch receptor activation requires a mechanical pulling force generated by Epsin-mediated endocytosis of its ligand, which induces a conformational change that exposes the S2 proteolytic cleavage site within the receptor's Negative Regulatory Region (NRR). Subsequent cleavage by ADAM10 and γ -secretase releases the Notch intracellular domain (NICD), which translocate to the nucleus and regulates transcription of target genes. Aberrant Notch signaling has been implicated in multiple diseases, including T-cell acute lymphoblastic leukemia and neurodevelopmental disorders. In this study, the fundamental mechanisms of canonical Notch activation are investigated while examining structural variation in Notch receptors across species. Prior work from our laboratory revealed that Notch signaling in *Caenorhabditis elegans* occurs through a noncanonical mechanism that bypasses the requirement for ligand endocytosis. This difference arises from the absence of a conserved leucine "plug" within the NRR that protects the cleavage site, resulting in a lowered mechanical force threshold for receptor activation. To assess how specific NRR features influence mechanosensitive cleavage, the goal is to analyze Notch receptors from eight species exhibiting distinct NRR architectures. Using comparative structural analyses, NRR domains from *lin12* fly, *flylin12*, *stichopus japonicus*, *strongylocentrotus purpurulus*, *octopus binacuolides*, *priapulid*, and *playyhelminth capitella* were selected because of their distinct plugs or S2 cleavage regions. The study aims to demonstrate that subtle sequence variations, particularly in the leucine plug and S2 cleavage site, can significantly alter the mechanical requirements for Notch activation, highlighting the evolutionary plasticity of Notch signaling and offering new insight into ADAM protease recognition of mechanosensitive substrates.

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Development of an Automated Manual Dexterity Assessment Instrument

Presenter(s): Jagraj Parmar

Author(s): Jagraj Parmar, Wesley Cooke, Joseph Hauger, Bao-Ling Adam, William Jordan

Faculty Sponsor(s): Joseph Hauger, PhD, Bao-Ling Adam, PhD and William Jordan, MD

Affiliation(s): Department of Physics and Biophysics, Department of Surgery

ABSTRACT

Manual dexterity is a critical competency in many occupations, particularly in medicine where such skills directly impact procedural performance. Despite the importance of fine motor skills, many medical schools and residency programs do not have objective methods to measure or train such skills. One type of manual dexterity assessment currently used requires the test subject to use surgical tweezers to place 2.5 cm long metal pins into one hundred small vertical holes spaced 1 cm apart and arranged in a square. The amount of time required for the subject to place successive groups of twenty pins and the total time required to place all one hundred pins is measured by a trained observer using a standard stopwatch. This method has demonstrated problematic inter-rater reliability and is labor intensive. To address these challenges, we have developed an automated device that uses a 100 g capacity load cell interfaced to an HX711 amplifier and ATMEGA328P microcontroller to detect the placement of each pin. By measuring the total weight of the inserted pins, the exact number is determined. The microcontroller is programmed to obtain the pin count every 0.2 seconds throughout the duration of the test. Using an internal clock, the microcontroller measures and records the elapsed time for each successive group of twenty inserted pins. These times are recorded to an SD card file for analysis. The test subject is guided by information provided on a liquid crystal display and indicator lights. The enclosure, perforated board, weighing plate, and internal circuit mounts are all 3D printed using standard filament. A custom printed circuit board is used to connect all electronic components. This automated manual dexterity assessment instrument provides objective measurements, allows the test subject to conduct self-assessment, and eliminates timing variations among staff observers.

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Exploring the Glutamatergic Interaction Between Astrocytes and Neurons: Implications for Alcohol Use Disorder Pathophysiology

Presenter(s): Bansari Patel

Author(s): Bansari Patel, Dr. Seungwoo Kang

Faculty Sponsor(s): Dr. Seungwoo Kang, Dr. Georgios Kallifatidis, and Dr. Nadia Jilani-Hyler

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ABSTRACT

Alcohol withdrawal is frequently associated with hyperalgesia, a heightened sensitivity to pain that increases vulnerability to relapse. Prior research shows that glutamate imbalance, especially in the amygdala, may contribute to this problem, and astrocytes play a key role in clearing excess glutamate through GLT1. This academic background helped guide the focus of the study. The purpose of this project is to better understand how reduced expression of the astrocytic glutamate transporter GLT1 affects neuronal activity in the basolateral amygdala (BLA) during alcohol withdrawal. To investigate this, an astrocyte-specific GLT1 conditional knockdown mouse model (GLT1^{ΔF/F} × GFAP-Cre) was used. GLT1 expression was reduced using a Cre-loxP genetic approach. Immunofluorescence staining, Western blotting, and c-Fos immunohistochemistry were performed to measure changes in neuronal activation. Brain images were collected using a Zeiss Imager, and c-Fos-positive neurons in the BLA were quantified using ZEN Blue software. Group comparisons were analyzed with unpaired two-tailed t-tests. The results showed that mice with reduced astrocytic GLT1 had significantly higher levels of neuronal activation during withdrawal. GLT1 knockdown mice displayed nearly double the number of highly c-Fos-positive neurons compared to control animals. These findings indicate that even a partial reduction in GLT1 disrupts normal glutamate balance, increases BLA excitability, and may contribute to higher pain sensitivity during withdrawal. Overall, this study highlights the importance of astrocytic GLT1 in regulating amygdala activity and suggests that restoring GLT1 function could help reduce withdrawal-related pain and relapse risk in alcohol use disorder.

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Cre-LoxP System for Genetic Manipulation in Retinal Neurons

Presenter(s): Harsh Patel

Author(s): Harsh Patel, Shriya Tailor, Lotem Kol, Ebenezer Quainoo, and Lin Gan

Faculty Sponsor(s): Lin Gan, PhD

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ABSTRACT

The Cre-LoxP system is a genetic manipulation tool that recombines flanked DNA sequences. In this work, the application of the Cre-LoxP system was studied in retinal neurons. Using a gene editing tool, a DNA sequence called Cre was inserted downstream of the *Lhx4* promoter. This insertion allowed the *Lhx4* promoter to initiate the transcription of the Cre sequence. The sequence produced a modified estrogen receptor that was fused to a Cre recombinase protein. When induced by a drug, tamoxifen, Cre recombinase detaches and translocates into the nucleus to recombine flanked DNA sequences. The mice with this genetic modification were crossed with another mouse line that produced an EYFP protein, a type of enhanced yellow fluorescent protein; this cross generated mice with both the Cre and EYFP sequences. The EYFP protein helped visualize the activity of the Cre recombinase. In this study, mouse retinal cells were examined to monitor the specificity and expression of the Cre sequence at the time of tamoxifen exposure. Negative controls lacked either the Cre sequence, the administration of tamoxifen, the EYFP sequence, or any combination of the three. The absence of the Cre sequence, the tamoxifen treatment, and the EYFP sequence meant the inability to produce the Cre recombinase protein, the prevention of Cre recombinase from entering the nucleus, and the inability to observe Cre recombinase activity, respectively. Following tamoxifen exposure, retinal tissues were then collected, sectioned, stained, and analyzed. Preliminary results indicated the presence of the EYFP protein in retinal cells of experimental mice groups with all three factors. On the other hand, negative control samples lacked EYFP expression. This meant the presence of all 3 factors was needed to successfully monitor the specific expression of Cre under the *Lhx4* promoter. Future studies could apply the Cre-LoxP system to trace the expression of genes during retinal development.

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Role of LRRK2 in Regulating Axonal Growth and Transport

Presenter(s): Jenny Patel

Author(s): Jenny Patel, Stephen Tymanskyj

Faculty Sponsor(s): Stephen Tymanskyj, PhD

Affiliation(s): Augusta University, Department of Biological Science

ABSTRACT

Neurons are the fundamental units of the nervous system; they are specialized cells that transmit electrical and chemical signals throughout the brain and body to coordinate processes ranging from movement to thought. The nervous system consists of two divisions: the central nervous system (CNS)— composed of the brain and spinal cord— and the peripheral nervous system (PNS)— composed of the nerves throughout the body. Parkinson's Disease (PD) is a progressive neurodegenerative disorder affecting movement. Common symptoms of PD include tremor, rigidity, and bradykinesia (slow movement). It primarily impacts the CNS by causing degeneration of dopaminergic neurons in the substantia nigra. Dopaminergic neurons are specialized brain cells located in a small region of the brain that synthesize and release dopamine. In addition to electrical signals, neurons utilize axonal transport. Axonal transport is an essential process responsible for moving cellular organelles such as, vesicles, from the soma to the axon terminals. Kinesin and Dynein are the motor proteins responsible for maintaining axonal terminals for transport of cargos. Kinesin is responsible for anterograde transport, while dynein is responsible for retrograde transport. Leucine-rich repeat kinase 2 (LRRK2) is a multifunctional gene that encodes dardarin. Dardarin is a large, multidomain enzyme with kinase and GTPase activity. LRRK2 regulates intracellular pathways involved in mitochondrial homeostasis, vesicular transport, autophagy, and axonal growth and transport. LRRK2 is a major genetic contributor to PD, with mutations found in both familial and sporadic cases. LRRK2 is composed of 2,526 amino acids arranged into seven domains that regulate enzymatic activity and protein–protein interactions, many of which are linked to PD. LRRK2 has been notably associated with the regulation of axonal growth and transport. Mutations in LRRK2 increases kinase activity, leading to hyperphosphorylation of Rab GTPases, which are key regulators of intracellular trafficking and transport. This raises the question that mutations in LRRK2 impair axonal growth and transport by altering kinase activity and disrupting intracellular trafficking pathways. To analyze this question, cellular and molecular techniques will be used to establish LRRK2 constructs by creating primers targeting specific domains. Consequently, LRRK2 DNA is tagged with GFP, which is a green, fluorescent tag—and three of the red, fluorescent tags LAMP (lysosomal marker), mitochondrial marker, and BDNF (brain-derived neurotropic factor) to assess effects of intracellular trafficking and axonal transport. Ultimately, my intention for this project is to quantify axonal transport using live-cell imaging and assessing how LRRK2 mutations affect vesicular trafficking along microtubules.

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Alternatively Spliced VEGF-A Isoforms Differentially Regulate Ischemic Endothelial Genetic Programs

Presenter(s): Kripa Patel

Author(s): Kripa Patel

Faculty Sponsor(s): Vijay Ganta, PhD

Affiliation(s): Department of Biological Sciences, Department of Vascular Biology Center

ABSTRACT

The VEGF-A isoforms are a group of protein isoforms thought to have implications in angiogenesis, the growth of new blood vessels. However, these isoforms have yet to be studied intensively. As we learn more about the interactions of these isoforms in our cells, we will be better able to understand their regulators, receptors, and targets. If proven to act as effective therapeutic agents, these isoforms have a variety of implications, including uses in treating circulatory diseases like peripheral artery disease (PAD), which affects hundreds of millions worldwide. In the experiment, HUVEC (human umbilical vein endothelial) cells were treated with V165a, V165b, and VEGF-A_x recombinant proteins at equimolar concentrations under stimulated hypoxia and serum starvation (HSS) conditions meant to emulate the pathological conditions in the vasculature of a patient with peripheral artery disease. Untreated HUVECs served as controls in these experiments. A total of 4 samples were used and tested against each isoform treatment. RT-qPCR was performed to measure differences in the gene expressions of PRSS2, A2M, IFIT2, and APCDD1 for the VEGF-A isoforms against control. These genes were already thought to be affected by changes to the VEGF-A protein, either via upregulation or downregulation. Endogenous loading controls HPRT and GAPDH were also used for comparison. The results of the study indicated that the RNA expression did not match the differences observed in the previously studied RNA-Seq datasets. This indicates that further validation with increased sample size 'n' or time course experiments are needed to confirm and validate the RNA-Seq data. The current gene expression changes using qPCR did not recapitulate existing RNA-Seq data, indicating the necessity to check additional genes to identify the targets of these VEGF-A isoforms. Even with all that we know about these biomolecules, there has been no success in translating the benefits of VEGF-A isoforms to human patients. Once validated, the isoforms can be used to produce therapies for individuals suffering from peripheral artery disease.

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Seasonal Shifts in Wildlife Activity Within Shallow Riparian Corridors

Presenter(s): Sahil Patel and Sasha Hammarlund

Author(s): Sahil Patel, Sasha Hammarlund, and Robert Cromer

Faculty Sponsor(s): Robert Cromer, PhD

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ABSTRACT

Riparian corridors are areas of vegetation adjacent to streams, rivers, and other water bodies and can be considered interfaces between two different ecosystems. In modified landscapes, riparian corridors can function as corridors that facilitate the movement of wildlife among fragmented habitats. Riparian corridors have a significant role in facilitating the movement of wildlife in developed landscapes; however, little is known about the extent of the contribution of shallow stream systems to the seasonal movement of wildlife. Small stream systems and drainage culverts are common in developed landscapes and often parallel roads and residential corridors, yet little is known about the ecological significance of these riparian features in facilitating the movement of wildlife. Trail cameras have been used in various ecological surveys to determine wildlife activity patterns as well as to detect the presence of terrestrial vertebrates with minimal disruption to the surrounding environment. The cameras, which remain operational through motion-activated capabilities, can be used effectively to survey wildlife presence with minimal human intervention. In this study, motion-activated trail cameras installed in shallow riparian corridors as well as associated culverts in the Augusta metropolitan region will be used to survey wildlife activity during the late winter to early spring season. The cameras will be installed in areas with potential for wildlife movement across riparian corridors and culverts, including areas traversing streams as well as areas that may provide connections between adjacent green spaces. Initial survey results, conducted over a two-week period during late winter, indicate independent wildlife detections in an environment with minimal baseline activity. While it is expected that wildlife activity will be reduced during the winter months, initial results suggest that these shallow riparian systems support wildlife activity during this period. The study will continue into early spring, with further surveys conducted to determine changes in wildlife detection frequency as temperatures rise and vegetation growth increases. This study will provide baseline information on wildlife activity in shallow riparian systems, as well as further information on how these systems function in human-altered landscapes.

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Preparation of Stable Tau Oligomers

Presenter(s): Saniya Patel

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Faculty Sponsor(s): Stephen Tymanskyj, PhD

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ABSTRACT

Tau is a microtubule-associated protein that is essential in maintaining neuronal structure, axonal stability, and intracellular transport. Mutations in tau are associated with a wide variety of neurodevelopmental and neurodegenerative conditions, collectively referred to as tauopathies, including Alzheimer's disease. Alzheimer's disease is a progressive neurodegenerative condition marked by cognitive decline, memory loss, and neuronal loss, and is pathologically linked to the buildup of abnormal tau aggregates within neurons. In these conditions, tau forms oligomers and larger aggregates that are toxic to neurons and eventually lead to cell death. Curiously, these tau oligomers can migrate from one neuron to another, facilitating the spread across the brain. How a protein from inside a diseased neuron is able to be excreted, enter a healthy neuron, and subsequently cause aggregation remains unclear. Progress in comprehending these mechanisms has been limited by challenges associated with generating purified, functional tau suitable for controlled studies. To address this limitation, a protein purification protocol was optimized that allows for the isolation of purified, functional tau protein. This was achieved by generating a recombinant expression from a bacterial expression vector followed by an affinity-based protein isolation. The purified tau proteins were then seeded onto healthy neuronal cultures to model tau uptake and propagation. After three days in culture, the neurons were fixed and examined to measure the extent of tau uptake into the recipient neurons, allowing further assessment of early events in tau transmission. Further experiments will apply the same methodology using tau containing mutations identified in human patients to determine whether these mutations increase or decrease the rate of tau uptake into neurons. This work establishes a system to study tau propagation between neurons and assesses how disease-associated tau variants contribute to the progression of Alzheimer's disease and related tauopathies. Grasping the processes behind tau transmissions is crucial for elucidating the advancement of Alzheimer's disease and associated tauopathies. In the end, these findings could aid in creating treatment approaches focused on halting the spread of tau pathology, reducing the advancement of neurodegenerative diseases, and might aid in pinpointing essential molecular processes related to tau propagation.

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Oligodendrocyte Histone Acetylation in Preterm White Matter

Presenter(s): Simran Patki

Author(s): Simran Patki, Hailey Netherton, and Evan Goldstein

Faculty Sponsor(s): Evan Goldstein, PhD

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ABSTRACT

Premature birth is a significant healthcare challenge that affects millions of families. Many infants born prematurely have not had a chance to fully develop. Preterm infants with underdeveloped lungs are often exposed to a hypoxic environment due to a lack of oxygen reaching their brain. This can cause diffuse white matter injury (DWMI) in which the development of white matter in the brain is negatively impacted. In the central nervous system, oligodendrocytes (OLs), members of the oligodendrocyte lineage cells (OLCs) are responsible for constructing myelin, an integral component of white matter. Myelin forms a sheath around the axons of neurons, giving them a protective layer and aiding in the speed and propagation of action potentials. Myelination primarily occurs postnatally and is vulnerable to the negative effects of hypoxia. Chromatin remodeling is heavily involved in OL maturation. In oligodendrogenesis, oligodendrocyte progenitor cells (OPCs) terminally differentiate into pre myelinating OLs which mature into myelinating OLs. From the literature, it is seen that an increase in acetylation is correlated with OL differentiation and maturation. As a result, the acetylation of histone 3 lysine 9 (H3K9) influences OL development, and therefore, myelination. The results from our preclinical mouse studies of DWMI suggest that histone modifications have a role in disease pathogenesis. This model also showed that in preterm infants, there is an increase in H3K9 acetylation, an accumulation of pre- myelinating OLs, and a delay in OL maturation. This research intends to determine whether the abnormal acetylation of H3K9 found in the preclinical mouse model is also found in preterm infants. Postmortem human brain samples from the Childrens' National Hospital (Washington, DC) were examined. Antibodies that were distinct to OL and H3K9 markers were used and ImageJ software was used to quantify and assess the acetylation of H3K9 in OLs. Data analysis must be done to understand the relationship of H3K9 acetylation in preterm infants and in the preclinical mouse trials. We hypothesize that preterm infants will have increased H3K9 acetylation in OLs. This research has the potential to influence therapeutic strategies for preterm birth survivors who have suffered from DWMI.

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The Impact of Virtual Reality on Affect-Regulated Exercise

Presenters(s): Adam Porter

Authors(s): Andrew Moore, PhD and Adam Porter

Faculty(s): Andrew Moore, PhD

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ABSTRACT

In exercise, external stimuli can be used to reduce feelings of discomfort by shifting attentional focus. In previous studies with music, it has been shown that people select a higher exercise intensity when instructed to maintain a work rate that felt “good” to them. This purpose of this study was to determine if this benefit of higher selected work rate was present when utilizing a virtual reality headset to provide visual stimulus during affect-regulated exercise. In a repeated-measures crossover design, healthy young adults ($N = 27$; 18 women/9 men; $M_{\text{age}} = 19.9 \pm 1.0$ years, $M_{\text{bodyfat}} = 23.0 \pm 8.8\%$) completed two 20-min exercise sessions on a cycle ergometer. Intensity was self-adjusted to a Feeling Scale value of +3 (“good”) on a scale of -5 to +5. A VR condition in which a first-person viewpoint video of a bike ride in natural scenery was displayed, and a Control condition with only a physical laboratory setting visible, were completed in a randomized and counterbalanced order separated by 1 week. Selected work rate was assessed objectively (average power output and heart rate) and subjectively (rating of perceived exertion; RPE) every 5 min. Additionally, enjoyment was assessed after each session with the Physical Activity Enjoyment Scale (PACES). Repeated-measures analyses were completed as appropriate using SPSS, version 30 ($\alpha = 0.05$). The results of this study showed that there was no significant difference between VR and Control for power output (77.17 ± 30.53 vs 76.65 ± 27.99 W), heart rate (134.37 ± 24.49 vs. 135.21 ± 25.09 bpm), or RPE (3.24 ± 1.25 vs. 3.06 ± 1.21), and no differences at matched time points ($p > 0.05$ for all). There was a significant difference in the PACES scores between the conditions, with VR being higher (107.51 ± 14.10 vs. 94.07 ± 18.38 ; $p \leq 0.001$, $d = 0.73$). This showed that VR could increase the enjoyment of the exercise while maintaining a similar objective and subjective work rate to the control condition, which could have implications for exercise adherence.

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Tamoxifen Attenuates Retinal Neurovascular Injury in Diabetic Retinopathy

Presenter(s): Avneesh Prabakar

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Faculty Sponsor(s): Modesto A. Rojas, PhD

Affiliation(s): Pharmacology and Toxicology, Vascular Biology Center, Vision Discovery Institute. Augusta University, Augusta Georgia.

ABSTRACT

In previous studies, we have shown that retinal neurovascular injury is mediated by the buildup of cholesterol esters, promoting chronic inflammation via surrounding microglia. A key enzyme is acyl-coenzyme A: cholesterol transferase-1 (ACAT-1), which converts free cholesterol to cholesterol esters. In diabetic models, ACAT-1 activity is upregulated leading to increased cholesterol ester buildup and neurovascular injury. Here, we investigate tamoxifen as a novel inhibitor of cholesterol ester accumulation and its effect on ACAT-1 activity in diabetic retinopathy. Wild-type mice were separated into four injection groups: vehicle (corn oil), streptozotocin (STZ), STZ/Tamoxifen, and Tamoxifen only. At 5 weeks post-birth, STZ/Tamoxifen and Tamoxifen groups received daily intraperitoneal injections of Tamoxifen (100 mg/kg) consecutively for five days. At 9 weeks post-birth, STZ and STZ/Tamoxifen groups received daily intraperitoneal injections of Streptozotocin (60 mg/kg) consecutively for five days. Urine was tested to ensure mice in these groups became diabetic. At 16 weeks, mice were subjected to optomotor response (OMR) and electroretinogram (ERG) testing to assess visual function. At 18 weeks, retinas were collected and processed for cholesterol ester formation (filipin staining) and Western blot. Plasma was collected to assess plasma cholesterol ester levels. Mice with STZ-induced diabetes (STZ only) exhibited significantly higher levels of retinal cholesterol esters ($p < 0.05$) compared to control. However, mice treated with Tamoxifen and STZ showed a significant decrease in retinal cholesterol ester buildup compared to STZ-only mice. Furthermore, mice treated with tamoxifen and STZ had a significant decrease in plasma cholesterol esters compared to STZ-only mice. Tamoxifen preserved visual functions in diabetic mice as compared with vehicle group ($p < 0.05$). Treatment of STZ-induced diabetic mice with Tamoxifen exhibited significant decreases in cholesterol ester accumulation and rescue from RNV phenotypes. However, cholesterol levels did not significantly fluctuate between the groups. For future experiments, it is worth investigating the specific mechanism of Tamoxifen in decreasing cholesterol ester formation in relation to ACAT-1.

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Temporal Prevalence of *kdr* in *Aedes albopictus*, South Richmond County

Presenter(s): Erin Przywara

Author(s): Erin Przywara, Anthony Jones, and JT Wicker

Faculty Sponsor(s): Jennifer Baltzegar, PhD

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ABSTRACT

Knockdown resistance (*kdr*) is a genetic mechanism that reduces mosquito susceptibility to pyrethroid-based insecticides, which are commonly used in vector control programs targeting mosquitoes, specifically *Aedes albopictus*. Pyrethroid resistance poses a significant challenge to controlling mosquito populations, as these insecticides are a primary tool in managing disease vectors and preventing outbreaks of arboviral diseases such as dengue chikungunya, and Zika, which impact human health. Resistance arises from genetic mutations in the voltage-gated sodium channel, which prevent pyrethroids from effectively disrupting the mosquito's nerve function, thus allowing resistant mosquitoes to survive insecticide exposure that would normally be lethal. Understanding the prevalence of *kdr* in local mosquito populations is crucial for anticipating insecticide failure and optimizing future vector control efforts. This ongoing study aims to assess and characterize the prevalence of knockdown resistance (*kdr*) across multiple seasons in *Aedes albopictus* populations from South Richmond County. Specifically, it seeks to identify temporal patterns of pyrethroid resistance that may threaten the local mosquito control efforts and public health outcomes. Adult mosquitoes are collected from multiple sites across South Richmond County using standardized trapping methods. The collected specimens are identified as *Aedes albopictus* and processed for molecular analysis. Allele-specific PCR screening of isolated DNA is used to detect *kdr*-associated mutations. Genetic variants linked to pyrethroid resistance are determined based on the melting temperature profiles of amplified DNA fragments generated during melt curve analysis. This information can help vector control programs protect the efficacy of pyrethroid insecticides. Additionally, the baseline data collected will serve as a reference for future surveillance, allowing public health officials to monitor the emergence and ongoing spread of insecticide resistance. Ultimately, these results are expected to support evidence-based strategies that reduce arboviral disease transmission, improve local vector control decision-making, and enhance the overall effectiveness of ongoing mosquito management initiatives in South Richmond County.

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Analytical Geometric Approach to Minkowski Space Time Diagrams

Presenter(s): Guluna Qazi

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Faculty Sponsor(s): Asanka Amarasinghe, PhD

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ABSTRACT

Minkowski diagrams are visual representations developed by Hermann Minkowski to describe motion in Einstein's special theory of relativity. They allow us to represent changes in position and motion in space and time within a single picture. In these diagrams, different observers, moving at constant speeds relative to one another, use different coordinate systems. However, unlike ordinary graphs used in basic geometry, the axes for a moving observer are not simply tilted; they are also scaled differently. This difference in scaling comes from the way space and time are connected in special relativity. The quantity that remains constant between observers, known as the space-time interval, follows rules that differ from those of standard plane geometry. As a result, although Minkowski diagrams look like familiar flat (Euclidean) graphs, they do not fully obey the usual rules of planar geometry. This creates difficulties when we try to apply straightforward geometric calculations to describe motion consistently across different frames of reference. In this project, we introduce a mathematical projection that maps the coordinate system of a moving observer onto that of a stationary observer within the same Minkowski diagram. The purpose of this projection is to adjust the scaling between the two coordinate systems so that standard geometric methods can be used without conflict. Importantly, the method preserves the space-time interval, ensuring that the core physical principle of special relativity remains intact. Using this projection approach, we show that key results of special relativity—moving clocks “tick” slower and moving objects are observed to be shorter—can be derived using planar geometric methods. This method avoids the need for more advanced mathematical tools often associated with hyperbolic geometry. The project is still ongoing. We are currently generalizing this projection method to be used under all conditions in special relativity using standard analytical geometry. As such, the introduction, background, and overall findings thus far will be included in the poster. A central question guiding our work is whether this projection between frames naturally arises from the idea that all coordinate systems in a Minkowski diagram share a common origin.

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Comparative Analysis of Amyloid- β Pathology in 5xFAD Mice Using Antibodies

Presenter(s): Sriya Rajanala

Author(s): Sriya Rajanala and Yun Lei

Faculty Sponsor(s): Yun Lei, PhD; Xin-Yun Lu, MD, PhD

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ABSTRACT

Alzheimer's disease (AD) is a neurodegenerative disorder characterized by progressive cognitive decline and two major pathological hallmarks in the brain: the deposition of β -amyloid (A β) peptides in extracellular senile plaques and the presence of tau filaments in intracellular neurofibrillary tangles. A β is produced through the sequential cleavages of the amyloid precursor protein (APP) by β -secretase and γ -secretase. γ -Secretase cleaves APP at multiple sites, generating A β peptides with heterogeneous C-termini, among which A β 40 and A β 42 are the predominant isoforms found in AD brain tissue. Notably, A β 42 is more hydrophobic and fibrillogenic, and is therefore the principal species deposited in amyloid plaques. Monomeric A β can aggregate into soluble oligomers and insoluble fibrils, contributing to disease progression. Because A β exists in multiple isoforms and aggregation states, the use of antibodies with distinct epitopes and conformational specificities is necessary for accurate assessment of A β pathology. In this study, I examined the A β pathology in 5xFAD mice, an established AD mouse model expressing human APP and PSEN1 transgenes with five familial AD-linked mutations, at different ages (3, 6, and 12 months). Mice were transcardially perfused under anesthesia through the ascending aorta using phosphate buffer, followed by 4% paraformaldehyde. After perfusion, I dissected, cryoprotected, and cut the brains into 40- μ m coronal sections. I processed the free-floating sections for immunostaining using several antibodies with distinct specificities: the 6E10 antibody recognizing all forms of A β (including APP), the MOAB-2 antibody specifically recognizing A β (without cross-reacting with APP), and the GT622 antibody selectively recognizing A β 42. I also used Methoxy-X04 fluorescent dye that binds to fibrillar A β plaques to better visualize the pathology. After staining, I imaged the brain sections using the KEYENCE all-in-one fluorescence microscope. I quantitatively analyzed A β plaque load in the entorhinal cortex, hippocampus, and prefrontal cortex using ImageJ/Fiji software. In this study, a comparative framework for antibody and dye-based detection of A β pathology was established and accurately evaluated the changes in A β plaque burden across disease stages in 5xFAD mice.

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Stress, Resilience, Distress Tolerance and College Adjustment

Presenter(s): Autumn Richards

Author(s): Autumn Richards and Sabina Widner

Faculty Sponsor(s): Sabina Widner, PhD

Affiliation(s): Department of Psychological Sciences

ABSTRACT

College is an exciting and stressful time in most students' lives with personal, social, and academic challenges. A student's ability to adjust well to college relates to the likelihood they will graduate and meet other professional goals. This study examined how resilience, distress tolerance, and perceived stress correlated with college adjustment. This study hypothesized that college adjustment would be positively correlated with resilience and distress tolerance and college adjustment would be negatively correlated with perceived stress. This study also tested whether resilience or distress tolerance would moderate, or buffer, the relationship between perceived stress and college adjustment. Participants consisted of 109 undergraduate students from Augusta University who completed measures of college adjustment, resilience, distress tolerance, and perceived stress. Results indicated that higher college adjustment was associated with higher levels of resilience, higher levels of distress tolerance, and lower levels of perceived stress. Neither resilience nor distress tolerance were moderators of the relationship between perceived stress and college adjustment. Based on these results, exploratory analysis was conducted to determine if mediation could better explain how distress tolerance or resilience may contribute to the relationship between perceived stress and college adjustment. Mediation analysis results indicated significant indirect effects for both distress tolerance and resilience as mediators in the relationship between perceived stress and college adjustment. These results suggest that perceived stress may lead to changes in resilience and distress tolerance levels which may, in turn, lead to changes in college adjustment. The direct effects between perceived stress and college adjustment remained significant without the mediators in the model, suggesting the importance of knowing students' stress levels in understanding college adjustment scores. These results highlight the importance of stress management for college students and demonstrate the need for student-focused, college-based initiatives for education on stress management.

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Comparative Analysis of Normal and Diseased Human Skin

Presenter(s): Megan Rowland

Author(s): Megan Rowland and Dr. Soma Mukhopadhyay

Faculty Sponsor(s): Dr. Soma Mukhopadhyay

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ABSTRACT

Human skin is a structurally complex and continuously renewing barrier whose integrity depends on coordinated regulation of proliferation, immune signaling, and dermal–epidermal adhesion. The purpose of this systematic study is to synthesize histopathological and molecular evidence comparing normal human skin with carcinoma, psoriasis, and epidermolysis bullosa (EB) to gain insight about shared and distinct mechanisms of epithelial instability and to evaluate the progression in techniques for detection, and treatments for these clinical conditions. We hypothesize that despite differing etiologic drivers (oncogenic, inflammatory, and genetic), these disorders converge on predictable structural and regulatory failures that compromise epithelial stability. Identifying these convergent mechanisms strengthens diagnostic precision and informs the development of targeted, mechanism-based treatments. A comprehensive review of peer-reviewed literature, dermatopathology atlases, and validated digital histology repositories was conducted. Fifteen documented cases per disease were comparatively analyzed to ensure consistent structural and molecular evaluation. Normal skin architecture was characterized by emphasis on epidermal stratification, basement membrane integrity, and adhesion complexes, including collagen IV, laminin-332, and integrins. Disease states were then systematically examined. Carcinoma demonstrates dysregulated cell-cycle control and aberrant proliferative signaling, resulting in loss of polarity and invasive growth. Psoriasis reflects immune-mediated keratinocyte hyperproliferation sustained by pro-inflammatory cytokine networks and altered differentiation programs. EB results from inherited mutations in structural adhesion proteins, producing mechanical fragility and dermal–epidermal separation. Comparative analysis revealed recurrent patterns of architectural disruption, adhesion failure, and signaling dysregulation across all three conditions. While the initiating molecular events differ, downstream instability of epithelial organization represents a shared pathological principle. Correlating histological findings with molecular mechanisms directly informs therapeutic strategy; oncogenic pathway identification supports targeted inhibitors in carcinoma; cytokine profiling guides biologic therapies in psoriasis; and molecular localization of adhesion defects underpins emerging gene and protein replacement approaches in EB. This systematic study supports the hypothesis that diverse dermatologic disorders converge on common vulnerabilities in epithelial regulation. Integrating histopathology with molecular biology provides a translational framework which could be implemented in the diagnosis and contribute to improved dermatologic care.

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SmartGrass: Implementation of Automated Sensors for Continuous Grass Monitoring

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Faculty Sponsor(s): Genevieve Reeves, PhD

Affiliation(s): Department of Physics and Biophysics, City of Augusta, Utilities Department

ABSTRACT

Municipal landscaping is important for environmental sustainability and ecosystem health. However, rather than using a data-driven maintenance schedule, most municipalities rely on fixed, schedule-based mowing rotations. This schedule-based approach results in inefficient resource allocation, with some areas being over-maintained while others are neglected. Over-mowing stresses grass, reduces soil quality, increases weed proliferation, and contributes to unnecessary fuel consumption and labor costs. In contrast, under-maintained areas may become overgrown, with the possibility of increased safety hazards, and more intensive remediation. These challenges motivate the need for a smart, automated system that can provide real-time information about grass growth and support need-based maintenance strategies. Our SmartGrass sensor prototype employs a distance sensor interfaced to an Arduino microcontroller. Experiments have been conducted in outdoor testing locations on the Augusta Health Science Campus. During these experiments, the sensors are mounted at a fixed height, and a baseline measurement of grass height is recorded. After sufficient baseline data is acquired, the grass is manually cut to simulate routine mowing. Subsequent measurements are taken and compared to the pre-cut grass height. These results demonstrate reliable detection of changes in grass height. To date, we have experimented with two distinct sensors. The MaxBotix MB7092 uses a sonar signal and is capable of consistently detecting changes in grass height with centimeter-level accuracy, recording measurements of 76 cm before cutting and 88 cm after cutting. The less expensive VL53L1X sensor uses light detection and ranging (LiDAR) and is capable of measurements with millimeter-level accuracy, with baseline measurements of 38.5 cm and post-cut measurements of 44.9 cm. These results demonstrate the technical feasibility of the approach and validate SmartGrass's potential for practical deployment. In order to become a practical solution, our system will transition to a Particle Boron cellular-enabled microcontroller powered by a solar energy system. This will facilitate long-term autonomous outdoor data collection and allow measurements to be transmitted to a cloud-based platform for remote monitoring. In the long term, insight gained from this project will inform future sensor-based monitoring systems, including a planned application involving real-time water level monitoring within Augusta's sewer infrastructure to enable early identification and precise localization of potential issues such as blocked flow and flooding.

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Role of Polyamines in Retinopathy of Prematurity Complications

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ABSTRACT

Retinopathy of Prematurity (ROP) is a sight-threatening vascular disease that affects premature infants and remains a leading cause of childhood blindness worldwide. The disease is characterized by an initial phase of vasoobliteration followed by hypoxia-driven pathological angiogenesis, resulting in retinal neovascularization (RNV) and potential retinal detachment. Although current treatments target advanced stages of the disease, there remains a critical need to identify molecular pathways that drive early pathological vascular growth and can be therapeutically targeted. Emerging evidence suggests that dysregulation of the ornithine decarboxylase (ODC) and polyamine pathway may contribute to abnormal angiogenesis. ODC is the rate-limiting enzyme in polyamine synthesis, and elevated polyamine levels have been associated with enhanced endothelial proliferation and vascular remodeling. In this study, we investigated whether pharmacological inhibition of ODC using α -difluoromethylornithine (DFMO), an FDA-approved irreversible ODC inhibitor, could reduce pathological retinal neovascularization. To evaluate this, we utilized the oxygen-induced retinopathy (OIR) mouse model, a well-established preclinical model that recapitulates key features of human ROP. DFMO treatment was assessed at postnatal day 17, a time point corresponding to peak neovascularization. Morphometric analysis of retinal flat mounts stained with Isolectin B4 demonstrated a significant reduction in neovascular tuft area in DFMO-treated retinas compared to vehicle-treated controls ($p < 0.01$; $n = 9-11$ eyes per group). These findings suggest that ODC inhibition attenuates pathological retinal angiogenesis *in vivo*. To further explore the underlying cellular mechanisms, primary human retinal microvascular endothelial cells were subjected to hypoxia serum starvation (HSS) to mimic ischemic conditions observed in ROP. HSS significantly increased ODC expression and intracellular and extracellular polyamine levels ($p < 0.01$; $n = 4$). Importantly, DFMO treatment attenuated these hypoxia-induced changes, supporting a mechanistic link between ODC activation, polyamine accumulation, and endothelial dysfunction. This work is ongoing, and additional studies are being conducted to elucidate the downstream signaling pathways by which ODC-mediated polyamine synthesis promotes neovascularization. To strengthen causal evidence, we will also confirm our findings using endothelial cell-specific ODC knockout mice. Collectively, our preliminary findings suggest that targeting the ODC-polyamine axis may represent a novel and translational therapeutic strategy to reduce pathological neovascularization and improve long-term visual outcomes in premature infants with ROP.

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Preventing Adverse Maternal and Neonatal Outcomes in Adolescent Mothers

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ABSTRACT

Adolescent pregnancy remains a public health concern in the United States, with teenager mothers facing higher risks of preterm birth, low birth weight, longer NICU admissions, and maternal complications. Inadequate prenatal care contributes to these poor outcomes. Understanding which prenatal care strategies most effectively improve positive outcomes for adolescent mothers is essential for guiding nursing practice.

A comprehensive literature review was completed using PubMed and CINAHL Plus to identify studies between 2015 and 2025. Terms included were prenatal care, adolescent pregnancy, teen mothers, maternal outcomes, and neonatal outcomes. The search found 8,111 results; by removing 8,097 duplicates, 31 articles were screened by title and abstract. The studies included focused on implementing prenatal care strategies and outcomes of infants or mothers with inconsistent prenatal care. Twelve articles were reviewed, but only six met the full criteria and were included in this review.

Across six studies the literature continuously demonstrated early, continuous, comprehensive, adolescent-focused prenatal care improves maternal and neonatal outcomes. Four of the studies, three quantitative studies and a quality improvement report, indicated group and team-based models strengthened patient engagement, improved communication between providers and adolescents, and showed improved adherence to care plans. Multidisciplinary and teenage-specific care offered psychosocial support, infection screening, and improved maternal contraceptive postpartum and reduced rates of preterm births. In contrast, inadequate prenatal care was linked to higher rates of complications, NICU admissions, and lengthened hospital stays.

Early, consistent, and supportive approaches of prenatal care are essential for improving outcomes to promote maternal and neonatal health. Adolescent group prenatal care and multidisciplinary, teenage-specific teams stand out as effective approaches because they offer education, emotional support, and a sense of connection that traditional care can lack. Nurses play a key role in promoting access to care, building trust, and providing supportive, nonjudgmental care. Future research should identify which specific components of prenatal programs most effectively impact outcomes of adolescent pregnancies.

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The Impact of Blood Pressure Variability on Microglia Reactivity

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ABSTRACT

Blood pressure variability (BPV), characterized by large, repeated fluctuations in arterial pressure, is increasingly recognized as an independent risk factor for cognitive decline and dementia. This study aims to determine how chronic BPV influences neuroinflammatory responses in the brains of middle-aged male and female mice (12–14 months old). Neuroinflammation is a fundamental physiological process that supports pathogen clearance and tissue repair. In the central nervous system, these processes are primarily mediated by the resident immune cells called *microglia*. Under homeostatic conditions, microglia survey the parenchyma and release trophic and anti-inflammatory factors. In disease states, however, they can transition from a pro-survival, homeostatic phenotype to a pro-inflammatory, potentially neurotoxic state. Because chronic BPV is associated with low-grade systemic and central inflammation and often precedes the onset of hypertension, it is hypothesized that sustained BPV will promote a shift toward a pro-inflammatory microglial phenotype. To model BPV, mice will be implanted with a programmable infusion pump to receive pulsatile Angiotensin II (Ang II), a potent arteriolar vasoconstrictor, administered via six pulses daily (3.1 µg/hour every 3–4 hours) for 25–30 days. Previous work supports the presence of robust blood pressure fluctuations (from 100 to 180 mmHg) during infusion, but mice do not develop hypertension as their averaged mean arterial pressure did not exceed 130 mmHg. Over the course of 20–25 days, the pressure averaged 120 mmHg. Microglial structure will be assessed in CX3CR1-GFP reporter mice, in which microglia express green fluorescent protein, enabling high-resolution visualization of the cells' soma and processes. A quantitative skeleton analysis pipeline generated through ImageJ will be used to evaluate microglial density, branch complexity, and soma morphology. To further define microglial phenotype, brains will undergo immunohistochemical analysis for markers associated with anti-inflammatory or pro-inflammatory states. This work is ongoing, with analysis in progress. By characterizing how chronic BPV alters microglial structure and transition towards various activation states, this study seeks to identify a mechanistic link between vascular instability and neuroinflammatory processes relevant to cognitive decline.

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ECOD Assay Method Development in a Blackworm Model

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Faculty Sponsor(s): Faith Wiley, PhD

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ABSTRACT

Lumbriculus variegatus, commonly known as the California blackworm or mudworm, is a freshwater Annelid species. The objective of this study was to determine the suitability of blackworms as models for the ECOD assay. The ECOD assay is a method that measures the activity of specific detoxification enzymes (cytochrome P450s) by incubating specimens with the substrate 7-Ethoxycoumarin (7-EC) and measuring any resulting 7-Hydroxycoumarin (7-HC). While it has been performed in vertebrates and some invertebrates as a biomarker of contaminant exposure, blackworms have yet to be studied as a potential model. Initial tests were performed to determine appropriate concentrations of the substrate and measure the approximate difference in fluorescence between the substrate 7-EC and predicted product excreted 7-HC. Samples of both the reactant and product (alone and in combination) were measured to create standard curves of concentration and fluorescence. Then, several batches of five worms each were submerged in substrate 7-EC at varying concentrations for approximately 24 hours, after which the surrounding solutions were measured for fluorescence and compared to the standard curve. Several problems were encountered throughout these preliminary tests. To address inconsistent spikes of fluorescence due to floating waste originating from the worms' culture environment, a method was developed to "clean" the worms by transferring them to different containers of spring water each day, for 3-4 days before an experiment was performed. A second problem was the overlap in fluorescence spectra between 7-EC and 7-HC, making it difficult to measure 7-HC at the low concentrations excreted by the worms. A pH buffer of approximately 10.5 was added to the samples when they were transferred to the 96-well black plates. This increased the fluorescence of the 7-HC but was still not adequate to differentiate it from the substrate signal. As a solution, the most recent trials have incubated the worms in substrate for 30 min, followed by a series of washes to remove all 7-EC from test wells. Aliquots of the surrounding water were then measured for fluorescence at several time points between 15 min and 24 hours. Preliminary data suggests that 7-HC secretion is evident within 15 minutes of substrate removal and appears to plateau by 30 minutes, but further testing is needed. Future experiments will repeat these preliminary tests to check for consistency as well as develop a protein assay to standardize measurements to account for variability in worm size.

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Oligodendrocyte Histone Trimethylation in Preterm Infant White Matter

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Faculty Sponsor(s): Evan Goldstein, PhD

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ABSTRACT

Preterm birth continues to be a significant issue in healthcare, affecting up to 1.5% of all live births. Survivors of premature birth are often left with neurodevelopmental disabilities, including cognitive, psychiatric, and motor impairments that have profound impacts on quality of life. These issues arise as premature infants have underdeveloped respiratory tracts leading to poor respiratory function and inadequate blood oxygenation. Poor blood oxygenation exposes the brain to a state of hypoxia, which impairs white matter development. This is known as diffuse white matter injury (DWMI). In the central nervous system, there are oligodendrocyte lineage cells (OLCs) which give rise to oligodendrocytes (OLs), the myelin producing cells of the brain. OLs wrap myelin around axons, forming a protective layer that enables electrical impulses to travel at a faster rate. Myelination occurs primarily after birth, so this process is subject to disruption due to hypoxia. OL maturation involves chromatin remodeling, and the methylation status of histone 3-lysine 9 (H3K9) is important for OL development and formation of myelin. H3K9 trimethylation is a repressive mark that promotes OL differentiation and maturation. Therefore, the trimethylation of H3K9 influences OL development and myelination. Findings from our preclinical mouse model of DWMI suggest that histone modifications are implicated in disease pathogenesis. The aim of this research is to investigate whether the differential H3K9 trimethylation observed in the mouse model of DWMI is conserved in preterm infants. Postmortem human brain samples of preterm infants from the biobank of the Children's National Hospital (Washington, DC) were analyzed. Immunohistochemistry was used to label H3K9 trimethyl marks and OLIG2-expressing OLCs. H3K9 trimethylation in white matter OLCs was assessed utilizing the ImageJ image analysis software. Completing this study could validate the findings of the mouse model of DWMI and deepen understanding of preterm birth-related injuries and their pathogenesis. We hypothesize that the preterm infant samples will display an increase in H3K9 trimethylation in OLs. This research has the potential to inform therapeutic strategies for survivors of preterm birth.

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Design and Biological Evaluation of Curcumin-Derived Hybrids with Multi-Target Activity

Presenter(s): Sudhan Sivakumar

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Faculty Sponsor(s): Siva S. Panda, PhD

Affiliation(s): Department of Chemistry and Biochemistry, Augusta University

ABSTRACT

Curcumin, a naturally occurring polyphenolic compound derived from *Curcuma longa*, has long been recognized for its anti-inflammatory, antioxidant, antimicrobial, and anticancer properties. Despite its broad therapeutic potential, its clinical translation remains limited due to challenges such as low aqueous solubility, rapid metabolism, poor systemic stability, and minimal oral bioavailability. To overcome these barriers, this research explores two complementary medicinal chemistry strategies, molecular hybridization and amino acid conjugation, to develop next-generation curcumin derivatives with improved pharmacological profiles and reduced toxicity. The first part of this work focuses on curcumin hybrids that incorporate dichloroacetate, a metabolic modulator known to influence cancer cell glycolytic pathways. These hybrid molecules were synthesized using optimized coupling conditions to preserve their chemical integrity and were then evaluated in breast cancer models. In vitro assays with non-transformed mammary epithelial cells, estrogen-receptor-positive breast cancer cells, and triple-negative breast cancer cells showed that the hybrid derivatives selectively inhibit cancer cell proliferation at nanomolar concentrations while exhibiting minimal toxicity toward normal cells. A lead hybrid candidate was subsequently tested in a spontaneous mouse mammary tumor model, where oral administration significantly reduced primary tumor growth, decreased pulmonary metastases, and improved overall survival, without causing detectable liver, kidney, or hematological toxicity. Computational docking supported the proposed mechanism of action, demonstrating strong binding affinity to key cancer-associated kinase targets and predicting favorable ADME properties consistent with drug-like behavior. The second part of this research examines curcumin–amino acid conjugates designed to improve solubility, metabolic stability, and targeted biological activity. Variations in amino acid identity, stereochemistry, and protecting groups created a diverse library of conjugates with distinct functional properties. Many conjugates displayed robust anti-inflammatory activity in a carrageenan-induced rat paw edema model, with some outperforming established NSAIDs and demonstrating significantly reduced ulcerogenic liability. Additional assays revealed potent antimicrobial activity against Gram-positive and Gram-negative bacteria and notable analgesic activity in both peripheral and central pain models. Some deprotected derivatives also showed antiproliferative effects across lung, breast, and prostate cancer cell lines. These findings were supported by 2D-QSAR modeling, which identified key physicochemical descriptors that govern anti-inflammatory and antibacterial potency.

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A Green One-Pot Strategy for Constructing Bis-Benzothiazoles via Benzotriazole Activation

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ABSTRACT

Advancing sustainable synthetic methodologies is crucial for the progress of modern organic chemistry, particularly in the synthesis of heterocycles with diverse applications in pharmaceuticals and materials science. Benzothiazoles and their bifunctional derivatives constitute a significant class of bioactive scaffolds, recognized for their antimicrobial, anticancer, antiviral, and anti-inflammatory properties. Conventional methods for synthesizing these compounds often rely on harsh reaction conditions, metal catalysts, or environmentally hazardous reagents, limiting scalability and sustainability. This work presents a green, metal-free synthetic protocol that enables efficient one-pot access to bis-benzothiazole structures via benzotriazole-mediated activation of dicarboxylic acids. The strategy employs stable N-acylbenzotriazole intermediates that react synergistically with 2-aminothiophenol under mild, room-temperature conditions in acetic acid. This transformation delivers excellent yields across a diverse range of substrates, demonstrating operational simplicity, high atom economy, and compatibility with various aromatic, heteroaromatic, and aliphatic linkers. Acetic acid serves a dual role as both solvent and Brønsted acid promoter, enhancing electrophilicity, stabilizing key intermediates, and facilitating dehydration during ring closure. Optimization studies have shown its superiority over commonly employed solvents, bases, and microwave-assisted conditions, all of which yielded low or negligible results. To complement the experimental observations, density functional theory (DFT) calculations provided thermodynamic and mechanistic insights, showing that while overall reaction enthalpies approach thermoneutrality, the benzotriazole activation step ensures a consistently favorable pathway for cyclization. The data underscore the significance of substituent effects, indicating that planar aromatic linkers benefit from stabilizing π - π interactions, whereas heteroatom-rich linkers may induce destabilizing polarization effects. These findings align with the experimental performance trends and highlight the mechanistic advantages of employing benzotriazole as an activating group. This study introduces a sustainable and broadly applicable methodology for the synthesis of bis-benzothiazoles that eliminates the need for metal catalysts and hazardous reagents. The combination of mild reaction conditions, high efficiency, and clear mechanistic insights demonstrates the potential of benzotriazole-based activation strategies as valuable tools in green heterocyclic synthesis, with significant prospects for future applications in medicinal chemistry, materials science, and environmentally responsible practices.

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The Role of Map7D1 in Neuron Development and Intracellular Transport

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ABSTRACT

The brain consists mostly of cells called neurons, which assist in communication between the brain and rest of the body. Microtubules contribute to the neural circuits formed by these neurons by affecting neuron morphology, intracellular transport, and synapse development. Microtubules are also known to be regulated by microtubule-associated proteins (MAPs), particularly in stabilization, organization, and interactions with motor proteins such as kinesin and dynein. Disruption of microtubule regulation can impair neuronal development and has been linked to neurodegenerative diseases. A unique microtubule-associated protein, MAP7, has been shown to interact with both microtubules and the transport protein kinesin-1.

Structure-function analyses have shown that particular domains within MAP7 control the binding to microtubules and kinesin-1. Inclusion of the kinesin-1 binding domain allows for axon and branch growth, while other domains allow for branch formation. MAP7 has shown to prevent branch retraction with its ability to increase microtubule stability, as microtubules were more stable at branches where MAP7 was located. With MAP7's ability to bind kinesin-1 and microtubules, a novel mechanism may be possible to control microtubule-based transport during axon growth.

The focus of this study is an isoform of MAP7 called MAP7D1. Despite prior information and studies on MAP7, little is known about the mechanisms of MAP7D1. The primers were designed, PCR was run, and restriction cloning was performed to generate the individual domains of MAP7D1 which are equivalent to those identified in MAP7. These were then tagged with fluorescent markers for live cell imaging and visualization. The constructs were transfected into live neurons along with markers for tubulin to visualize microtubule localization and dynamics. In addition, fluorescent intracellular cargos such as lysosomes and mitochondria were co-expressed, and the movement frequency and duration between the different MAP7D1 domains were quantified. Kinesin-1 will also be co-tagged alongside the MAP7D1 domains to determine similarities and differences between MAP7D1 and MAP7, and to ultimately assess if MAP7D1 can interact or control protein transport through axons as well.

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Quantitative Analysis of MRI in Early Alzheimer's Disease Detection

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Faculty Sponsor(s): Dr. Christina Wilson

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ABSTRACT

Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by neuropathological alterations that precede overt cognitive impairment by years to decades. This temporal dissociation between biological onset and symptomatic expression necessitates the refinement of early diagnostic modalities capable of detecting subtle neuroanatomical and functional degeneration during preclinical and prodromal stages. Although positron emission tomography (PET) and cerebrospinal fluid (CSF) biomarkers have demonstrated molecular specificity, magnetic resonance imaging (MRI) offers a noninvasive and widely accessible platform for high-resolution structural and functional quantification. Despite its broad clinical availability, the cumulative diagnostic performance of MRI across imaging modalities and successive technological periods from 2010 through 2025 remains insufficiently quantified within a unified analytical framework.

A structured quantitative review of 127 peer-reviewed studies was conducted using a predefined Boolean search protocol. Studies were stratified by imaging modality, including structural MRI (sMRI), functional MRI (fMRI), diffusion tensor imaging (DTI), arterial spin labeling (ASL), and magnetic resonance spectroscopy (MRS). From each investigation, key diagnostic performance parameters were extracted, including overall classification accuracy and modality-specific measures of statistical dispersion.

Across all modalities, MRI revealed a weighted mean diagnostic accuracy of 91.9 percent with a standard deviation of 6.7 percent. Seventy-six percent of investigations provided full or partial statistical support for MRI as an early diagnostic instrument. fMRI exhibited the highest mean classification accuracy at 95.5 percent with comparatively low dispersion. Multimodal MRI frameworks demonstrated improved classification stability relative to single-modality approaches. Publication frequency increased by more than 700 percent between 2010 and 2025, corresponding with methodological advancements in machine learning (ML) implementation and quantitative imaging analytics.

The consistency and magnitude of aggregated diagnostic performance metrics substantiate MRI as a quantitatively robust and clinically scalable modality for early AD identification. Its demonstrated reproducibility across imaging techniques and study populations positions it as a foundational instrument for advancing preclinical detection frameworks. As methodological precision continues to evolve, MRI is positioned to play a central role in shaping future diagnostic paradigms, facilitating earlier intervention strategies and strengthening the evidentiary foundation for standardized neurodiagnostic integration.

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Analyzing the Effect of MAP7 D3 on Neuronal Morphology

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ABSTRACT

Neurons are specialized cells in the nervous system that transmit signals throughout the body and have long projections called axons that require strong internal support. Microtubules provide this support by maintaining neuronal shape and serving as tracks for transporting materials within the cell. Microtubule-associated proteins (MAPs), such as MAP7D3, are critical for stabilizing and organizing these microtubules, directly influencing neuron growth, branching, and connectivity. MAP7D3 activity ensures that neurons can efficiently transport materials along their axons and dendrites, supporting neuronal development and communication. Disruption of MAP7D3 can lead to altered neuron morphology and impaired connectivity.

Proteins like MAP7D3 are made up of distinct regions called domains, each with specific functions. The N, P, and C regions of MAP7B D3 were examined to determine how distinct structural domains contribute to microtubule regulation. The N region is known to mediate microtubule binding, whereas the P and C regions are suspected to regulate kinesin-1 activation and recruitment, therefore influencing microtubule-based transport. The goal of my study is to see how MAP7 D3 impacts neuronal morphology and determine how different domains of the protein contribute to neurite growth, function, and branching. To study the individual domains of MAP7 D3, specific DNA sequences were isolated, amplified, and confirmed. Cultured neurons were transfected with MAP7 D3 tagged with fluorescent proteins to visualize the neurons under confocal microscopy.

For future plans, my study will work on viewing the transfected neurons under confocal microscopy, continuing the development of tagging MAP7 D3 with GFP, another fluorescent protein, to allow for additional imaging. It will also continue to work on combining domain constructs to create an NP and PC domain to compare domain interactions and their effects on neuronal morphology. By understanding how MAP7D3 effects neuronal morphology, this research may provide insight into how neural circuits are formed and maintained. This research can help with understanding how MAP7 D3 contributes to neuron shape, organization, development, and maintaining proper function.

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The Effects of *Curcuma longa* (Turmeric) Derivatives on Pain-Pressure Threshold

Presenter(s): Lydia Utomwen

Author(s): Lydia Utomwen¹ and Dawn Langley-Brady²

Faculty Sponsor(s): Dawn Langley-Brady, PhD

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ABSTRACT

Approximately 60 million Americans live with chronic pain -- costing the U.S. economy 600 billion dollars annually. Adverse side effects and the risk of addiction are common characteristics of traditional pharmacological approaches to pain. Aromatherapy, the use of aromatic plant extracts, is a non-invasive therapy method for chronic pain. *Curcuma longa* (turmeric) is known for its pain-relieving and anti-inflammatory properties, and its chemical component curcumin has been widely studied. Holistically, turmeric has not been well-studied, nor have its derivatives been studied using Pain-Pressure Threshold (PPT). PPT is the minimum amount of force applied before the stimulus is uncomfortable to the participant. This study aimed to determine the effects of three turmeric derivatives (carbon dioxide extract [CO₂], essential oil [EO], and hydrosol) on post-PPT scores. The study utilized a one group pretest-posttest design. Healthy participants were recruited using convenience sampling and completed a series of eligibility questionnaires. The protocol included controlled lab conditions and participant study preparations, and incorporated five visits respectively: Skin control, placebo – jojoba, hydrosol, EO, and CO₂ extract. The latter two derivatives were diluted to 10% in jojoba for safety. Each visit consisted of a) a 15-minute rest/acclimation period; b) marking the midpoint of the right forearm; c) Pre-PPT testing; d) skin well placement and 20-minute substance absorption period; e) skin well removal and arm cleaning; and f) post-PPT testing. A hand-held Wagner Pain Algometer was used to quantify the levels of pressure experienced by the participants. Participants verbalized when the pressure sensation became uncomfortable and the measurement was recorded. The data was analyzed in SPSS using descriptive statistics and paired sample T-tests. The study sample was five participants (n = 5) whose demographics included: mean age = 21.40 ± 1.14 years; 60% male, 40% female; and 60% black, 40% white. There were no significant changes in post-PPT scores for the derivatives (p = 0.135 to 0.427). The placebo post-PPT scores were significant (p = 0.029). This difference may be caused by jojoba's anti-inflammatory properties. The non-significant findings may be attributed to our small sample size and participant outliers. The trend towards significance for skin control (p = 0.076) suggests potential habituation. Future studies should include larger and more diverse sample sizes.

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The Effects of Different Stathmin Isoforms on Microtubule Dynamics

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ABSTRACT

Every year, 5000 people are diagnosed with ALS (CDC, 2025). ALS is caused by the degeneration of upper and lower motor neurons, which causes muscle failure. This results in difficulty walking, slurred speech, and behavioral changes (Mayo Clinic, 2024). Some cases of ALS are passed down genetically, but the cause of most ALS cases is unknown. To address this, scientists are trying to understand the function of certain genes and proteins linked to ALS, such as Stathmin-2. In motor neurons, microtubules transport cargo. Stathmin-2 regulates microtubule expression and stabilizes them. The loss of the protein TDP-43 causes the mis-splicing of Stathmin-2, which destabilizes microtubules and hinders transportation. Since Stathmin-2 is not the only protein regulating microtubules, other isoforms were studied to understand their effect on microtubules and EB3 comet expression. The purpose of this study is to explore how different stathmin isoforms affect microtubule dynamics in live cells. This was accomplished by selecting three different stathmin isoforms: Stathmin-1, Stathmin-2.1, and Stathmin-2.2. Each isoform was replicated through PCR, separated by gel electrophoresis, extracted from the gel, and cloned so it could be tagged with GFP for identification. Then, neuronal glioblastoma and embryonic cortical neurons were transfected with the different stathmin isoforms along with markers for the cytoskeleton, such as actin, tubulin, and EB3, a fluorescent label for polymerizing microtubules. Live imaging for these cells was done over a period of two minutes for each isoform. The preliminary results show that there were fewer EB3 comets in Stathmin-2.2 compared to GFP. They also show that there were more EB3 comets in Stathmin-1 compared to GFP. Since images of Stathmin-2.1 appeared too bright, there is no conclusion on how Stathmin-2.1 affects microtubule expression. The number of EB3 comets in each movie from each isoform can be quantified, and values such as mean speed, maximum speed, track duration, and track distance can be measured. These findings will be used to fully understand how each isoform of stathmin affects microtubule expression and its overall role in ALS. Additionally, other stathmin isoforms can be analyzed, and those results can be compared with the results of this study. This study shows that different stathmin isoforms affect microtubule stability, growth, and expression. Current results show that Stathmin-1 promoted microtubule growth, producing more EB3 comets. However, Stathmin-2.2 decreased microtubule growth and produced fewer EB3 comets. Further experimentation with Stathmin-2.1 is underway.

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Fractional Quantum Hall Physics in Rapidly Rotating Fermions and Excitons

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Author(s): Brooke Vos, Theja DeSilva, and Asanka Amarasinghe

Faculty Sponsor(s): Theja DeSilva, PhD and Asanka Amarasinghe, PhD

Affiliation(s): Department of Physics and Biophysics

ABSTRACT

The Fractional Quantum Hall Effect is a striking manifestation of two-dimensional electron systems under the effect of high magnetic fields. The magnetic field, applied perpendicular to the electron system, will deflect the charged electrons perpendicular to their original path and perpendicular to the magnetic field, into what is known as the transverse direction. This effect emerges from the interplay of the force exerted on charged particles by magnetic and electric fields, known as the Lorentz force, quantum mechanics, and interactions among the system, giving rise to rich and exotic phenomena. Remarkably, the same physics can be realized with neutral atoms caught in a rapidly rotating trap. This is due to the Lorentz force being replaced by the Coriolis force, which is the force acting upon objects in motion within a rotating frame. Recent experiments have been conducted to find the momentum distribution of two interacting subatomic particles with a half-integer spin, known as fermions, in a system under the effects of the Quantum Hall Effect. This research investigates this momentum distribution theoretically and finds an excellent agreement between theory and experiment. Building on this foundation, the framework of study can be expanded to investigate fractional quantum Hall states of excitons in a bilayer. Excitons are a newfound particle discovered by Brown University, in which an excited electron leaves its original position, leaving behind a positively charged hole. When these two fractional charges are taken together, they are called excitons. This excitonic system opens new avenues for exploring strongly correlated phases. The results of this research provide theoretical insights into emergent quantum Hall physics in both ultracold atomic systems and excitonic platforms and highlight their potential relevance to future quantum technologies. As this study is continued, it will theorize experimental setups in which ultracold atomic systems could be used in the place of excitonic systems by using atomic bilayers rotating opposite to each other to simulate equivalent physics.

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Effects of Pollutant Presence on Southeastern Fish Communities

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Faculty Sponsor(s): Randal Signer, PhD

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ABSTRACT

Freshwater stream health is a critical driver of aquatic biodiversity and fish well-being, as declines in water quality can significantly increase physiological stress and disease susceptibility in fish populations across diverse freshwater ecosystems and regions. While many studies provide only a single snapshot of stream conditions, this project addresses the need for a long-term perspective by comparing historical water quality data with contemporary field samples to evaluate changes in stream systems in the CSRA. Because these nutrients are biologically significant and can influence fish health and disease prevalence, tracking their trends over time is essential for predicting emerging risks and assessing ecosystem stability. Framed within the One Health concept which means plant, animals and human health all affect one another, this study recognizes the interconnectedness of environmental, animal, and human health, emphasizing that freshwater quality impacts not only fish communities but also public health and regional sustainability. With this, it could inform local and national nonprofits to fight for better laws and improve CSRA predictive ecological modeling. The significance of this research is the gap in nutrient monitoring, limited fish disease data with water quality testing, and the potential to identify high risk river systems and prevent further ecosystem destabilization. Understanding CSRA nutrient trends with One Health framework and assessing ecosystem stability will help determine whether there is an urgent risk to fish populations in the CSRA region. By integrating historical datasets with modern ecological monitoring, this research aims to provide a more comprehensive understanding of stream health. Preliminary analyses suggest that an increase in pollutant concentrations over time will play a crucial role in physiological stress in fish, increased disease case rates, and a positive correlation with poor ecosystem stability. General data on fish disease case rates and preliminary survey analyses across multiple monitored stream sites will be included.

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Sugar Protection of Myoglobin Secondary Structure Following Protein Denaturation

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ABSTRACT

Protein preservation plays an important role in maintaining biological function during protein transport and storage, especially for proteins like myoglobin that depend on proper folding to remain functional. Loss of protein structure during denaturation can interfere with biological activity and limit experimental or practical use. Sugars have been shown to help preserve protein secondary structure by stabilizing the surrounding environment through reduced molecular motion and maintenance of the protein's folded state during denaturation. This study aims to examine how different sugars influence the stability of myoglobin's secondary structure when the protein is exposed to different denaturing conditions. Myoglobin serves as an ideal protein model for this study because it's well characterized and representative of proteins with primarily α -helical structure (approximately 75%). Changes in secondary structure will be monitored using Fourier-transform infrared (FTIR) spectroscopy through the changes in the lineshape of the amide absorption bands, which provide insight into the effect of sugar in protein preservation. To investigate sugar-based protection, myoglobin samples will be analyzed under controlled laboratory conditions designed to allow direct comparison between treatments. Baseline FTIR spectra will be collected prior to denaturation to establish reference structural profiles. Following this, the protein will be exposed to increased temperature or altered pH, two common sources of protein destabilization that reflect different types of structural stress. Different sugars will be introduced to protein samples to evaluate their ability to preserve secondary structure under each condition. FTIR spectra collected following denaturation will then be compared to baseline measurements to assess structural changes across treatments. This study investigates the ability of varied sugars to preserve myoglobin secondary structure during denaturation. Changes in α -helical content are anticipated to depend on both the denaturation method used and the presence of sugar. Heat- and pH-induced denaturation should affect myoglobin differently, leading to changes in spectral patterns. Comparing these outcomes will allow for evaluation of sugar effectiveness across multiple denaturation methods. Understanding how sugars protect protein structure under different stress conditions is relevant to improving protein handling and storage practices. This work will provide new insights into protein preservation and storage in areas like biomedicine, biotechnology and food science.

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Assessing Knockdown Resistance in Northern CSRA *Aedes albopictus* Mosquitoes

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Faculty Sponsor(s): Jennifer Baltzegar, PhD

Affiliation(s): Department of Biological Sciences

ABSTRACT

Insecticide resistance is a growing concern for mosquito control management, particularly in regions that heavily rely on chemical interventions. Knockdown resistance (*kdr*) describes mutations that make mosquitoes less affected by insecticides. As resistance increases, control programs may face greater difficulty managing mosquito populations using chemical treatments, and complicated public health responses may follow. Insecticides are still widely used and relied upon nationwide. Monitoring mosquito resistance is critical for supporting effective vector control and reducing the risk of disease transmission. Understanding where resistance is present and at what frequency is an important step in improving mosquito management strategies. This study aims to quantify the prevalence of *kdr* alleles in *Aedes albopictus* (*Ae. albopictus*) populations collected from the northern regions of the Central Savannah River Area (CSRA). *Ae. albopictus* was selected for this study due to its role as a vector for several pathogens and its widespread distribution throughout the CSRA. Mosquito samples are collected from sixteen established sites divided between the northern and southern regions of Richmond County. Focusing on the northern region allows for deeper examination of localized resistance patterns that may be influenced by regional control practices. Resistance status will be determined through a molecular analysis of individual mosquito samples. DNA will first be isolated and then examined using PCR-based methods with melt curve analysis to identify resistance genotypes. Genotype data will be grouped by collection site and region to support comparison of resistance prevalence between northern and southern areas. This study is expected to show variation in *kdr* prevalence across geographic regions, with possible differences in genotype distribution between northern and southern regions. These results should yield new insights into localized environmental pressures and clarify how resistance is distributed at a regional scale. This information may also help identify areas where resistance monitoring should be prioritized in future efforts. By identifying regional resistance patterns, this work can help guide targeted insecticide use and contribute to more effective vector control strategies in the CSRA.

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Invasion Potential and Gene Expression Changes in Nuclear Factor-KappaB Altered Macrophages in Triple-Negative Breast Cancer

Presenter(s): Kayla Wilder

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ABSTRACT

Triple-negative breast cancer (TNBC) is an invasive and aggressive form of breast cancer. The canonical nuclear factor-kappaB (NF- κ B) transcriptional pathway is often overexpressed in TNBC. Previous studies have shown that the chemokine CXCL10 had a significant increase in expression in bone marrow-derived macrophages (BMDM) from p65 knockout mice co-cultured with TNBC cells. To study the interplay between NF- κ B and other signaling molecules within the tumor microenvironment of TNBC, novel p65 knockout (KO) mice co-cultured with AT-3 TNBC cells, treated or untreated with CXCL10, were used in an invasion assay to assess invasion potential. Results showed that the addition of CXCL10 in AT-3 cells alone and in AT-3 cells co-cultured with control BMDMs led to higher migratory cell counts, while lower migratory cell counts were observed in AT-3 cells co-cultured with p65 KO BMDMs treated with CXCL10.

Verification of previous mouse-based research on the interplay between the NF- κ B signaling pathway, TNBC, and other target molecules was also performed using human-derived macrophages. To manipulate the NF- κ B pathway in a human system, a siRNA transfection was performed against the p65 transcription factor. The siRNA transfection was optimized, and RT-PCR analysis showed that one treatment condition (5 μ l siRNA for 24 hours) significantly reduced p65 mRNA. Lastly, qPCR analysis was used to determine whether CXCL10, CXCR3, and IL-1 β were altered in a similar manner as in our mouse system. IL-1 β levels were higher in p65 siRNA macrophages co-cultured with MDA-MB-231 TNBC cells compared to co-cultured control siRNA macrophages, which supported our mouse-based results. MDA-MB-231 TNBC cells, when co-cultured with SC macrophages that have been transfected with either control or p65 siRNA, show an increase in CXCL10 expression compared to the cancer cells alone. However, CXCR3 and CXCL10 expression showed a significant decrease in p65 siRNA macrophages co-cultured with MDA-MB-231 TNBC cells.

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Navigating Administrative Systems and the Black Student Experience

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Affiliation(s): Office of The Registrar

ABSTRACT

Universities rely on administrative systems to manage enrollment, academic records, and student progress; however, these systems also shape how students experience access, communication, and institutional support. This research examines how administrative processes, particularly within a university Registrar's Office, influence the academic navigation and experiences of Black undergraduate students at Augusta University. While administrative offices are often viewed as procedural units, their communication practices and policies play a critical role in student understanding, retention, and overall success.

This study was developed through my internship experience in Augusta University's Registrar's Office, where I observed how students interact with institutional policies, deadlines, and academic procedures. The purpose of this project is to explore how administrative communication affects students' ability to interpret requirements, resolve academic concerns, and feel supported within institutional systems. The central research question asks: how do administrative structures and communication practices impact the experiences of Black students navigating university processes? Using a qualitative and practice-informed approach, this research combines reflective institutional analysis, observations from professional internship engagement, and a review of existing scholarship on student success and higher education administration. Particular attention is given to communication clarity, accessibility of information, and the ways students interpret institutional messaging. Rather than focusing solely on student deficits, this project evaluates how institutional structures themselves may unintentionally create barriers to understanding or engagement.

Expected outcomes suggest that clearer communication strategies, culturally responsive outreach, and proactive administrative engagement can improve students' confidence in navigating university systems. Findings aim to demonstrate that administrative offices are not neutral spaces but active contributors to student success and belonging. By highlighting the relationship between administrative communication and student experience, this research encourages universities to reconsider how operational practices influence equity outcomes. This project contributes to interdisciplinary conversations about higher education, communication, and student development by positioning administrative services as key partners in promoting inclusive academic environments. Ultimately, the study advocates for intentional communication practices that strengthen transparency, accessibility, and institutional trust for all students.

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The Role of TikTok in the Palisades Fire Crisis Response

Presenter(s): Lilly Williamson

Author(s): Lilly Williamson, Dr. Carrie Reif-Stice

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ABSTRACT

The Palisades Fire ignited on January 7, 2025, in Los Angeles County. Burning more than 23,000 acres, the fire destroyed 6,837 structures and damaged an additional 973 (Rodriguez, 2025). With 12 confirmed fatalities, the Palisades fire is the third deadliest and most destructive wildfire in California's history (Rodriguez, 2025). During the crisis, images and videos circulated on social media, providing real-time information on the unfolding fire and impacted areas. Specifically, videos on TikTok served as a lens into the crisis and the community's response in the aftermath. Hashtags, such as #palisadesfire, #palisades, and #fires, allowed individuals to share mandatory evacuation orders, safety routes, and personal accounts of the wildfire's impact. TikTok became a space for citizens to perceive risks and make calculated decisions regarding response efficacy. Although prior research has explored the growing importance of social media and crisis response (e.g., Anthony et al., 2019), TikTok remains an understudied platform.

Guided by the Extended Parallel Processing Model (Witte, 1992), this study uses the Risk Behavioral Diagnostic Scale (RBD) (Witte, 1996) to assess individuals' perception of threat and efficacy during the Palisades Fire. As defined by the RBD, perceived efficacy reflects individual beliefs regarding the likelihood that recommended behaviors will keep them safe or mitigate harm (e.g., evacuating and stocking supplies). Efficacy can be further broken down into self-efficacy (i.e., I can do the things needed to keep me safe) and system efficacy (i.e., systems in place within our society will help keep us safe; Bani-Amerian & Venette, 2020). This study also extends the conceptualization of the RBD scale with the addition of joint efficacy (i.e., solutions that can only be achieved by working together). Therefore, we conducted a content analysis using 120 videos on TikTok covering the Palisades Fire to explore risk perception and response efficacy.

Findings highlight the importance of users utilizing TikTok to engage in crisis communication, especially by sharing real-time updates and localized information. Results also indicate that joint efficacy should be further examined as a construct of the RBD scale. For example, several videos demonstrated how creators worked with commenters to visit different locations to update them on the status of their homes. Given TikTok's popularity, scholars should continue to explore how individuals use this platform to communicate risk and coordinate responses during a crisis. Finally, practitioners should collaborate with TikTok creators to develop strategies to construct and disseminate effective risk messaging.

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Greater Netrin-1 in Male ADPKD Mice Enhances Cystogenesis Than Females

Presenter(s): Lillian Witherington

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Faculty Sponsor(s): Riyaz Mohamed, PhD

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ABSTRACT

Autosomal dominant polycystic kidney disease (ADPKD) is the most common inherited cause of end-stage renal disease (ESRD). ADPKD is characterized by excessive cellular proliferation and fluid-filled cyst formation in the kidneys, which contribute to the development of ESRD and kidney failure. Notably, a sex difference has been observed in ADPKD, with males exhibiting larger cyst growth and faster disease progression than females in both clinical cases and rodent ADPKD models. However, the underlying mechanisms driving these differences remain poorly understood. Netrin-1, a laminin-related secreted protein that is upregulated following kidney injury, is known to play roles in cell proliferation, migration, and tumor growth. However, its involvement in ADPKD and potential sex-specific differences has not been explored. We hypothesize that greater upregulation of netrin-1 in male mice contributes to accelerated cyst growth compared to females. To assess this, 9-10-week-old male and female wild-type (WT) and *Pkd1* mutant mice were randomized to receive either vehicle or a netrin-1-specific neutralizing monoclonal antibody for 4 weeks. At 14 weeks of age, all mice were euthanized, and blood and kidney samples were collected for histological, biochemical, and Western Blot analyses. Renal netrin-1 protein levels were measured using both Western Blot and enzyme-linked immunosorbent assay (ELISA). Renal function was evaluated via measurement of blood urea nitrogen (BUN), cell proliferation in kidney was measured via ki67 staining, and cyst growth was determined by measurement of the kidney to body weight ratio. Our results showed that *Pkd1* mutant mice had significantly higher renal netrin-1 levels than WT mice, along with higher BUN, increased cell proliferation, and more cyst growth. Moreover, male *Pkd1* mutant mice had greater netrin-1 levels, cell proliferation, worse kidney function, and greater cyst growth than age-matched females. Notably, the neutralization of netrin-1 significantly reduced cell proliferation, cyst growth, and improved kidney function in both sexes. However, the effect was more pronounced in males when compared to females. These findings suggest that netrin-1 plays a key role in the sex-specific differences seen in renal cyst progression. Future work will aim to elucidate the impact of sex hormones and the underlying mechanisms by which netrin-1 mediates differences in cyst growth between both sexes.

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Oligodendrocyte H3K9me3 Dynamics in Mice Housed in an Enriched Environment

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Faculty Sponsor(s): Dr. Evan Goldstein

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ABSTRACT

Developmental myelination is essential for processing information and transmitting electrical signals efficiently between neurons. Myelin related diseases, such as multiple sclerosis, damages myelin and manifests as negative effects like motor dysfunction. It has been established that the environment can influence mice myelination through enriched environment (EE) methods. EE provides mice with a multitude of interactions within their environment compared to a standard environment (SD): 8-12 mice cages, rotation of toys, and voluntary physical activity. During prolonged EE, oligodendrocytes (OLs), the supporting glial cells responsible for producing myelin, activity enhances myelination both anatomically and genetically. Mice exposed to EE have many differentially expressed genes (DEGs) in their OLs, therefore, understanding these modifications may be novel targets for minimizing myelin-related diseases. Histone modifying enzymes (HMEs) are one notable group of DEGs after EE, and their presence corresponds with specific histone modifications. Oligodendrocyte progenitor cells (OPCs) are precursor cells that can differentiate into OLs and histone-3-lysine 9-trimethylation (H3K9me3), a histone modification, plays a role in the OPC differentiation process. Deleting H3K9me3 causes impaired OLs development and affects the electrical conduction between myelinated neurons. Therefore, EE conditions might affect the presence of this histone mark due to differentially expressed HMEs. Additionally, SETDB1 is a methyltransferase that trimethylates H3K9, ultimately recruiting H3K9me3. This pathway is required for early developmental myelination in mice, further validating H3K9me3's role in myelination.

The project compared 2 groups of mice: Mice housed in an EE and standard environment (SD). Wild-type mice were housed in their respective conditions from postnatal day 15 (P15) to P45, P60 or P90. At experimental endpoint, mice were euthanized by cardiac perfusion and brains were dissected. Brains were then cryopreserved, sliced, and stained using different antibodies to label OL lineage cells and H3K9me3. A confocal microscope was used to examine tissue, and ImageJ to quantify histone marks. Quantification revealed that H3K9me3 prevalence in P45 OPCs increased, but decreased significantly in P60 EE mice. The results indicate a reversal of prevalence for H3K9me3, which suggests that H3K9me3 has implications for regulating myelination at later time points. Specifically, there is a possibility that the decrease of H3K9me3 expresses genes that induce myelination in enriched environments.

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A Process for Tailoring Scientific Abstracts for Patient Audiences

Presenter(s): Taylor Grace Yancey

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Faculty Sponsor(s): Lauren Cafferty, MA

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ABSTRACT

Effective communication in healthcare settings requires tailoring complex medical information to the needs and preferences of patients. The importance of adapting health messages lies in ensuring that healthcare professionals and patients can understand each other when speaking about complex health information. The purpose of this study is to reveal the process and importance of medical translation and produce an accessible resource about research to patient audiences. Translating science into accessible information for patients is a long process that requires consideration of how patients will receive the information being provided to them. The more accessible scientific information becomes to the public, the more people feel empowered to make decisions pertaining to their health. In this project, I began with an abstract from a scientific study. I researched successful examples of medical translation and found that it is important to keep only the information that patients can read, understand, and apply to their own experiences. Then, I rewrote the abstract from the original study, highlighting what is important for patients to know. Additionally, I used the Hemmingway writing check to ensure the writing remained at the correct reading level. The reading level that most patients comprehend is a 6th grade level. Lastly, the finished abstract was shared with community members who provided suggestions on how to improve comprehension. In total, the translated abstract underwent more than 10 rounds of revisions. The result of this project is a tailored version of the original scientific abstract that patients can read, understand, and apply to their own healthcare experiences. Translating science into accessible information is important because it gives patients the power to advocate for themselves and to be informed. It is crucial that patients are aware of the scientific resources at their disposal as it may help them apply scientific knowledge to their own lives.

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Nature to Novel: Ursolic Acid Synthetic Hybrids for Cancer Treatment

Presenter(s): Hayden Yi

Author(s): Hayden Yi

Faculty Sponsor(s): Siva S. Panda, PhD

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ABSTRACT

Natural products are pivotal in contemporary drug discovery, providing a wealth of chemical scaffolds with considerable therapeutic potential. Ursolic acid, a pentacyclic triterpenoid commonly found in edible plants, has attracted considerable research attention for its anticancer properties; however, it presents significant challenges, including poor bioavailability, rapid metabolic clearance, and limited efficacy against aggressive tumor types. This project investigates a molecular hybridization strategy to enhance the therapeutic attributes of ursolic acid by modifying two critical functional sites, thereby creating novel conjugates with improved anticancer efficacy. Using optimized synthetic protocols, three distinct series of ursolic acid-based hybrids were synthesized via click chemistry, alkylation, and secondary amine coupling. These reactions yielded the intended molecules in high yields while ensuring excellent regioselectivity. The structural characterization of these hybrids was corroborated by Nuclear Magnetic Resonance (NMR), Infrared (IR) spectroscopy, and mass spectrometry. The synthesized conjugates were assessed for antiproliferative activity against human bladder cancer cell lines (5637 and T24), a triple-negative breast cancer model (MDA-MB-231), and a non-tumorigenic breast epithelial cell line (MCF-12A) to evaluate selectivity. Several of the novel hybrids exhibited significantly enhanced potency relative to the parent natural product, particularly those featuring heterocyclic amine substituents at the modified C-28 position. Among the newly developed compounds, select conjugates demonstrated potent cytotoxicity against both bladder and breast cancer cells while exhibiting reduced toxicity toward non-tumorigenic cells. Notably, one hybrid exhibited robust activity against chemotherapy-resistant bladder cancer cells, underscoring its potential utility in cases where conventional therapeutic agents are ineffective. Mechanistic investigations suggest that the anticancer activity of these conjugates may engage multiple pathways, including autophagy and programmed cell death. Preliminary pharmacokinetic evaluations in animal models indicated favorable stability and a promising safety profile. This investigation illustrates that molecular hybridization is an effective approach for transforming a biologically active natural product into a more potent and selective anticancer agent. These findings underscore the potential of ursolic acid-based hybrids as candidates for further mechanistic exploration and potential advancement to therapeutic applications.

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