



CROSS-CAMPUS TRANSLATIONAL BIOMEDICAL RESEARCH SYMPOSIUM

Where Oklahoma's Brightest Minds Move Discovery to Healthcare



CROSS-CAMPUS
Translational
Biomedical Research Symposium



SEPTEMBER 25, 2026

SAMIS EDUCATION CENTER | 1200 CHILDREN'S AVE. | OKLAHOMA CITY, OK 73104



CROSS-CAMPUS
Translational
Biomedical Research Symposium



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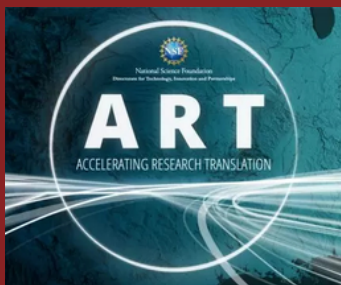
OFFICE OF TECHNOLOGY COMMERCIALIZATION
The UNIVERSITY of OKLAHOMA



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OKBioStart
From Lab to Life



The Stephenson Cancer Center and its activities are made possible through funding from the Oklahoma Tobacco Settlement Endowment Trust (TSET)



WELCOME



Dear Symposium Participants,

Welcome to the inaugural Cross-Campus Translational Biomedical Research Symposium (CCTBRS): Moving Discovery to Healthcare. We are delighted to bring together researchers, clinicians, engineers, entrepreneurs, commercialization experts, institutional leaders, and innovators from across the University of Oklahoma and the broader Oklahoma innovation ecosystem. This symposium is an opportunity to celebrate the discoveries taking place across our community while, importantly, focusing on how we can work together to move those discoveries toward meaningful impact for patients, healthcare, and society.

We are delighted to welcome over 300 participants, from OU Norman, Health, and Tulsa campuses, as well as from other Oklahoma Institutions and organizations, including Oklahoma State University, ARL Bio Pharma, Novartis Pharmaceutical Company, and OMRF . In addition to the seasoned researchers and practitioners, we are extremely encouraged to welcome a large number of young scholars, including junior faculty members trainees, postdoctoral fellows, graduate students, medical school students, undergraduate students, and high school students. Together, we have ~ 150 posters representing a broad range of research topics.

Throughout the day, you will hear from researchers and innovators whose work demonstrates the many pathways from discovery to real-world application. Our program includes presentations on translational biomedical research, behavioral science, health data, imaging and perfusion, surgical extended reality, antimicrobial therapeutics, and other emerging technologies.

The symposium is intentionally designed to be interactive. In addition to scientific presentations and poster sessions, we will explore practical questions surrounding commercialization and entrepreneurship, intellectual property, FDA and regulatory



pathways, clinical research engagement, funding, manufacturing, and interdisciplinary collaboration. These conversations are intended not only to identify barriers, but to generate solutions, strengthen partnerships, and help each of us identify the resources and relationships needed to advance our work.

We especially encourage you to take advantage of the consultation opportunities, workshops, and guided networking sessions. Translation rarely happens in isolation, and the afternoon networking activities are designed to connect you with people and expertise in areas such as intellectual property, funding, commercialization, regulatory strategy, manufacturing, clinical trials, engineering, and clinical collaboration.

Whether you are at the beginning of an idea, actively developing a discovery, preparing for clinical translation, or exploring commercialization, we hope you will find valuable connections and practical insights throughout the symposium. Whether you are an experienced researcher/ practitioner or a young scholar, we hope you will share your own experiences, ask questions, and engage openly with colleagues from disciplines and backgrounds different from your own, gain confidence, and move your translational research and entrepreneurship to the next level.

Thank you for being part of this community and for contributing your expertise, curiosity, and enthusiasm. Together, we have an opportunity to strengthen the translational research ecosystem at OU and across Oklahoma—and to help move promising discoveries from the laboratory toward the patients and communities who can benefit from them.

We look forward to an engaging day of discovery, collaboration, and connection.

Warmly,

Wei R. Chen, PhD and Doris M. Benbrook, PhD; Symposium Co-Chairs



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	LEVEL 2 HALLWAY	RAINBOLT AUDITORIUM	BASEMENT	LEVEL 1 HALLWAY	BOARDROOM EAST	BOARDROOM WEST
11:00 AM		Learning Activity : Success in Finding Translational Resources Panel		Poster		
11:30 AM		Networking Break		Judging		
12:00 PM		Pick up Lunch				
12:30 PM		Panel : Commercialization and Entrepreneurship	Roundtable: Clinical Researcher Engagement and Barriers & Cross-Disciplinary Translational Collaboration			
1:00 PM				Poster	Consultation:	Consultation:
1:30 PM		Break		Judging		
2:00 PM		Workshop : FDA Regulatory Pathway for Drugs	Workshop: IP & FDA Regulatory Considerations: Medical Devices & Software	Poster	Office Hours	Office Hours
2:30 PM						
		Break				
3:00 PM		Flash Talks:				
3:30 PM		Oral Competition		Display		
4:00 PM		Break				
4:30 PM		Learning Activity: Pitching with Ron Bolen	Learning Activity: Group Networking, Translational Connections			
5:05 PM						
5:30 PM			Closing			
6:00 PM						
6:30 PM			Reception			
7:00 PM						
7:30 PM						

CCTBRS Program Agenda

TIME	SESSION	LOCATION
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7:30 – 8:25 AM	Breakfast & Registration	Level 2 Hallway
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WELCOME SESSION

8:25 – 8:30 AM	Wei R. Chen, PhD & Doris Mangiaracina Benbrook, PhD	Rainbolt Auditorium
8:30 – 8:45 AM	Lara Ferry, PhD	Rainbolt Auditorium
8:45 – 9:00 AM	John Klier, PhD	Rainbolt Auditorium
9:00 – 9:15 AM	Robert Mannel, MD	Rainbolt Auditorium

SPEAKERS

9:15 – 9:30 AM	Michelle vanDellen, PhD	Rainbolt Auditorium
9:30 – 9:40 AM	BREAK	Rainbolt Auditorium
9:40 – 9:45 AM	Elizabeth Hile, PT, PhD, NCS, CLT	Rainbolt Auditorium
9:45 – 10:00 AM	H. Anne Pereira, PhD	Rainbolt Auditorium
10:00 – 10:15 AM	David C. Kendrick, MD, MPH	Rainbolt Auditorium
10:15 – 10:30 AM	Qinggong Tang, PhD	Rainbolt Auditorium
10:30 – 10:45 AM	Jeff Potts, PhD & Christian El Amm, MD	Rainbolt Auditorium
10:45 – 11:00 AM	Mark M. Mims, MD	Rainbolt Auditorium

LEARNING ACTIVITY

11:00 – 11:30 AM	Success in Finding Translational Resources Panel	Rainbolt Auditorium
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POSTER SESSIONS

8:00 AM – 5:00 PM	Posters on Display	Level 1
11:00 AM – 12:00 PM	Poster Session 1 Judging	Level 1
11:30 AM – 12:00 PM	Networking Break / Poster Viewing	Level 1
1:00 – 2:00 PM	Poster Session 2 Judging	Level 1

LUNCH & ROUNDTABLES

12:00 – 12:15 PM	Pick up Lunch	Rainbolt Auditorium
12:15 – 1:30 PM	Commercialization and Entrepreneurship Pathways Panel	Rainbolt Auditorium
12:00 – 12:15 PM	Pick up Lunch	Basement Room B
12:15 – 1:30 PM	Clinical Researcher Engagement and Barriers Roundtable & Cross-Disciplinary Translational Collaboration Roundtable	Basement Room B
1:30 – 1:45 PM	BREAK	Basement Room B

CCTBRS Program Agenda

CONSULTING AGENCY OFFICE HOURS

1:00 – 4:00 PM	OKCatalyst — Jaime Hough, PhD Startup OU — Micah Headley Fabrication Lab — Chase Greeno	Boardroom East
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1:00 – 4:00 PM	OKBioStart — Emi Kiyotake, PhD & Martha Tsaliki, PhD Office of Technology and Commercialization — Andrew Pollock, MBA	Boardroom West
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LEARNING ACTIVITIES (CONCURRENT)

1:45 – 2:45 PM	FDA Regulatory Pathway for Drugs Workshop	Rainbolt Auditorium
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2:45 – 3:00 PM	BREAK	Rainbolt Auditorium
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1:45 – 2:45 PM	IP & FDA Regulatory Considerations: Medical Devices & Software Workshop	Basement Room B
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2:45 – 3:00 PM	BREAK	Basement Room B
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ORAL COMPETITION

3:00 – 3:08 PM	Snehal Patel, MD, PhD	Rainbolt Auditorium
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3:10 – 3:18 PM	Grant Chesbro, PhD	Rainbolt Auditorium
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3:20 – 3:28 PM	Soumyabrata Munshi, MD, PhD	Rainbolt Auditorium
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3:30 – 3:38 PM	Kaili Liu, PhD	Rainbolt Auditorium
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3:40 – 3:48 PM	David Miller, PhD	Rainbolt Auditorium
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3:50 – 3:58 PM	Edriss Mirnia, PhD	Rainbolt Auditorium
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3:58 – 4:15 PM	BREAK	Rainbolt Auditorium
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LEARNING ACTIVITIES (CONCURRENT)

4:15 – 5:30 PM	How to Pitch Your Innovation — with Ron Bolen, JD	Rainbolt Auditorium
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4:15 – 5:30 PM	Group Networking, Translational Connections	Basement Room B
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WRAP-UP

5:30 – 5:45 PM	Announcement of Winners and Closing	Basement Room B
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5:45 – 7:30 PM	RECEPTION	Basement Room B
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CROSS-CAMPUS
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COMMITTEES

STEERING COMMITTEE



Wei R. Chen, PhD
Co-Chair

*Stephenson Chair, Professor, and Director,
Stephenson School of Biomedical Engineering,
The University of Oklahoma*



**Doris Mangiaracina
Benbrook, PhD**
Co-Chair

*Associate Director for Translational Research,
Stephenson Cancer Center, Presidential Professor,
Presbyterian Health Foundation Member,
Harold Hamm Diabetes Center,
The University of Oklahoma Health Campus*



**Michelle
vanDellen, PhD**

*Professor and George Kaiser Family
Foundation Chair in Clinical Translational Science,
Stephenson Cancer Center,
The University of Oklahoma Health Campus*



**Nicholette Davis,
AuD, MBA**

*Executive Director, OKBioStart
The University of Oklahoma*



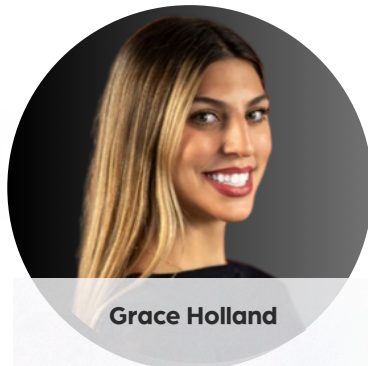
**Zachary Pope,
PhD, ACSM-EP**

*Assistant Professor, Department of Health Promotion
Sciences, Hudson College of Public Health & TSET Health
Promotion Research Center, Stephenson Cancer Center,
The University of Oklahoma Health Campus*



Dalton Krueger

*Admin/Finance Coordinator,
OKBioStart
The University of Oklahoma*



Grace Holland

*Marketing Program Manager
OKBioStart
The University of Oklahoma*



**Jill Rosewood-
Evans, MS**

*Events & Conferences Manager,
Stephenson Cancer Center
The University of Oklahoma Health Campus*

ROUNDTABLE & PANEL SUBCOMMITTEE



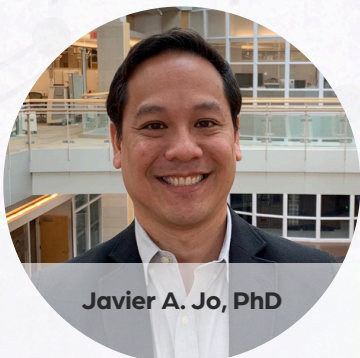
Fatima Merchant, PhD
Co-Chair

*Professor SBME, Director of
Biomedical Data Science GCoE,
The University of Oklahoma*



Nicholette Davis,
AuD, MBA
Co-Chair

*Executive Director, OKBioStart
The University of Oklahoma*



Javier A. Jo, PhD

*Professor and George Kaiser Family
FounTed and Cuba Webb Presidential Professor of
Electrical and Computer Engineering; Director,
NIH-P20 COBRE Oklahoma Center of Medical
Imaging for Translational Cancer Research The
University of Oklahoma*



Martha Tsaliki, PhD

*Program Manager
OKBioStart
The University of Oklahoma*



H. Anne Pereira, PhD

*Regents' Professor, David L. Boren
Professor & Henry Zarrow Presidential
Professor, Department of Pharmaceutical Sciences,
The University of Oklahoma
Health Campus*



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SPEAKER & MODERATOR SUBCOMMITTEE



**Bulent Ozpolat,
PhD, MD
Co-Chair**

*Endowed Chair Professor,
The University of Oklahoma*



**Qinggong Tang, PhD
Co-Chair**

*Associate Professor of Stephenson
School of Biomedical Engineering,
The University of Oklahoma*



**Michelle
vanDellen, PhD**

*Professor and George Kaiser Family
Foundation Chair in Clinical Translational Science,
Stephenson Cancer Center,
The University of Oklahoma Health Campus*



Vivek K. Bajpai, PhD

*Assistant Professor School of Sustainable
Chemical, Biological and Materials Engineering,
The University of Oklahoma*



Faizah Bhatti, MD, MS

*Professor of Pediatrics,
Ophthalmology & Cell Biology,
The University of Oklahoma*



Elizabeth Hile, PhD

*Associate Professor,
Director of Cancer Rehabilitation Sciences Program,
The University of Oklahoma Health Campus*



Vibhuti Agrahari, PhD

*Assistant Professor, Department of
Pharmaceutical Sciences
The University of Oklahoma Health Campus*

ABSTRACTS, POSTERS AND ORAL PRESENTATIONS SUBCOMMITTEE



Anne Kasus-Jacobi, PhD
Co-Chair

*Associate Professor
The University of Oklahoma Health Campus*



Zachary Pope,
PhD, ACSM-EP
Co-Chair

*Assistant Professor, Department of Health Promotion Sciences,
Hudson College of Public Health & TSET Health Promotion
Research Center, Stephenson Cancer Center,
The University of Oklahoma Health Campus*



Martha Tsaliki, PhD

*Program Manager
OKBioStart
The University of Oklahoma*



David Miller, PhD

*Assistant Professor of Biomedical Engineering,
The University of Oklahoma*



Tingting Gu, PhD

*Associate Director of Advanced Light Microscopy
core, Assistant professor, School of Biological
Sciences, The University of Oklahoma*



Emi Kiyotake, PhD

*Concept Program Manager
OKBioStart
The University of Oklahoma*



Chongle Pan, PhD

*Professor of Computer Science and
Biomedical Engineering,
The University of Oklahoma*



Lin Wang, PhD

*Research Assistant Professor,
The University of Oklahoma*

LEARNING ACTIVITIES SUBCOMMITTEE



Elizabeth Hile, PhD
Chair

*Associate Professor,
Director of Cancer Rehabilitation Sciences Program,
The University of Oklahoma Health Campus*



**Doris Mangiaracina
Benbrook, PhD**

*Associate Director for Translational Research,
Stephenson Cancer Center, Presidential Professor,
Presbyterian Health Foundation Member,
Harold Hamm Diabetes Center,
The University of Oklahoma Health Campus*



**Sharukh Khajotia,
BDS, MS, PhD**

*Associate Dean for Research and
Innovation, The University of Oklahoma
Health Campus*



John Klier, PhD

*Dean and Professor, Gallogly
College of Engineering, The
University of Oklahoma*



**Michael Schade,
JD, CLP**

*Executive Director, Research Legal
Strategy & Assistant General
Counsel, The University of
Oklahoma*



H. Anne Pereira, PhD

*Regents' Professor, David L. Boren
Professor & Henry Zarrow Presidential
Professor, Department of Pharmaceutical
Sciences, The University of Oklahoma
Health Campus*



Paul DeAngelis, PhD

*Presidential Professor, Department of Biochemistry &
Physiology, The University
of Oklahoma Health Campus*



Huynh Le

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**Faizah Bhatti,
MD, MS**

*Professor of Pediatrics,
Ophthalmology & Cell Biology,
The University of Oklahoma*



Andrew Pollock, MBA

*Managing Director for the Office of Technology
Commercialization,
The University of Oklahoma*



CROSS-CAMPUS Translational Biomedical Research Symposium



WELCOME SPEAKERS

WELCOME SESSION

Rainbolt Auditorium

Moderators: Jeffery S. Volz, SE, PE, PhD

7:30 am – 8:25 am

Breakfast & Registration

8:25 am – 8:30 am

Welcome & Introduction

Wei R. Chen, PhD co-chair - Stephenson Chair, Professor, and Director, Stephenson School of Biomedical Engineering, University of Oklahoma

Doris Mangiaracina Benbrook, PhD co-chair - Associate Director for Translational Research, Stephenson Cancer Center, Presidential Professor, Presbyterian Health Foundation Member, Harold Hamm Diabetes Center, University of Oklahoma OUHC

8:30 am – 8:45 am

From Discovery to Impact II: Organizing Ourselves for Translation to Strengthen Oklahoma and Beyond

Lara Ferry, PhD – Professor of Biology, Vice President and Chief Research Innovation Officers, OU

8:45 am – 9:00 am

Translational biomedical research at OU engineering and biomanufacturing

John Klier, PhD – Dean of the Gallogly College of Engineering OU

9:00 am – 9:15 am

Stephenson Cancer Center Update

Robert Mannel, MD – Director of Stephenson Cancer Center OUHC

WEI R. CHEN, PHD

*Stephenson Chair, Professor, and Director,
Stephenson School of Biomedical Engineering,
The University of Oklahoma*



Dr. Wei R. Chen received his B.S. degree in Physics from Shandong University, Jinan, China, in 1982 and his Ph.D. in Theoretical High-Energy Particle Physics from the University of Oregon in 1988. He transitioned his research focus from physics to cancer research in the early 1990s. Dr. Chen joined the Stephenson School of Biomedical Engineering (SBME) at the University of Oklahoma (OU) in 2020 and currently serves as Stephenson Chair, Professor, and Director of SBME. He is a co-Leader of the Cancer Therapeutics Program at the Stephenson Cancer Center, OU Health Sciences Center. He is also the Director of the Oklahoma Center of ImmunoEngineering (OCIE), funded through the NIH Centers of Biomedical Research Excellence (COBRE) program. A pioneer in the field of immunophotonics, Dr. Chen and his team developed a localized ablative immunotherapy for metastatic cancers that combines local ablation with the local administration of novel immunostimulants.

This innovative approach has demonstrated promising results in both preclinical studies and early clinical trials. Dr. Chen has published more than 210 peer-reviewed articles (h-index: 64; over 12,500 citations) and holds 12 U.S. patents as well as multiple international patents. He has secured more than \$20 million in research funding from state and federal agencies, foundations, and industry sponsors. Dr. Chen was elected as a Fellow of SPIE (International Society for Optics and Photonics) in 2007, a Fellow of AIMBE (American Institute for Medical and Biological Engineering) in 2022, and a Senior Member of NAI (National Academy of Inventors) in 2024. Prior to joining OU, he served as Dean of the College of Mathematics and Science at the University of Central Oklahoma (UCO) from 2016 to 2020. An accomplished educator, Dr. Chen received the 2008 U.S. Professor of the Year award and the 2011–2012 U.S. Fulbright Lecturing/Research Award. He was also honored with the Oklahoma Foundation for Excellence's Medal for Excellence in Teaching in 2011 and the SPIE Educator Award in 2012. Dr. Chen is a co-founder and board director of Immunophotonics, Inc., a biotech company. Immunophotonics received the US FDA approval in April 2023 for a Phase 1b/2a multicenter clinical trial for patients with late-stage colorectal cancer, non-small cell lung cancer, and soft tissue sarcoma, using the novel therapy developed by Dr. Chen's team.

DORIS MANGIARACINA BENBROOK, PHD

*Associate Director for Translational Research,
Stephenson Cancer Center, Presidential Professor,
Presbyterian Health Foundation Member,
Harold Hamm Diabetes Center,
The University of Oklahoma Health Campus*



Doris Mangiaracina Benbrook, PhD, is the Associate Director of Translational Research at the Stephenson Cancer Center, Presbyterian Health Foundation Presidential Professor in the Gynecologic Oncology Section, Obstetrics and Gynecologic Oncology Department at the OU Health Campus and Member of the Harold Hamm Diabetes Center. Her research is focused on developing drugs for prevention and treatment of gynecologic cancers and other diseases. Her major objective is to ensure that the drugs do not cause harmful side effects. She recruited and led multidisciplinary teams to bring a drug (SHetA2, also called OK-1) from concept to a Phase 1 clinical trial (clinicaltrials.gov: NCT04928508) entirely within an academic setting with no pharmaceutical or dilutive funding. The Phase 1 trial showed no dose-limiting side effects of SHetA2 and the investigational drug is now being translated into an NCI-funded Phase 2 trial. Dr. Benbrook is sharing her knowledge by developing and directing translational research curriculum at the University of Oklahoma and mentoring the translation of discoveries of others.

She serves as the Multi-Principal Investigator of the Route 66 Endometrial Cancer SPORE and Principal Investigator of a Partnering PI Department of Defense Ovarian Cancer Research Program grant.

Dr. Benbrook earned her PhD in Biochemistry and Molecular Biology from Loyola University Medical Center in Maywood, IL, USA. As a postdoctoral researcher at the La Jolla Cancer Research Foundation (now the Burnham Institute) in San Diego, CA, USA, she discovered, cloned and characterized a retinoic acid receptor, thyroid hormone receptor and cholesterol receptor. During her tenure as a Research Fellow at the Imperial Cancer Research Fund (now Cancer Research UK) she discovered crosstalk between transcription factors demonstrating integration between different signal transduction pathways.

LARA FERRY, PHD

Professor of Biology, Vice President and Chief Research Innovation Officers, The University of Oklahoma



With over 25 years in academia, Lara is an accomplished field and laboratory scientist, and an award-winning teacher and mentor. She joined the University of Oklahoma in 2026 as the inaugural Vice President and Chief Research Innovation Officer, and Professor of Biology. In her leadership role, she is tasked with leveraging her prior experiences to bring commercialization, strategic engagement, partnerships, and venture development together under one leader so that discoveries at OU move faster into real-world use, delivering economic impact to Oklahoma and our nation. She collaborates closely with faculty and academic units to advance discovery research, both basic and applied, in alignment with the university's strategic goals. Prior to serving at OU, Lara was at Arizona State University for 16+ years, most recently as the Vice President for Research and President's Professor. She has served in leadership roles on both the academic and research sides of the house, working to deliver on the promise of the state university in meeting the needs of the community and the region. Lara is a driver of change, connecting passion with opportunity, seeking meaningful and lasting impact. She is a champion of the benefits that

universities can and should provide for individuals, the state, and our nation.

Lara is broadly trained as a comparative physiologist and functional morphologist, broadly interested in the evolution of jaws. Her recent research is focused on the material properties of cartilage in the jaws of elasmobranch (sharks). She completed her postdoctoral training at the University of California Davis. She received her PhD in the Comparative Physiology Graduate Group at the University of California at Irvine, her MS in Marine Science from San Francisco State University/Moss Landing Marine Laboratories, and her BS in Biology from Cal Poly San Luis Obispo. In addition to her administrative appointment, she is a Professor of Biology in the School of Biological Sciences in the Dodge College of Arts and Sciences at OU.

JOHN KLIER, PHD

Dean and AT&T Chair of the Gallogly College of Engineering and professor in the School of Chemical, Biological and Materials Engineering.

John Klier joined OU in July of 2020 where he serves as dean and AT&T Chair of the Gallogly College of Engineering and professor in the School of Chemical, Biological and Materials Engineering. He joined OU from the University of Massachusetts–Amherst where he served as professor and department head of Chemical Engineering. He previously worked in the private sector with Dow Chemical Co. in technology and technical management roles, included leading Core R&D materials, Dow Coating Materials and Performance Materials and Chemicals divisional research, development and technical service organizations. In 2014 he was named Distinguished Fellow, the highest technical position at Dow.



His research activities include advanced coatings, new polymer materials, renewable and recyclable plastics, and polymer colloids for targeted therapy and other applications.

Klier is a member of the National Academy of Engineering and the National Academy of Inventors. His service to the national academies includes vice chair of the National Materials and Manufacturing Board and recent committee chair and committee positions.

ROBERT MANNEL, MD

Director of the Stephenson Cancer Center at the University of Oklahoma Health Sciences Center



Robert Mannel, MD, is the Director of the Stephenson Cancer Center at the University of Oklahoma Health Sciences Center. Since 2010 he has held the Rainbolt Family Endowed Chair in Cancer. In addition, he serves as the Associate Vice Provost for Cancer Programs at OUHSC, and he is a tenured Professor in the OU Department of Obstetrics and Gynecology. As the Director of the Stephenson Cancer Center, Dr. Mannel oversees all clinical, research, administrative and educational activities related to oncology medicine and cancer research at OU. Under his leadership, the Stephenson Cancer Center has grown to become the largest provider of cancer care in Oklahoma. When he was appointed Director in 2007, the Stephenson Cancer Center provided care for 1 out of every 20 cancer patients diagnosed in Oklahoma. Today, it is 1 out of every 6. This year, the Cancer Center will provide care for nearly 3,000 new adult and pediatric cancer patients.

During his time as Director, the amount of cancer research funding to investigators at OU has increased from under \$5 million to nearly \$50 million. The Stephenson Cancer Center is now the #1 ranked cancer center nationally for the number of patients participating in National Cancer Institute-sponsored clinical trials for promising new cancer therapies.

Dr. Mannel currently serves as the Chair for NRG Oncology, one of four organizations that make up the National Clinical Trials Network. NRG Oncology is the largest group within the Network, placing more cancer patients on NCI clinical trials than any other organization. Dr. Mannel graduated from the University of Texas – College of Medicine at Galveston and completed his residency in obstetrics and gynecology at Scott and White Hospital in Temple, Texas. After residency, he completed a fellowship in gynecologic oncology at the University of California – Irvine. In 1989, he joined the faculty at the University of Oklahoma and served as Chair of the OU Department of Obstetrics and Gynecology from 1997 to 2013. He was instrumental in developing the Section of Gynecologic Oncology at the University of Oklahoma into one of the top gynecologic oncology programs in the country.



MODERATOR

JEFFERY S. VOLZ, SE, PE, PHD

Associate Dean for Partnerships and a Lloyd G. and Joyce Austin Presidential Professor in the School of Civil Engineering and Environmental Science (CEES)

Jeffery S. Volz, SE, PE, PhD, serves as Associate Dean for Partnerships in the Gallogly College of Engineering at the University of Oklahoma. He is also the Lloyd G. and Joyce Austin Presidential Professor and former Director of the Donald G. Fears Structural Engineering Laboratory in the School of Civil Engineering and Environmental Science.

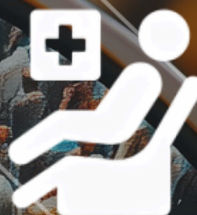
As Associate Dean, Dr. Volz leads strategic partnership development with industry, nonprofit organizations, state and local governments, and tribal nations. His work focuses on advancing translational research, fostering innovation, and strengthening pathways from discovery to deployment.

As a faculty member, Dr. Volz has authored more than 125 peer-reviewed publications and presented his research at numerous national and international scientific conferences. He has secured more than \$26 million in total research funding, including nearly \$5 million in individual research credit. His research integrates structural engineering and materials science, with a focus on material innovations that enhance structural performance, durability, and resilience.

Before entering academia, Dr. Volz spent 16 years in professional practice, contributing to the structural design and construction of major commercial and residential buildings.



CROSS-CAMPUS Translational Biomedical Research Symposium



INVITED SPEAKERS

INVITED SPEAKERS

Rainbolt Auditorium

Moderator: David Miller, PhD

9:15 am – 9:30 am	Leveraging Behavioral Science to Accelerate Biomedical Translational Efforts Michelle vanDellen, PhD - Professor and George Kaiser Family Foundation Chair in Clinical Translational Science, Stephenson Cancer Center, OUHC
9:30 am – 9:40 am	BREAK
9:40 am – 9:45 am	Introduction to Founders Elizabeth Hile, PT, PhD, NCS, CLT – Associate Professor, Program Director, PhD Program, Director, Cancer Rehabilitation Services and Research Lab, Stephenson Cancer Center, OUHC
9:45 am – 10:00 am	Bad bugs, no drugs, cationic antimicrobial peptides to the rescue H. Anne Pereira, PhD - Regents' Professor, David L. Boren Professor & Henry Zarrow Presidential Professor, Department of Pharmaceutical Sciences, OUHC
10:00 am – 10:15 am	Health Data Utilities as sources of Real-World Data David C. Kendrick, MD, MPH - Chair Department of Medical Informatics, OU School of Community Medicine, Associate Vice Provost for Strategic Planning, OU Health Campus
10:15 am – 10:30 am	From Organ Imaging to Organ Perfusion: Advancing Translation Through Academic–Clinical–Industry Collaboration Qingong Tang, PhD - Associate Professor of Stephenson School of Biomedical Engineering (SBME), Endowed Stephenson Professor, OU
10:30 am – 10:45 am	Surgical Extended Reality: At the intersection of modeling, optics and physical AI Jeff Potts, PhD - Chief Technology Officer at Xironetic Christian El Amm, MD - Professor of Surgery, Craniofacial Surgery, Plastic and Reconstructive Surgery, University of Oklahoma, Xironetic founder
10:45 am – 11:00 am	From Clinical Need to Clinical Reality: Translating an Idea Beyond the Bedside Mark M Mims, MD – Associate Professor of Otolaryngology - Head and Neck Surgery, OU Health Campus



MICHELLE VANDELLEN, PHD

Professor and George Kaiser Family Foundation Chair in Clinical Translational Science, Stephenson Cancer Center, The University of Oklahoma Health Campus

Leveraging Behavioral Science to Accelerate Biomedical Translational Efforts

Despite decades of investment in translational science, an average of 17 years may pass between scientific discovery and routine practice, suggesting that biological innovation alone is insufficient to achieve population health impact.

Although biomedical discoveries are often viewed as progressing through a linear translational pathway from bench to bedside and ultimately to community impact, human behavior influences every stage of this process. Behavioral science—which is concerned with understanding how people think, feel, and act—can complement discoveries along this continuum. Basic and translational scientists—and sometimes behavioral scientists themselves—frequently assume that because they themselves are people, they can accurately predict how others will respond to interventions, technologies, messages, or health recommendations. People, however, are complex and substantial heterogeneity exists in how individuals in different social contexts and systems will think, feel, and act.

Behavioral science provides rigorous theories, methods, and empirical approaches for understanding and predicting the complexity. In this presentation, I highlight how behavioral science resources across University of Oklahoma campuses can be integrated throughout the translational continuum, from discovery and preclinical research to clinical implementation, commercialization, and community practice.

With better coordination, I argue that behavioral science integrated into earlier stages of translational science may reduce the delay between discovery and practice, ultimately increasing the extent to which innovations achieve their intended clinical impact.

MICHELLE VANDELLEN, PHD

Professor and George Kaiser Family Foundation Chair in Clinical Translational Science, Stephenson Cancer Center, The University of Oklahoma Health Campus

About Dr. VanDellen

Dr. VanDellen is a trained experimental Social Psychologist who currently serves as Professor and George Kaiser Family Foundation Chair in Clinical Translational Science in the Department of Health Promotion Sciences at the University of Oklahoma Health Campus.

Prior to joining the faculty at the University of Oklahoma in 2024, she earned her PhD in Social Psychology from Duke University and spent 12 years on faculty in the Department of Psychology at the University of Georgia. Her research investigates the basic mechanisms of how motivation and relationship processes combine to produce health behavior change in individuals and relationship systems.

Dr. vanDellen has been funded by the National Cancer Institute since 2020, with projects leveraging motivational processes to increase quitting in couples where both members smoke cigarettes. Additional funding for her research comes from the Presbyterian Health Foundation, the TSET Health Promotion Research Center, and the American Cancer Society through an Institutional Research Grant.

Here at the University of Oklahoma, Dr. vanDellen is pursuing new lines of research to integrate epigenomic feedback into smoking cessation interventions and regulatory science research that addresses the impact of messaging on food evaluation and consumption.

Outside of the lab, Dr. vanDellen spends free time experiencing the benefits of motivation and relationship for health by hiking in National Parks across the US with her husband.



H. ANNE PEREIRA, PHD

*Regents' Professor, David L. Boren Professor & Henry Zarrow
Presidential Professor, Department of Pharmaceutical
Sciences, The University of Oklahoma Health Campus*

Bad bugs, no drugs, cationic antimicrobial peptides to the rescue

This presentation will provide a high-level overview of a basic academic scientist's journey into the entrepreneurial world. It will explore the long and often tortuous path from the excitement of discovery to the challenges of development and commercialization, focusing on a first-in-class peptide therapeutic designed to treat severe multidrug-resistant Gram-negative infections.

The talk will highlight preclinical studies that led to two promising applications based on bioactive peptides derived from the neutrophil-associated immune defense molecule CAP37. One application targets ocular infections, while the other is aimed at pulmonary diseases, including cystic fibrosis and hospital- or ventilator-acquired pneumonia.

H. ANNE PEREIRA, PHD

*Regents' Professor, David L. Boren Professor & Henry Zarrow
Presidential Professor, Department of Pharmaceutical
Sciences, The University of Oklahoma Health Campus*

About Dr. Pereira

Dr. H. Anne Pereira is Regents' Professor in the Department of Pharmaceutical Sciences, in the College of Pharmacy at the University of Oklahoma Health Campus. She has been a member of the Health Campus since 1992 when she joined the faculty of the Department of Pathology as an assistant professor.

She rose through the ranks to full professor with tenure in Pathology, and in 2009 was appointed Professor of Pharmaceutical Sciences and served as the Associate Dean for Research, College of Pharmacy from 2009 to 2020. She was named the Henry Zarrow Presidential Professor in 2008 and the David L Boren Professor in 2018. She served as the Dean of the Health Sciences Graduate College from 2014 to 2025.

Dr. Pereira received her PhD in Pathology from the University of Melbourne in Australia and then served as a postdoctoral fellow/senior research associate at Emory University in Atlanta, before accepting her position at the University of Oklahoma. Her research focuses on the multifunctional aspects and identification of bioactive peptides based on the innate immune system molecule, CAP37 and has had a longstanding record of federal and national funding.

She has the distinction of being named a Fellow by the American Association for the Advancement of Science (AAAS) and an inaugural fellow of the American Association of Colleges of Pharmacy (AACP) Academic Research Program. Dr. Pereira is the holder of over 50 US and foreign patents and is the Co-founder and Chief Scientist of Biolytx, an OU spin-off company responsible for the commercial development of peptide therapeutics which she developed.

She was named a Fellow of the National Academy of Inventors in 2015 for her highly prolific spirit of innovation in creating or facilitating outstanding inventions that have made a tangible impact on quality of life, economic development, and the welfare of society.



DAVID KENDRICK, MD, MPH

*Chair Department of Medical Informatics, OU School of
Community Medicine, Associate Vice Provost for Strategic
Planning, The University of Oklahoma Health Campus*

Health Data Utilities as sources of Real World Data

As methods of machine learning and AI evolve, the availability of Real World Data, untouched by inference or algorithm, has become a critical natural resource, especially in healthcare.

A review and comparison of the potential sources of RWD in healthcare will be provided and a roadmap for achieving sustainable and secure access to this critical natural resource for the generation of new knowledge and continuous improvement of the human condition will be provided.

The creation, evolution, and impact of Health Data Utilities as a centerpiece of this critical infrastructure will be reviewed as well as specific opportunities broad availability of HDUs offer.

DAVID KENDRICK, MD, MPH

*Chair Department of Medical Informatics, OU School of
Community Medicine, Associate Vice Provost for Strategic
Planning, The University of Oklahoma Health Campus*

About Dr. Kendrick

Dr. David Kendrick is a nationally recognized leader in health data interoperability, population health, value-based care, quality improvement, and learning health systems. As Strategic Advisor to Civitas Networks for Health, he contributes to several national initiatives, including co-authoring the National HIO Survey, serving on the Health Data Utility (HDU) Steering Council, and advising the Patient Centered Data Home® program, which connects health information exchanges and HDUs across the country to support secure, consent-based health data sharing.

A native of rural Oklahoma, Dr. Kendrick returned to his home state in 2008 to found and chair the Department of Medical Informatics at the University of Oklahoma School of Community Medicine. He currently serves as Associate Vice Provost for Strategic Planning at the OU Health Sciences Center. His department delivers health information technology, analytics, care management, and quality improvement services that support physicians and improve outcomes for more than 250,000 patients statewide.

In 2009, Dr. Kendrick founded MyHealth Access Network, Oklahoma's state-designated Health Data Utility, serving more than 4 million patients and facilitating over 130,000 clinical encounters daily. During his 15-plus years as CEO, MyHealth became a CMS-aligned network and a national leader in adopting FHIR-based standards for secure health data exchange while supporting multiple Centers for Medicare & Medicaid Innovation value-based care programs.

Dr. Kendrick previously served as Senior Counsel for Interoperability to the National Coordinator for Health IT, testified before the U.S. Senate on health data interoperability, and served on the NCQA Board of Directors, including as Chair from 2019 to 2022. He lives in Tulsa, Oklahoma, with his wife, Christie, and their four children.



QINGGONG TANG, PHD

Associate Professor of Stephenson School of Biomedical Engineering (SBME), Endowed Stephenson Professor, The University of Oklahoma

Health Data Utilities as sources of Real World Data

Assessing the structure, function, and viability of human organs remains an important challenge in transplantation, biomedical research, and therapeutic development.

This presentation will first highlight our development and translation of optical coherence tomography and complementary multimodal imaging technologies for evaluating donor organs. By integrating quantitative imaging with histopathology, functional measurements, and clinically relevant outcomes, we aim to identify imaging biomarkers that can improve organ assessment and support informed decision-making.

Building on these imaging studies, I will introduce our human organ perfusion platform, which provides a translational environment for maintaining and studying viable organs outside the body. The platform combines controlled perfusion, multimodal imaging, physiological monitoring, tissue analysis, and computational methods to investigate organ injury, function, therapeutic response, and recovery.

It also creates opportunities to evaluate emerging imaging systems, medical devices, and treatment strategies under clinically relevant conditions. Through collaboration among engineers, clinicians, pathologists, basic scientists, and industry partners, these efforts demonstrate how integrated imaging and organ-perfusion technologies can accelerate the translation of laboratory innovations toward biomedical and clinical applications.

QINGGONG TANG, PHD

Associate Professor of Stephenson School of Biomedical Engineering (SBME), Endowed Stephenson Professor, The University of Oklahoma

About Dr. Tang

Dr. Qinggong Tang is an Associate Professor and Endowed Stephenson Professor in the Stephenson School of Biomedical Engineering at the University of Oklahoma. He directs the Biophotonic Imaging Laboratory and serves as Director of the Medical Imaging Technology Development Core within the NIH-funded Oklahoma Center for Medical Imaging and Cancer Research. He is also Chair of the Translation Stimulation Committee in the Stephenson School of Biomedical Engineering and an Associate Member of the Stephenson Cancer Center.

Dr. Tang's research focuses on developing and translating advanced biomedical imaging technologies for organ transplantation, image-guided interventions, and disease assessment. His laboratory develops optical coherence tomography, polarization-sensitive optical coherence tomography, photoacoustic imaging, ultrasound, and multimodal imaging systems to characterize tissue structure, function, viability, and treatment response. A major emphasis of his work is the evaluation of human donor organs, including the kidney, liver, lung, and heart, with the goal of improving organ assessment and supporting clinical decision-making.

His team has also established a human organ perfusion and imaging platform that integrates controlled perfusion, physiological monitoring, multimodal imaging, tissue analysis, and computational methods. This platform enables clinically relevant studies of organ injury, therapeutic response, and recovery, while supporting the evaluation of emerging imaging systems, medical devices, and treatment strategies. His translational research is strengthened through collaborations with clinicians, pathologists, engineers, basic scientists, organ-procurement organizations, and industry partners.

Dr. Tang is committed to mentoring undergraduate students, graduate students, postdoctoral researchers, and early-career investigators. His work has led to federally funded research programs, multiple patents and patent applications, and entrepreneurial efforts aimed at advancing imaging technologies toward clinical and commercial use. His recent honors include the University of Oklahoma Vice President for Research and Partnerships Award for Excellence in Research in Engineering and Applied Science, the Nancy L. Mergler Faculty Mentor Award for Undergraduate Research, and the OKBioStart Translational Research Leadership Award.



CHRISTIAN EL AMM, MD

*Professor of Surgery, Craniofacial Surgery, Plastic and
Reconstructive Surgery
The University of Oklahoma*

***Surgical Extended Reality: At the intersection of
modeling, optics and physical AI.***

Surgical Extended Reality (SXR), Surgical Robotics and Medical AI are emerging technologies that offer a spectrum of promising Human Augmentations, with potential transformative impact.

Based on our experience developing a pioneering Surgical Extended Reality system, this talk will emphasize how these diverse technologies are interconnected, interdependent, and share the same underlying first principles.



JEFF POTTS, PHD

CTO at Xironetic

CHRISTIAN EL AMM, MD

*Professor of Surgery, Craniofacial Surgery, Plastic and
Reconstructive Surgery
The University of Oklahoma*

About Dr. Amm

Christian El Amm joined OU in 2006. His practice focuses on Reconstructive challenges in congenital and acquired deformities, including craniofacial malformations, trauma, burns and cancer.

He also started the OU Craniofacial Lab that leverages engineering science in reconstruction. Dr El Amm is part of several cross-campus collaborations that involve Computational Modeling, Surgical Extended Reality, and other projects.



MARK M MIMS, MD

*Associate Professor of Otolaryngology - Head and Neck
Surgery, OU Health Campus*

ABSTRACT

Surgical Extended Reality (SXR), Surgical Robotics and Medical AI are emerging technologies that offer a spectrum of promising Human Augmentations, with potential transformative impact.

Based on our experience developing a pioneering Surgical Extended Reality system, this talk will emphasize how these diverse technologies are interconnected, interdependent, and share the same underlying first principles.

MARK M MIMS, MD

*Associate Professor of Otolaryngology – Head and Neck
Surgery, OU Health Campus*

About Dr. Mims

Mark M. Mims, MD, is an Associate Professor of Otolaryngology–Head and Neck Surgery and Plastic Surgery at the University of Oklahoma Health Sciences Center, where he also serves as Medical Director of the OU Physicians ENT Clinic and Associate Program Director for the Otolaryngology residency. He is double board-certified in Otolaryngology–Head and Neck Surgery and Facial Plastic and Reconstructive Surgery, with clinical and academic interests spanning complex facial trauma, reconstructive surgery, facial nerve disorders, wound healing, surgical education, and the development of technologies for care in resource-limited and austere environments.

Dr. Mims is the founder and director of Operation Ukraine at OU Health, a multidisciplinary international surgical education initiative established in response to the war in Ukraine. Through the program, Ukrainian military and civilian surgeons representing multiple surgical specialties have traveled to Oklahoma for advanced training in trauma, reconstruction, and other areas directly relevant to wartime care. The program has also expanded into bilateral educational initiatives focused on translating lessons from modern combat casualty care to both Ukrainian and American surgical teams.

His translational research focuses on identifying clinical problems that can be addressed through robotics, computer vision, and autonomous medical systems. He is a co-inventor of the REACH system, a platform being developed to enable rapid, image-guided vascular access when conventional medical personnel or resources may be limited. His work explores how autonomous and human-supervised technologies can extend critical medical capabilities to battlefield, disaster, mass-casualty, and other challenging environments.

Across his clinical, educational, and research activities, Dr. Mims is particularly interested in bridging the gap between bedside observation and deployable technology—bringing clinicians, engineers, researchers, and end users together to move promising ideas toward practical improvements in patient care.

MODERATORS



DAVID MILLER, PHD

Assistant Professor, Stephenson School of Biomedical Engineering (SBME), Adjunct Assistant Professor, Department of Neurosurgery, Affiliate Member, Stephenson Cancer Center, University of Oklahoma

Dr. David Miller is an Assistant Professor of Neuroengineering at the University of Oklahoma (OU) Biomedical Engineering Department and also holds an adjunct Assistant Professor position with the OU Department of Neurosurgery.

Dr. Miller received his PhD in Biomedical Engineering from the University of Texas at Austin, where he was an NSF Graduate Research Fellow and NIH T32 Fellow in Imaging Science and Informatics.

He completed postdoctoral fellowships at UT Austin, and at Massachusetts General Hospital and Harvard Medical School where he was an NIH U19 trainee. At OU, Dr. Miller leads the Translational Neuroimaging Lab which focuses on developing and translating novel neuroimaging and neuroengineering technology to better understand and treat brain diseases.

Dr. Miller is also co-founder of Dynamic Light, which is commercializing surgical imaging technology for real-time and continuous blood flow visualization.

MODERATORS



ELIZABETH HILE, PT, PHD, NCS, CLT

Associate Professor, Director of Cancer Rehabilitation Sciences Program, The University of Oklahoma Health Campus

Dr. Elizabeth Hile is a Tenured Associate Professor of Rehabilitation Science in the OU Health Campus College of Allied Health and Director of the Cancer Rehabilitation Research Laboratory at the OU Health Stephenson Cancer Center where she also practices as a Board-certified Specialist in Neurological Physical Therapy. Her research focuses on improving the detection and rehabilitation of peripheral neuromotor sequelae that impair balance and gait during cancer treatment. Over the past 10 years, her laboratory has secured funding from the Oklahoma TSET, Presbyterian Health Foundation, and National Cancer Institute P30 pilot funding program to develop a novel portable biomedical device and associated software platform to characterize hallux force generation in diverse clinical and research settings.

She is a primary inventor on the University of Oklahoma patent for the resulting system, Quantification of Hallux Extension (QuHalEx™). In close collaboration with a team of OU Norman engineers, Dr. Hile continues to advance the QuHalEx™ device toward commercialization, with a focus on the reliability and responsiveness of the load-time signal output for detecting, phenotyping, and monitoring motor peripheral neuropathy in cancer survivors. She also studies the device's utility for clinical applications in diabetic peripheral neuropathy and motor neuron disease (MND), the latter as a partnership with podiatrists and translational scientists at the University of Queensland-Brisbane. As a coinvestigator on a dietitian-led NIH R03, she oversees the use of existing human performance technology and clinic-derived muscle images to prospectively quantify sarcopenia during cancer treatment. Dr. Hile has authored more than 20 peer-reviewed publications and has presented her team's work at national and international scientific conferences.

In addition to her research, Dr. Hile is dedicated to mentoring graduate students, postdoctoral fellows, and early-career investigators. She serves on the Cancer Prevention and Control Committee for the National Research Group in Oncology and the Oncology Academy of the American Physical Therapy Association. In the past five years, her contributions to Oncology Rehabilitation were recognized nationally through the Oncology Academy's Research Award and the Jerry F. Walton Service Award.

Dr. Hile earned her Ph.D. in Rehabilitation Sciences from the University of Pittsburgh and completed postdoctoral training in mobility and aging at the Pittsburgh Pepper Center (NIA Older Americans Independence Center). She is dedicated to advancing the university's mission to improve healthcare through collaborative translational research and clinical practice.



LEARNING ACTIVITIES

WORKSHOP

FDA REGULATORY PATHWAY FOR DRUGS



Rainbolt Auditorium 1:45 pm – 2:45 pm

Description: What does it take to move a promising therapeutic from the laboratory into clinical testing? This workshop will explore the regulatory and clinical pathway for new drug development through the perspectives of researchers, clinicians and administrators. Panelists will discuss the steps required to advance a therapeutic candidate toward human studies, including practical knowledge, FDA-Mandated preclinical testing requirements, clinical trial planning, IND design and maintenance, and interactions with the FDA. Through practical examples and firsthand experience, participants will gain a clearer understanding of the decisions and evidence needed to move a scientific discovery toward a therapy that can ultimately reach patients.

Session Objectives:

- Help researchers understand the role of FDA regulatory strategy in drug development.
- Highlight common challenges researchers encounter when transitioning a therapeutic from laboratory research into clinical development.
- Describe the type of preclinical studies needed for FDA allowance of proposed clinical trials.
- Describe the information required in applications for FDA pre-Investigational New Drug (IND) meetings and for INDs.
- Provide practical perspectives from individuals involved in therapeutic development, clinical trials, and patient care.

INSTRUCTORS



Nicole Wilson, PhD, MD, FACS
Pediatric Surgeon, Oklahoma
Children's OU Health



Doris Benbrook, PhD
Associate Director of
Translational Research, OU Health
Stephenson Cancer Center



Kristal Black, MPH, CCRP
Senior Program Administrator for
Quality & Investigator-Initiated Trials
Program, OU Health Stephenson
Cancer Center

ABOUT

ABOUT OUR INSTRUCTORS



Nicole Wilson, PhD, MD, FACS

Pediatric Surgeon, Oklahoma
Children's OU Health

Nicole A. Wilson, PhD, MD, FACS, is a pediatric surgeon and biomedical engineer. She is Associate Professor of Surgery and Vice Chair for Research in the Department of Surgery at the University of Oklahoma Health Sciences Center, where she also directs OU CARES, the Center for the Advancement of Research Excellence in Surgery. She holds a graduate faculty appointment in biomedical engineering and is board certified in both general and pediatric surgery.

Dr. Wilson trained as an engineer before she trained as a surgeon, and her research lives at that intersection. Her NIH-funded lab studies post-operative abdominal adhesions and antimicrobial photodynamic therapy, and builds imaging and computational tools that move discoveries toward patients. She is the sponsor-investigator on an active FDA Investigational New Drug application, repurposing methylene blue as a light-activated therapy for infected burn wounds.



Kristal Black, MPH, CCRP

Senior Program Administrator for Quality &
Investigator-Initiated Trials Program, OU
Health Stephenson Cancer Center

Kristal Black is the Senior Program Administrator at the University of Oklahoma Health Sciences Center Stephenson Cancer Center Clinical Trials Office. Her expertise focuses on FDA regulatory applications and early phase drug development. Over the past 16 years, she has provided knowledge and expertise in regulatory affairs and clinical research operations to ensure the best quality in clinical research studies and project management in academia and industry. She assists with interdisciplinary collaborations with colleagues from CMC, translational and clinical development in early stage investigational drugs and clinical trial design. She has submitted multiple IND applications to FDA for early-stage oncology clinical trials and continues to assist investigators with their IND submissions.

WORKSHOP

IP & FDA REGULATORY CONSIDERATIONS: MEDICAL DEVICES & SOFTWARE

Basement Room B 1:45 pm – 2:45 pm



Description: Advancing a medical device or software innovation from research to clinical use requires early consideration of both intellectual property and regulatory strategy. This session brings together experts in IP, FDA regulation, clinical trials, and clinical practice to explore key considerations for protecting innovations and navigating the path toward clinical and commercial application. Through practical perspectives and real-world experience, panelists will highlight how early decisions around IP protection, regulatory requirements, validation, and clinical evidence can help researchers build a stronger pathway toward translation.

Session Objectives:

- Understand key IP and FDA regulatory considerations for medical devices and software.
- Explore how early IP and regulatory planning can support successful translation and commercialization.
- Recognize how intended use, product claims, and risk can influence regulatory pathways.
- Identify strategies for protecting innovations while advancing research and development.
- Learn when to engage IP, regulatory, clinical, and commercialization experts throughout the translational process.
- Help researchers understand when to engage regulatory professionals, clinicians, and clinical trial experts.

Moderator



Elizabeth Hile, PT, PhD, NCS, CLT

*Associate Professor, Program
Director, PhD Program, Director,
Cancer Rehabilitation Services and
Research Lab at OU Stephenson
Cancer Center*

Speaker



Kelly Kucharczyk

*Principal Consultant,
Project Medtech*

Panelist



David Miller, PhD

*Assistant Professor, Stephenson School of
Biomedical Engineering (SBME), Adjunct
Assistant Professor, Department of
Neurosurgery, Affiliate Member, Stephenson
Cancer Center, University of Oklahoma*



Kelly Kucharczyk

*Principal Consultant,
Project Medtech*

Kelly Kucharczyk is a Principal Consultant with 17 years in the industry, who is passionate about partnering with startups to develop get-to-market strategies that integrate the complexities of regulatory, reimbursement, and commercialization. Passionate about the brilliant minds and groundbreaking technologies that define the industry, she serves as a strategic ally helping companies intentionally scale for sustainable growth or position for a successful exit. With expertise in global market entry and FDA negotiations, Kelly is highly skilled in building "acquisition-ready" Quality Management Systems (QMS), Design History Files (DHF), and regulatory submissions. Her leadership background includes serving as a Director at NAMSA, where she guided global teams across Regulatory, Quality, and Medical Writing. Prior to this, Kelly spent nearly a decade at Stryker, driving regulatory strategies for new product development and managing complex M&A activities from initial due diligence through full post-acquisition integration. Outside of the medtech world, she is an outdoor enthusiast and avid gardener who can frequently be found foraging for wild foods, experimenting with new recipes in the kitchen, or collaborating on "toddler art" masterpieces.

LEARNING ACTIVITY

HOW TO PITCH YOUR INNOVATION WITH RON BOLEN, JD, MBA

Rainbolt Auditorium 4:15 pm – 5:30 pm



Description:

Effectively communicating an innovation is essential for building interest, gaining support, and creating new opportunities. This interactive workshop will introduce participants to the fundamentals of developing a clear and compelling pitch for their research or innovation. Through guided activities and hands-on practice, participants will develop and refine their own pitch, with opportunities to practice their delivery and receive constructive feedback.

Session Objectives:

- Understand the key elements of a clear and compelling pitch.
- Learn how to communicate complex research and innovation to diverse audiences.
- Develop and refine a concise pitch that communicates the problem, solution, and potential impact.
- Practice pitching in an interactive environment and receive constructive feedback.
- Build confidence in communicating research and innovation to potential partners, collaborators, funders, and other stakeholders.

INSTRUCTOR



Ronald E Bolen, JD, MBA

*Associate Professor of Entrepreneurship,
Michael F. Price College of Business, The
University of Oklahoma*

Ron Bolen, serves as an Associate Professor at the OU Price College of Business Tom Love Division of Entrepreneurship & Economic Development. Additionally, Ron is the founder and a Managing Director of da3co, LLC, a global energy consultancy. Ron has over 25 years of experience in launching, financing and advising new ventures. He has over 15 years direct experience in venture capital and private equity investing in the same space. His work in energy private equity includes both the power and oil & gas segments.

Ron was, most recently, Managing Director of HFBE Investment Banking leading that firm's energy practice. Prior to joining HFBE, Ron was Managing Director of The InvestLinc Group, managing the energy practice there. InvestLinc Group acquired Invictus Energy Group LLC, an international power project finance venture specializing in power optimization opportunities which Ron co-founded.

Ron was also an investment officer for DOE Enterprises, the venture capital subsidiary of DOE. There, Ron was responsible for making equity investments in early-stage energy technology companies.

Preceding his time at DOE Enterprises, Ron was a Senior Director of Portland General Electric where he established the company's private equity investment group as well as oversaw the non-regulated affiliate organization.

Ron's professional efforts have had a significant international flavor with extensive work in South and Southeast Asia, Latin America and the Former Soviet Union.

Ron has served as a venture capitalist, entrepreneur investment banker, and lawyer. Ron holds a BBA in Finance as well as a Juris Doctor from The University of Oklahoma. He also received a Masters of Business Administration with majors in both Finance and Taxation from New York University.

GROUP NETWORKING, TRANSLATIONAL CONNECTIONS

Basement Room B 4:15 pm – 5:30 pm



Description:

Translation is rarely accomplished alone. This interactive networking session is designed to connect participants with the people, expertise, and resources that can help move research forward. Topic-focused tables will bring together experts from across the university and Oklahoma innovation ecosystem to discuss areas such as intellectual property, funding, commercialization, regulatory strategy, manufacturing, and clinical trials. Dedicated clinician and engineering tables will also provide opportunities for researchers to make new connections and explore potential collaborations. Participants are encouraged to move freely between tables, ask questions, share their work, and connect with the people and resources most relevant to their translational needs.

Session Objectives:

- Connect participants with experts, resources, and potential collaborators across the translational ecosystem.
- Provide an informal environment for participants to ask questions and discuss their individual research needs.
- Create opportunities for researchers to connect directly with clinicians and engineers and explore potential collaborations.
- Help participants identify resources, expertise, and next steps that can support the advancement of their research.
- Encourage new cross-disciplinary relationships and connections across the university and broaden Oklahoma innovation community.

GROUP NETWORKING, TRANSLATIONAL CONNECTIONS

Basement Room B 4:15 pm – 5:30 pm



DISCUSSION TABLES

Andrew Pollock	·Intellectual Property: Patents, copyright, and protecting innovations
Yingtao Liu, PhD	·Manufacturing & Prototyping: Turning concepts into physical prototypes
Vy Trinh	·GMP Drug Production: Manufacturing considerations for therapeutic development
Gina L. McMillen DVM, PhD	·Commercialization & Licensing: Pathways for bringing university innovations to market
H. Anne Pereira, PhD	·Academic Entrepreneurship: Starting and operating a company while working in academia
Jaime Hough, PhD	·Finding Funding: Identifying funding opportunities and resources for translational research
Doris Mangiaracina Benbrook, PhD	·Regulatory & FDA: Navigating regulatory considerations and planning
John R. Clegg, PhD	·Collaborating with Engineers: Building productive interdisciplinary collaborations
Faizah Bhatti, MD	·Collaborating with Clinicians: Connecting research with clinical needs and expertise
Franklin Hays, PhD	·Finding University Collaborators: Using university platforms and resources to identify expertise
Susanna V. Ulahannan, MD, MEd	·Developing Clinical Trials: Moving research toward clinical evaluation and study development
Muhammad Furqan, MD	



ANDREW POLLOCK, MBA

Managing Director, Office of Technology Commercialization

Andrew Pollock is the Managing Director for the Office of Technology Commercialization, having joined the University of Oklahoma in 2014. Andrew manages the overall day to day operations of OTC and serves as a technology manager for non-life science related inventions from the Norman and HSC campuses. In these roles Andrew works directly with faculty, other members of OTC and outside counsel to formulate protection strategies for intellectual property developed at the University, as well as engaging with industry to amplify the impact of these discoveries through commercialization. He has lead multiple initiatives to improve the outcomes of University IP including the recent launch of the upfront licensing of IP in research agreements.

Prior to joining the OTC Andrew was involved in an early-stage startup in the Oklahoma City area focused on external fixation prosthetics, where he built upon his background in Biomedical Engineering, and cultivated experience in grant writing, clinical study design, regulatory filings, venture financing and product development, leading to the launch of 4 new products. He is a proud alumnus of OU with a 2002 degree in Zoology.



YINGTAO LIU, PHD

*Director, Sooner Advanced Manufacturing Laboratory (SAML),
Professor, Benjamin H. Perkinson Chair & William H. Barkow
Presidential Professor, School of Aerospace and
Mechanical Engineering, The University of Oklahoma*

Dr. Yingtao Liu is a Professor and Perkinson Chair in the School of Aerospace and Mechanical Engineering (AME) at the University of Oklahoma (OU). He also serves as the Graduate Liaison for AME and directs the Sooner Advanced Manufacturing Laboratory (SAML) at OU.

Dr. Liu's research focuses on advanced manufacturing and materials, spanning metal, polymer, and composite additive manufacturing; multifunctional materials; in situ sensing and monitoring; and digital twin frameworks for structural and functional components. In the realm of biomedical and translational engineering, his work bridges materials science and practical health applications, focusing on advanced biomaterials processing, biocompatible and structural alloy fabrication, integrated sensor architectures for diagnostic monitoring, and high-fidelity testing methodologies.

He has published extensively in premier journals and conference proceedings across mechanical engineering, materials science, and multidisciplinary design. Throughout his career, Dr. Liu has led multiple research projects supported by federal agencies and industry partners, advancing cutting-edge manufacturing solutions, material characterization, and cross-disciplinary translational technologies.



VY TRINH

Cytovance Biologics: Sr. Manager of Analytical Development

Vy Trinh obtained her PhD in Biochemistry at the University of Oklahoma in 2013. Vy currently works at Cytovance Biologics as the senior manager of the Analytical Development Department. As a senior manager, she serves as a subject matter expert for analytical services for potential clients requesting development and manufacturing services. Over her 4 years at Cytovance Biologics, Vy has helped transfer, develop, and qualify analytical methods for early- and late-stage clients.

The Analytical Development department is a subdivision of the Research and Development Department that supports small-scale analysis.



GINA L. MCMILLEN DVM, PHD

*Director, Office of Technology Commercialization
University of Oklahoma Health Campus*

Dr. Gina McMillen oversees the management of OU's Life Sciences Intellectual Property Portfolio. In this role she works with inventors and other members of OTC to develop an IP protection plan and commercialization strategy to move University innovations from the laboratory to the market. In addition to identifying technologies with commercial potential, she leads marketing efforts, licensing negotiations, and monitors compliance of licensees with contractual obligations.

Prior to joining OTC in 2008, Gina worked in the Veterinary biotechnology industry as a product development scientist for companion animal vaccines and as a clinical trials coordinator for veterinary biologics and diagnostics. She also has experience in the start-up space having worked with two early-stage Oklahoma companies where she served as PI and secured SBIR/STTR grant funding.

Dr. McMillen is a proud graduate of Kansas State University, having earned her DVM in 1989 and her PhD in Microbiology in 1995.



JAIME HOUGH, PHD

Outreach & Recruitment Manager, OKCatalyst

Jaime serves as the Outreach & Recruitment Manager for OK Catalyst and in this role helps connect innovators in the OK entrepreneurial ecosystem to funding resources available through the SBIR/STR programs. Jaime earned her PhD in American Studies from Purdue University.



FRANKLIN HAYS, PHD

Associate Dean for Research, Associate Professor, Department of Nutritional Sciences, Director, Redox and Nutritional Biochemistry Laboratory, Member, Harold Hamm Diabetes Center, Member, Data Institute for Societal Challenges, Associate Member, Stephenson Cancer Center



JOHN R. CLEGG, PHD

William H. Barlow Presidential Professor and an Assistant Professor of Biomedical Engineering at the University of Oklahoma

Dr. John R. Clegg is a William H. Barlow Presidential Professor and an Assistant Professor of Biomedical Engineering at the University of Oklahoma. He is concurrently an Adjunct Assistant Professor of Neurosurgery, a member of the Harold Hamm Diabetes Center, and a member of the Stephenson Cancer Center at the University of Oklahoma Health Campus. Dr. Clegg obtained his PhD from the University of Texas, Austin in the laboratory of Prof. Nicholas Peppas and completed postdoctoral training at Harvard University under Prof. Samir Mitragotri. His lab works at the frontier of “Neuro-Immuno-Engineering”, where engineering principles are applied to address basic and translational problems in neuro-immunology. To that aim, his team develops brain-inspired injectable hydrogels (i.e., “ParenchyGel”) for applications such as neurotrauma and brain tumors.

His team also utilizes polymeric and hydrogel nanoparticles to influence innate immune cell phenotype, with application in pre-conditioning donor immune cells for adoptive transfer or modulation of tissue-resident macrophages in sites of accumulation following systemic delivery of nanoparticle suspensions. These therapeutics are evaluated in cell culture, human organoid/spheroid, and rodent models. Dr. Clegg’s research has been recognized with several awards, including the NIH-NIGMS Maximizing Investigators’ Research Award, NSF GRFP, and the best paper award from the Controlled Release Society. He is an active member of the Biomedical Engineering Society (BMES), Society for Biomaterials (SFB), and Controlled Release Society (CRS).



FAIZAH BHATTI, MD

Professor of Pediatrics, Adjunct Associate Professor of Ophthalmology, Adjunct Professor Cell Biology, Member Center for Neuroscience, The University of Oklahoma

Dr. Bhatti is a clinical neonatologist and a post-doctoral trained basic, translational and clinical investigator. As an independently funded clinician scientist, she committed to improving outcomes of premature infants by understanding how blood vessel development is altered in inflammatory diseases. In particular, the focus of her research program is to elucidate the role of inflammation and surfactant proteins in abnormal blood vessel growth in the retina as it relates to retinopathy of prematurity.



SUSANNA V. ULAHANNAN, MD, MMED

Director, Phase I Clinical Trials Program

Susanna Ulahannan, MD, MMed, is an associate professor in the Section of Hematology/Oncology. She specializes in the treatment of gastrointestinal cancers and the conduct of Phase I clinical trials. She serves as Director of the Oklahoma TSET Phase I Program and as Chair of the institutional Protocol Review and Monitoring Committee. She is also the Chair of the Early Phase Disease Site Committee at the Stephenson Cancer Center.

At the national level, Dr. Ulahannan serves as Chair of the NRG Oncology Immunotherapy Subcommittee and as the institutional Principal Investigator for NRG in medical oncology. She is designated as an NCI Early-Stage Clinical Investigator. She is a member of the American Society of Clinical Oncology (ASCO), where she serves on the Gastrointestinal Guideline Advisory Board and the TAPUR/ASCO Data and Safety Monitoring Board. Dr. Ulahannan completed her hematology-oncology fellowship at the National Cancer Institute/National Institutes of Health in Bethesda, Maryland, and her internal medicine residency at Johns Hopkins University–Sinai Hospital in Baltimore, Maryland. She earned her medical degree and a Master of Medicine from Sahlgrenska Academy, University of Gothenburg, Sweden.



MUHAMMAD FURQAN, MD

Hematologist/Oncologist Stephenson Cancer Center, The University of Oklahoma Health Campus

Muhammad Furqan, M.D., is a board-certified medical oncologist and Professor of Medicine at the University of Oklahoma. He serves as Associate Director of Clinical Research at Stephenson Cancer Center, Associate Section Chief for Research in Solid Tumor Oncology, and holds the Mai Eager Anderson Endowed Chair in Clinical Trials. His clinical and research interests include thoracic and head and neck malignancies, early-phase clinical trials, and drug development.

Dr. Furqan is an active member of the Alliance Respiratory Committee and NRG Head and Neck Cancer Committee and serves on the AACI Clinical Research Innovation Steering Committee. He has led multiple investigator-initiated clinical trials supported by the NCI and industry and has served as site principal investigator for numerous early- through late-phase clinical trials. He currently serves as the national principal investigator of A082302, a Phase III trial evaluating immunotherapy in early-stage non-small cell lung cancer. In recognition of his contributions to NCI-funded clinical research, he received the NCI Cancer Clinical Investigator Team Leadership Award in 2020.



PANELS

SUCCESS IN FINDING TRANSLATIONAL RESOURCES PANEL

AUDITORIUM 11:00 AM – 11:30 AM

PANELISTS



Qinggong Tang, PhD

Associate Professor of Stephenson School of Biomedical Engineering (SBME), Endowed Stephenson Professor, The University of Oklahoma



Doris Mangiaracina Benbrook, PhD

Associate Director for Translational Research, Stephenson Cancer Center, Presidential Professor, Presbyterian Health Foundation Member, Harold Hamm Diabetes Center, The University of Oklahoma Health Campus



H. Anne Pereira, PhD

Regents' Professor, David L. Boren Professor & Henry Zarrow Presidential Professor, Department of Pharmaceutical Sciences, The University of Oklahoma Health Campus



David C. Kendrick, MD, MPH

Chair Department of Medical Informatics, OU School of Community Medicine, Associate Vice Provost for Strategic Planning, The University of Oklahoma Health Campus



Wei R. Chen, PhD

Stephenson Chair, Professor, and Director, Stephenson School of Biomedical Engineering, The University of Oklahoma



Elizabeth Hile, PT, PhD, NCS, CLT

Associate Professor, Director of Cancer Rehabilitation Sciences Program, The University of Oklahoma Health Campus

Description:

Building on the morning's founder presentations, this interactive session will explore the people, resources, and partnerships that help move research discoveries toward real-world impact.

Through a moderated discussion and audience Q&A, founders will share lessons from their translational journeys, while commercialization experts from across the university and innovation ecosystem will offer practical guidance on navigating the translation process and accessing available resources.

Session Objectives:

- Provide participants with an opportunity to ask questions of the morning's presenters and commercialization experts.
- Identify practical first steps for moving a discovery toward clinical or commercial application.
- Highlight resources available across the university and the broader Oklahoma innovation ecosystem.
- Share best practices and common lessons from founders' translational journeys.
- Identify gaps where additional resources or support may be needed.

MODERATORS



Huynh Le

Assistant Director, OKBioStart, The University of Oklahoma



Andrew Pollock, MBA

Managing Director, Office of Technology Commercialization

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HUYNH LE

Assistant Director, OKBioStart, The University of Oklahoma

Huynh Le is Assistant Director of OKBioStart at the University of Oklahoma, where she leads innovation and commercialization programs designed to help early-stage life science technologies advance toward startup formation and market readiness. In this role, she oversees the Concept and Launch programs, manages grant-funded initiatives, and works closely with researchers, entrepreneurs, industry partners, and Oklahoma's broader innovation ecosystem to support technology development, business strategy, funding, and commercialization.

Prior to joining OKBioStart, Huynh built extensive experience in biotechnology research, manufacturing, and biologics process development. At Cytovance Biologics, she served as Mammalian Bioprocess Manager, leading process development activities for biologics including monoclonal antibodies, enzymes, bispecifics, vaccines, and fusion proteins and supporting the translation of processes from development into manufacturing. She previously held manufacturing and cell culture leadership roles at Immuno-Mycologics (IMMY), where she worked with monoclonal and polyclonal antibodies and managed operations within regulated laboratory environments.

Huynh began her career in scientific research at the University of Oklahoma, studying microbial physiology, syntrophic metabolism, and microbiologically influenced corrosion. Her research contributed to multiple peer-reviewed publications in microbiology. She earned both her M.S. and B.S. in Microbiology from the University of Oklahoma.

COMMERCIALIZATION AND ENTREPRENEURSHIP PATHWAYS PANEL

AUDITORIUM 12:15 PM – 1:30 PM

PANELISTS



Lara Ferry, PhD

Professor of Biology, Vice President and Chief Research Innovation Officers, The University of Oklahoma



Mike Moradi

Co-Founder & General Partner, Cortado Ventures



Carol Curtis, PhD

Chief Executive Officer, Founder, Cadenza Bio



Paul DeAngelis, PhD

Presidential Professor, Department of Biochemistry & Physiology, The University of Oklahoma



Thomas C. Kupiec, PhD

CEO of ARL Bio Pharma and DNA Solutions Inc.

Description: This interactive panel will explore the pathways available to move university research from discovery to real-world impact. Panelists will share practical perspectives on evaluating commercial potential, protecting intellectual property, securing funding, building entrepreneurial teams, forming industry partnerships, and determining whether licensing or startup formation is the right path. The session will emphasize candid discussion and audience engagement rather than formal presentations.

Purpose & Intent: The purpose of this session is to demystify the commercialization process and help faculty, researchers, and innovators better understand how to advance promising discoveries beyond the laboratory. The discussion will highlight university and statewide resources, lessons from experienced entrepreneurs, and the perspectives of investors and commercialization professionals.

Expected Outcomes: Participants will leave the session with:

- A clearer understanding of the steps from discovery through commercialization
- Greater awareness of university, state, and external commercialization resources
- Practical insight into licensing, startup formation, funding, team development, and industry partnerships
- An understanding of common commercialization barriers and how to address them
- Actionable next steps for assessing and advancing their own discoveries

MODERATOR



Nicholete Davis, AuD, MBA

Executive Director, OKBioStart, The University of Oklahoma



CAROL CURTIS, PHD

Chief Executive Officer, Founder, Cadenza Bio

Dr. Carol Curtis holds a PhD in molecular and integrative physiology from the University of Illinois at Urbana-Champaign. She spent 4 years as a postdoctoral researcher at the Oklahoma Medical Research Foundation before leaving academic research to focus her career on early-stage venture development. Dr. Curtis managed operations, supervised R&D, interacted with FDA, and successfully secured both non-dilutive and investor financing as part of 4 preclinical drug development companies prior to moving into venture capital with i2E (now Plains Ventures), an Oklahoma City-based Seed to Series A venture firm. In 5 years with i2E, Dr. Curtis led financing for 18 early-stage companies in rounds totaling \$46M and served as a board member to 4 Seed to Series A life science companies.

In 2021, Dr. Curtis launched an independent consulting practice focused on supporting innovative scientists, early-stage life science ventures and investors. Over the past five years, she supported the launch of an emerging venture capital firm, built a life science venture accelerator, and launched Cadenza Bio, Inc., a biotech startup focused on development of small molecules for demyelinating and inflammatory disease. As Co-Founder and CEO of Cadenza Bio, she leads scientific and operational strategy, with a primary focus on advancing IND-enabling nonclinical safety and toxicology programs for the company's Multiple Sclerosis program and progressing preclinical development in endometriosis and menopause.



MIKE MORADI

Co-Founder & General Partner, Cortado Ventures

Mr. Moradi is a VC investor and entrepreneur, who left dental school to start his first company. That company failed, but was followed by many successful ventures in nanotechnology and biopharmaceuticals. Mike is a co-founder and general partner at Cortado Ventures, a top-decile early-stage VC firm. Mike is a co-founder of Sensulin, a diabetes-focused biopharma company, and was previously a co-founder and CEO of a pharmaceutical company targeting ocular complications of diabetes. He also co-founded a specialty Contract Research Organization (eyeCRO), which was the #264 fastest growing private company in the Inc. 500. He co-founded Nanopolaris (now Unidym), which was acquired in 2011 by a publicly traded South Korean company for \$145M USD, and NanoSource, which was acquired by DuPont in 2002, largely considered the first major acquisition in the nanotech space.

Mike was a trustee for the \$14B USD Oklahoma Pension Employees Retirement System (OPERS), and a member of the audit committee. He is an advisory board member for the Health Finance Institute, a director of Grand Founders, a regular attendee of the flagship TED conference, and the World Economic Forum in Davos. He holds a degree in biochemistry from the University of Oklahoma, with additional coursework from the John F. Kennedy School at Harvard University, Oxford University's Saïd Business School, Cambridge University's Judge Business School, the Lee Kuan Yew School of Public Policy at the National University of Singapore, and the University of Cape Town's Graduate School of Business. Mike was named to the World Economic Forum's (WEF) 2017-2022 class of Young Global Leaders (YGL), a 2025 Karman Pioneer, and also a 2024 Kauffman Fellow (KF Class 29). Mike is also a displaced person whose family settled in the US when he was very young.



PAUL DEANGELIS, PHD

Presidential Professor, Department of Biochemistry & Physiology, The University of Oklahoma

Paul L. DeAngelis (PhD, Univ of CA, Irvine, 1990) is a Presidential Professor at the University of Oklahoma in the Dept. of Biochemistry & Physiology. He has been working in the glycobiology field since 1981 using the tools of biochemistry and molecular biology on microbial and vertebrate polysaccharides and their anabolic enzymes or binding partners. His laboratory has discovered several new classes enzymes that polymerize an essential class of polysaccharides called glycosaminoglycans [GAGs]; these sugar polymers are used as medical devices, biomaterials, and therapeutics with \$ billion markets. Our basic science discoveries led to applied research focused on harnessing these enzymes to make defined, useful GAG polysaccharides in a series of patented and patent pending inventions.

Over the years, several GAG synthesis technologies have been licensed from OU to biotech companies including 4 start-ups based in OK City, Novo Nordisk, and Novozymes. He has developed several medical applications for GAGs including drug delivery technology (HEPtuneä), a targeted nanoparticle delivery of cargo (mRNA, siRNA, etc) to immune cells (HEPboostTM), and a new potent therapeutic class of inhibitor molecules for fibrotic disease, antiviral and oncological treatments (AlphaBlokTM).



THOMAS C. KUPIEC, PHD

CEO of ARL Bio Pharma and DNA Solutions Inc.

Dr. Thomas C. Kupiec is the CEO of ARL Bio Pharma and DNA Solutions Inc. Dr. Kupiec received his Ph.D in Pharmaceutical Sciences from the University of Oklahoma Health Sciences Center College of Pharmacy. He currently serves as a graduate faculty member at the OU Health Sciences Center and has held teaching appointments at several universities. He has published numerous articles and abstracts in a variety of fields including pharmaceutical sciences, forensic sciences and pharmacogenomics. Dr. Kupiec is often requested as a speaker at national pharmaceutical conferences and has spoken at ASHP, IACP, ACA and for state pharmaceutical associations.

Dr. Kupiec's vision of entrepreneurial research is manifested by his responsibilities at DNA Solutions and ARL, which include supplying technical expertise and business development in the pharmaceutical and forensic fields. Additionally, Dr. Kupiec offers consultation services for a variety of pharmacy related fields as well as expert witness testimony in the fields of forensic toxicology and pharmaceutical sciences. He has testified in over a hundred cases, both civil and criminal, in federal, state and municipal courts.

His professional experience includes various appointments at Northwest Toxicology Inc. (NWT), the Federal Aviation Administration (FAA) and the Oklahoma City Police Department. Dr. Kupiec is affiliated with numerous professional organizations, including the American Academy of Forensic Sciences, American Association of Pharmaceutical Scientists, American Society of Health-System Pharmacists, the Society of Forensic.



NICHOLETE DAVIS, AUD, MBA

Executive Director, OKBioStart, The University of Oklahoma

Dr. Nicholete Davis brings more than 15 years of healthcare industry, commercialization, and business development experience spanning medical technology, healthcare software, and life sciences. She currently serves as Executive Director of OKBioStart at the University of Oklahoma, where she leads programs supporting the translation and commercialization of university and research-based innovations, including market assessment, customer discovery, business model development, industry engagement, and commercialization strategy. Prior to joining OU, Dr. Davis held senior commercial leadership roles in healthcare technology and software companies, including Flywheel.io, an AI-enabled medical imaging software platform, and Whisper.ai, a venture-backed healthcare technology company. Her industry experience includes go-to-market strategy, enterprise sales, strategic partnerships, product commercialization, and scaling emerging healthcare technologies. Dr. Davis holds an MBA in Healthcare Administration and a Doctor of Audiology.



ROUNDTABLES

CLINICAL RESEARCHER ENGAGEMENT AND BARRIERS ROUNDTABLE CROSS-DISCIPLINARY TRANSLATIONAL COLLABORATION ROUNDTABLE



BASEMENT 12:15 PM – 1:30 PM

Description: This facilitated roundtable will bring together clinical researchers, physician-scientists, basic scientists, translational investigators, research administrators, and institutional leaders to identify opportunities for strengthening translational research across the University of Oklahoma. Participants will engage in two sequential small-group discussions focused on clinical research engagement and cross-disciplinary collaboration, followed by whole-group reporting and identification of shared priorities.

Purpose & Intent: The purpose of this session is to better understand the institutional, operational, and cultural barriers that limit translational research at OU and to develop practical recommendations for addressing them. Combining the two discussion themes into a single session will encourage dialogue across disciplines, colleges, campuses, and administrative units while helping participants identify opportunities for coordinated action.

The session is intended to move beyond identifying challenges by generating specific solutions, resources, and institutional changes that could improve clinical engagement and cross-disciplinary collaboration.

Expected Outcomes: Participants will:

- Identify the most significant barriers to clinical participation in translational research
- Examine challenges affecting collaboration across disciplines, colleges, and campuses
- Highlight successful collaboration models that OU could expand or replicate
- Identify missing resources, infrastructure, incentives, or institutional support
- Recommend practical actions to strengthen OU's translational research ecosystem
- Establish shared priorities that can inform future institutional initiatives

Moderator: Timothy VanWagoner, Ph.D., OSCTR Program Coordinator and Associate Director & Javier Jo, PhD, Director, NIH-P20 COBRE Oklahoma Center of Medical Imaging for Translational Cancer Research

Roundtable Leaders: David Kendrick, MD, Kelly Standifer, PhD, Taha Al-Juhaishi, MD, MBA, Michael Businelle, PhD, Karina Schreffler, PhD, Sharukh Khajotia, BDS, MS, Jeanie B. Tryggstad, MD, Gregory L. Blakey, MD, Martha K. Ogilvie, Ph.D, Judith James, MD, PhD, Jennifer Holter Chakrabarty, MD, Raid Aljumaily, MD, Samantha Harbert, CIP



TIMOTHY VANWAGONER, PH.D.

*OSCTR Program Coordinator Professor, Department of Pediatrics,
OU College of Medicine, Senior Associate Director, Oklahoma Clinical
and Translational Science Institute, Co-Program Leader, Cancer
Prevention and Control, OU Health Stephenson Cancer Center,
University of Oklahoma Health Campus*

Dr. VanWagoner is a Professor in the OU Health Department of Pediatrics and serves as the Senior Associate Director of the Oklahoma Clinical and Translational Science Institute (OCTSI). He has been involved in biomedical and patient-oriented research for more than 30 years. He serves in leadership capacities for a number of institutional awards related to building translational research infrastructure and increasing community engagement efforts, including as the Deputy Director and the Program Coordinator of the Oklahoma Shared Clinical and Translational Resources (OSCTR) since its inception. He also serves as a Multi-Principal Investigator (MPI) of two NIH-funded network hubs to increase rural and tribal participation in NIH-funded pediatric and adult clinical trials and directs the OCTSI Clinical Trials Unit that employs a cadre of clinical research nurses and coordinators to assist campus investigators to conduct clinical trials and research. He serves as the site PI in the NIH RECOVER Post-Acute Sequelae of COVID studies and the NIH-funded iSCORE-RC collaborative designed to increase the number of clinical trials and provide clinical research staff training opportunities. He also serves as an MPI on an NIH Center of Excellence grant to research Indigenous maternal morbidity and mortality. He is also the co-Program Leader for Cancer Prevention and Control at the OU Health Stephenson Cancer Center as well as a co-Investigator on other ongoing NIH studies to increase cancer screening in Oklahoma and improving cancer outcomes in American Indian populations.



JAVIER JO, PH.D.

*Ted and Cuba Webb Presidential Professor
Director, NIH-P20 COBRE Oklahoma Center of Medical Imaging for
Translational Cancer Research*

Javier A. Jo, Ph.D., received the B.S. in electrical engineering from the Pontificia Universidad Católica del Peru in 1996. In 1998, he joined the University of Southern California, Los Angeles, obtaining the M.S. in electrical engineering (signal and image processing) and the Ph.D. in biomedical engineering (physiological modeling), in 2000 and 2002, respectively. In 2002, Jo changed his research focus to the field of biophotonics and has continued working in this field since then. Prior to starting his academic appointments, he was a postdoctoral fellow within the Department of Surgery, at Cedars-Sinai Medical Center, Los Angeles (2002–2005), and a project scientist within the Department of Biomedical Engineering at the University of California, Davis (2006). In 2006, he joined the faculty of the Department of Biomedical Engineering at Texas A&M University, where he established the Laboratory for Optical Diagnosis and Imaging (LODI). Jo joined the faculty of the School of Electrical and Computer Engineering at the University of Oklahoma in 2019.

The overall mission of Jo's research program is to develop optical sensing and imaging technologies that will impact how we: (1) study pathophysiological mechanism underlying major human diseases; and (2) clinically manage patients suffering from these diseases. While most academic labs in the field of biomedical imaging focus on either instrumentation development or computational imaging science,

Javier Jo leads a very unique research program dedicated to addressing major unmet needs in both biomedical research and clinical practice through the design, development, and validation of both optical spectroscopy/imaging instrumentation and computational tools and methods for the nondestructive, non or minimally invasive morphological, molecular and physiological characterization of biological and engineered tissues at multiple spatial and temporal scales. He has received over \$8 million in external research funding as a PI/Co-PI from the National Institute of Health (NIH), the American Heart Association (AHA), the Cancer Prevention and Research Institute of Texas (CPRIT), and the Qatar National Research Fund (QNRF).

Jo teaches graduate and undergraduate courses in biomedical instrumentation, signal/image processing, and statistics and serves as a topical editor for OSA Optics Letters, and as a standing member of the NIH Clinical Translational Imaging Science (CTIS) Study Section.



KELLY STANDIFER, PHD

Chair, Pharmaceutical Sciences, Richard T. Anderson Chair in Neuroscience, College of Pharmacy, David L. Boren Professor, The University of Oklahoma Health Campus

Dr. Kelly Standifer is a David L. Boren Professor of Pharmacology, holds the Richard T. Anderson Chair in Neuroscience and serves as Chair of the Department of Pharmaceutical Sciences at the University of Oklahoma College of Pharmacy. Her research focuses on understanding how the brain responds to physical and emotional trauma to develop therapies for traumatic brain injury (TBI), post-traumatic stress disorder (PTSD) and co-morbid pain and anxiety. Over the past 15 years, her laboratory has secured funding from the Department of Defense to test the ability of novel drugs to reduce the long term effects of brain injury and traumatic stress and to guide the development of better treatments for patients.

Dr. Standifer has authored more than 95 peer-reviewed publications and has presented her work at numerous national and international scientific conferences. She is an active collaborator across multiple disciplines, partnering with clinicians, engineers, and basic scientists to accelerate the development of novel therapeutic strategies that improve patient outcomes.

In addition to her research, Dr. Standifer is dedicated to mentoring graduate students, postdoctoral fellows, and early-career investigators. She serves on several national scientific review panels and professional society committees and has received multiple awards recognizing her contributions to research, innovation, and scientific leadership. Dr. Standifer earned her Ph.D. in Pharmacology from the University of Florida and completed postdoctoral training in molecular pharmacology at Memorial Sloan-Kettering Cancer Center before joining the University of Houston as an Assistant Professor. She joined the faculty at the University of Oklahoma in 2006 as Professor and appointed as Chair in 2009. Her work continues to bridge fundamental discovery with preclinical and translational application, advancing the university's mission to improve healthcare through collaborative research.



TAHA AL-JUHAISHI, MD, MBA

Associate Chief Medical Officer for Cancer Services at OU Health Stephenson Cancer Center and an Associate Professor of Medicine at the University of Oklahoma College of Medicine

Dr. Taha Al-Juhaishi is an attending physician and clinical investigator at the University of Oklahoma Stephenson Cancer Center, the only NCI-designated cancer center in Oklahoma. He currently serves as the Associate Chief Medical Officer of Cancer Services and associate program director for the Hematology and Medical Oncology Fellowship.

In his clinical role, Dr. Al-Juhaishi cares for patients undergoing stem cell transplantation and cell therapy throughout all phases of their treatment journey. He is also actively involved in clinical research, serving as principal investigator on multiple clinical trials and associate member of the OU SCC Cancer Therapeutics Research Program. In addition, he contributes to medical education through his involvement in affiliated training programs.

Dr. Al-Juhaishi earned his medical degree from Weill Cornell Medicine in Qatar. He completed his residency in Internal Medicine and a fellowship in Hematology and Medical Oncology at Virginia Commonwealth University and the VCU Massey Cancer Center. He went on to pursue further specialized training in Stem Cell Transplantation and Cellular Therapies at The University of Texas MD Anderson Cancer Center in Texas..



MICHAEL BUSINELLE, PHD

*Peggy and Charles Stephenson Endowed Chair in Cancer, Professor of Family and Preventive Medicine, The University of Oklahoma
Co-Leader of the Stephenson Cancer Center's Cancer Prevention and Control Program*

Dr. Michael Businelle is the Peggy and Charles Stephenson Endowed Chair in Cancer, a tenured Professor in the Department of Family and Preventive Medicine at the University of Oklahoma, and Co-Leader of the Stephenson Cancer Center's Cancer Prevention and Control Program. He also Co-Directs the TSET Health Promotion Research Center which is staffed by over 145 employees. He is a national leader in the development of smartphone-based assessment and intervention tools. To date, his Insight™ mHealth platform has supported 118 studies including over 60 that were funded by the NIH. Since 2011, he has been the PI, Co-I, consultant, or mentor on 70 studies that have used smartphones to collect data and/or intervene in real-time. He currently leads 4 active NIH-funded randomized controlled trials focused on smoking cessation and/or alcohol cessation/reduction.



KARINA SCHREFFLER, PHD

Associate Dean for Research, Scholarship, & PhD Studies | Fran & Earl Ziegler College of Nursing, Professor and Fran and Earl Ziegler Endowed Chair in Nursing Research, The University of Oklahoma Health Campush

Dr. Karina Shreffler is a Professor of Nursing and Fran and Earl Ziegler Endowed Chair in Nursing Research at the University of Oklahoma and serves as the Associate Dean for Research, Scholarship, and PhD Studies in the Fran and Earl Ziegler College of Nursing. Her research focuses on the consequences of social stressors such as trauma exposure, economic hardship, and childhood adversity for perinatal and reproductive health, mental health, and health behaviors including substance use and medical help-seeking.

Dr. Shreffler is the contact MPI of the Center for Indigenous Resilience, Culture, and Maternal Health (CIRCLE), an NIH-funded Center of Excellence on Maternal Health Research. She also collaborates on maternal health studies funded by NIDA and NIGMS. She has authored more than 100 peer-reviewed publications and has presented her work at numerous national and international scientific conferences.

Dr. Shreffler earned her dual-title Ph.D. in Sociology and Demography from the Pennsylvania State University in 2007 and has been a faculty member at the University of Oklahoma since 2021. Her work advances the university's strategic plan to transform Oklahoma's future through community-engaged research activities that positively impact rural and Tribal Nation communities.



SHARUKH KHAJOTIA, BDS, MS, PHD

Senior Associate Dean for Research and Innovation, College of Dentistry, The University of Oklahoma

Dr. Khajotia received his dental degree from the University of Bombay, India. After practicing for a couple of years, he earned a Masters in Dental Biomaterials at Marquette University, and then earned a Ph.D. degree with Distinction in Oral Biology, with an emphasis in Dental Materials, from the Medical College of Georgia. He taught part-time during his Ph.D. studies. Dr. Khajotia joined the University of Oklahoma College of Dentistry as Assistant Professor and Chair of the Department of Dental Materials, and was subsequently promoted to Associate Professor with Tenure. He was then promoted to the rank of Professor, and was appointed as the Assistant Dean for Research and Advanced Programs before becoming the Associate Dean for Research.

Dr. Khajotia has broad research interests in the fields of Dental Biomaterials and Oral Biology. He also has a strong interest in educating dental and dental hygiene students, graduate students, post-doctoral fellows and junior faculty to become better practitioners via exposure to research, evidence-based practice and critical thinking skills. Dr. Khajotia gained training in the education of adult learners during his Ph.D. studies, and he continues to apply new teaching methodologies and pedagogies in his classroom.



JEANIE B. TRYGGESTAD, MD

Associate Professor of Pediatrics, Diabetes/Endocrinology, The University of Oklahoma Health Campus

Dr. Jeanie B. Tryggestad is a professor of pediatrics in the section of diabetes/endocrinology at the University of Oklahoma Health Sciences Center and holds the Paul and Ruth Jonas Chair in Diabetes/Endocrinology. She received her Bachelor of Science in Biology from Oklahoma Christian University. She completed her medical school education at the University of Oklahoma Health Sciences Center graduating with distinction. Dr. Tryggestad completed her residency in pediatrics and fellowship in pediatric endocrinology at the University of Oklahoma Health Sciences Center as well. She is board certified in pediatrics as well as pediatric endocrinology.

Dr. Tryggestad's clinical research interests are on the impact of youth onset type 2 diabetes on complications. She is the Oklahoma site PI for the NIH-funded DISCOVERY of predictors of type 2 diabetes in youth study. She was a Co-Principle Investigator for the NIH-funded TODAY trial in Oklahoma and served on the Comorbidity Assessment Committee. She is also the MPI for the Oklahoma Pediatric Clinical Trials Network.

Dr. Tryggestad's other research interest focus on the impact of maternal diabetes on the future cardiometabolic health of the offspring and the impact of obesity and diabetes on vascular function. She has been funded through NIH to understand the impact of maternal diabetes on miRNA expression and protein regulation in infants.

Dr. serves as the co-director of the type 2 diabetes comprehensive clinic in youth at OU Children's in Oklahoma City. Her clinical interests are focused on type 1 and type 2 diabetes with a special focus on Native American populations.



GREGORY L. BLAKEY, MD

Chair and Associate Professor, Presbyterian Health Foundation Chair in Pathology, Chief of Pathology Services, OU Health Campus

[Gregory L. Blakey, MD, is a graduate of the University of Oklahoma and the University of Texas Southwestern Medical School. He completed a residency in anatomic and clinical pathology at the University of Oklahoma College of Medicine and a fellowship in molecular genetic pathology at the Baylor College of Medicine.

Dr. Blakey holds the Presbyterian Health Foundation Chair in Pathology and is an associate professor. He has served as chair of OU's Department of Pathology and chief of pathology at OU Health since 2025.

A board-certified anatomic, clinical, and molecular genetic pathologist, Dr. Blakey previously served as vice chief of pathology and director of clinical pathology at OU Health. Subspecializing in molecular oncologic testing, he is an attending physician in the OU Health Molecular Pathology Lab.

Dr. Blakey has taught medical students foundational concepts in the biology of aging and cancer, along with laboratory medicine. He has received over a dozen teaching awards, winning the College of Medicine's Aesculapian Award for Excellence in Teaching three times and the Dewayne Andrews, MD, Excellence in Teaching Award twice. He also lectures allied health, nursing, and physician associate students at the OU Health Campus.

In graduate medical education, Dr. Blakey trains pathology residents in clinical laboratory management and molecular pathology. He was also previously fellowship director.

Dr. Blakey's research interests include computational pathology and translational diagnostics.



MARTHA K. OGILVIE, PH.D.

Senior Associate Vice President for Research at the University of Oklahoma Health Campus

Martha K. Ogilvie, Ph.D., serves as Senior Associate Vice President for Research at the University of Oklahoma, based at the OU Health Campus, where she partners with leadership across OU's campuses to strengthen and align the university's research enterprise. She leads strategic initiatives that improve how research is administered and supported institution-wide, helping advance OVPRP's impact-focused vision for research — including its priorities in health, energy, extreme weather, and national security.

With nearly three decades of experience in academic research administration, biotechnology, and data strategy, Ogilvie has held a series of progressively senior roles at OU Health Campus since 2007, including Associate Vice President for Research, Administrative Director of the Harold Hamm Diabetes Center, and Director of the Intellectual Property Management Office. Over the course of her career, she has provided leadership across research administration, clinical trials and industry contract administration, technology transfer, human research protections, and research infrastructure, consistently building programs and processes that strengthen institutional research capacity and support faculty success.

Ogilvie has chaired the OUHSC Conflict of Interest Committee, co-chaired the Strategic Plan's Pillar on Economic Growth through Research Translation, and served on the Oklahoma Applied Research (OARS) Advisory Committee and the Federal Demonstration Partnership's Transparency Risk Group. Before joining OU Health Campus, she held leadership roles at Celera Genomics/Applied Biosystems and Fannie Mae, and conducted postdoctoral research at the NIH's National Institute of Diabetes and Digestive and Kidney Diseases.

Ogilvie earned a Ph.D. and M.S. in Biochemistry and Molecular Biology from the University of Oklahoma Health Campus and a B.A. in Political Science from Grinnell College.



RAID ALJUMAILY, MD

Professor, Hematology/Oncology, The University of Oklahoma Health Campus

Raid Aljumaily, MD, professor, Hematology/Oncology, joined the OU Health Stephenson Cancer Center in 2014. His interest in thoracic oncology started during his fellowship years at Tufts Medical Center in Boston, where he learned a broad spectrum of methods and approaches to treating thoracic malignancies. Aljumaily continues to build upon this knowledge through utmost patient care and extensive research, particularly, a phase II study of low-dose paclitaxel with timed thoracic radiotherapy followed by adjuvant chemotherapy.

While in East Carolina University, he did a retrospective review of his experience treating lung cancer patients with oligometastatic disease. Dr. Aljumaily's background inspires him to link basic science findings to clinical research ideas for patients with lung cancer.

Dr. Aljumaily is a member of the lung committee of 2 national cooperative groups (SWOG and NRG) and the chair on a national NCI sponsored clinical trial, S1933 for a novel approach to treatment of patients with stage 2 and 3 non-small cell lung cancer.



JUDITH JAMES, MD, PHD

Associate Vice Provost for Clinical and Translational Science and Professor of Medicine and Pathology at the University of Oklahoma Health Campus

Judith A. James, MD, PhD, is Chair of the Arthritis and Clinical Immunology Program and holds the Lou Kerr Chair in Biomedical Research at the Oklahoma Medical Research Foundation. Dr. James is also the Associate Vice Provost for Clinical and Translational Science and Professor of Medicine and Pathology at the University of Oklahoma Health Sciences Center.

Dr. James' research interests focus on understanding the etiology and pathogenesis of lupus, Sjogren's syndrome and related disorders, the evolution and pathogenic mechanisms of autoantibodies in systemic rheumatic disease, and the interplay of genetic risk and environmental responses in systemic autoimmunity. Her work has made seminal contributions to understanding how autoimmune diseases start and the concept of humoral epitope spreading. She has published over 280 articles in journals such as the New England Journal of Medicine, Nature Medicine, Journal of Experimental Medicine, Annals of Rheumatic Disease and Arthritis and Rheumatology for example. Dr. James currently serves as the principal investigator for several large, multi-investigator NIHfunded grants, such as the U54 Oklahoma Shared Clinical & Translational Resources from NIGMS, UM1 Autoimmunity Center of Excellence from NIAID, and P30 Rheumatic Disease Research Cores Center from NIAMS.

Dr. James has conducted lectures for the American College of Rheumatology, International Systemic Lupus Erythematosus Meetings, among others. Dr. James has received several prestigious awards including the Evelyn V. Hess Award from the Lupus Research Foundation, the Stanley J. Korsmeyer award from the American Society for Clinical Investigation, the Presidential Early Career Award for Scientists and Engineers and the Dubois' Award from the American College of Rheumatology. She is a member of NIAMS Council with the National Institutes of Health and recently served as the elected Secretary-Treasurer of the American Society of Clinical Investigation. She has served on several other NIH advisory committees and chaired an NIH Roundtable regarding preclinical autoimmunity.

Dr. James also was selected to provide testimony supporting the NIH at the Noel Laureates' Hearing for the US Senate Appropriations Subcommittee. Dr. James received her medical degree and PhD in Immunology from the Oklahoma Health Sciences Center and is a board certified adult Rheumatologist. She continues to practice adult rheumatology, focusing on SLE, incomplete lupus, Sjogren's syndrome, and related rheumatic diseases.



JENNIFER HOLTER CHAKRABARTY, MD

*Professor of medicine, hematology/oncology, Adjunct Associate
Professor of radiation oncology*

Dr. Jennifer Holter Chakrabarty is a tenured Presidential Professor, the Clinical Program Director of Transplantation and Cellular Therapy, and the Gary McKinney Chair of Bone Marrow Diseases at The University of Oklahoma Stephenson Cancer Center. With over 20 years' experience in BMT/CT, she has authored seminal guidelines in Hematology/BMT/CT with the American Society of Hematology (ASH) and the American Society of Transplantation and Cellular Therapy (ASTCT). She is a Councillor for ASH, serving on its Executive Committee, and a board member for ASTCT. Her NIH-funded research studies graft failure and imaging of marrow transplant/repopulation. She is Past Chair of ASH Government Affairs, is active in task forces on immunotherapy and palliative care and serves as Chair of Education for the ACCESS Initiative of ASTCT/National Marrow Donor Program. She serves on the nominating committee for the Center for International Blood and Marrow Transplant Research, served on the Board of Directors for ASTCT, and is an ASTCT Fellow.



SAMANTHA HARBERT, CIP

*Director | Office of Human Research Participant Protection (HRPP)
Institutional Review Board (IRB), The University of Oklahoma Health
Campus*

Samantha Harbert, BS, CIP, is Director of the Office of Human Research Participant Protection (HRPP) and Institutional Review Board (IRB) for the University of Oklahoma Health Campus. With nearly 15 years of experience in human subjects protections, she provides strategic leadership for the HRPP, IRB operations, quality improvement initiatives, and outreach efforts that promote the ethical conduct of human subjects research. Throughout her career, Samantha has led and supported numerous institutional initiatives, including implementation of the Revised Common Rule, development of policies and procedures, AAHRPP accreditation, and research education programs. Her professional interests include promoting meaningful informed consent, enhancing participant understanding, and improving operational processes to strengthen participant protections and advance high-quality research. Samantha currently serves as Co-Chair of the Federal Demonstration Partnership Human Subjects Subcommittee and has been a Certified IRB Professional since 2016.



CONSULTATION

CONSULTING AGENCY OFFICE HOURS

LEVEL 1 – EAST/WEST BOARD ROOMS

1:00 PM – 4:00 PM

BOARD ROOM EAST

OK CATALYST

<https://www.okcatalyst.com/>

OKCATALYST

OK Catalyst is Oklahoma's hub for SBIR/STTR support, empowering local innovators, researchers, and entrepreneurs to compete for federal R&D funding through America's Seed Fund.



We specialize in helping small businesses, especially those new to the SBIR/STTR process, navigate complex program requirements, identify the right opportunities, and develop competitive proposals.

Jaime Hough, PhD

Outreach & Recruitment Manager, OKCatalyst

Startup OU

<https://www.ou.edu/innovationhub/startups>

STARTUP OU

Startup OU is the home base for Sooner entrepreneurs and innovators. We provide students, staff, and faculty at the University of Oklahoma with the community, resources, and mentorship they need to build a successful startup.



Micah Headley

Venture Programs Manager, Startup OU

CONSULTING AGENCY OFFICE HOURS

LEVEL 1 – EAST/WEST BOARD ROOMS

1:00 PM – 4:00 PM

BOARD ROOM EAST



OU Fab Lab

Chase Greeno

Project Manager, Startup OU/Fabrication Lab

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FABRICATION LAB

The Fabrication Lab (Fab Lab) offers state-of-the-art equipment and technical expertise to support product prototyping, from initial design to final build. Key offerings include: specialized workspaces for woodworking, metal working, electronic component fabrication, and physical design.

Whatever the interest, the Fabrication Lab offers a wide range of tools and a talented team of specialists to assist in bringing ideas, designs and prototypes to life.

CONSULTING AGENCY OFFICE HOURS

LEVEL 1 – EAST/WEST BOARD ROOMS

1:00 PM – 4:00 PM

BOARD ROOM WEST



Emi Kiyotaki, PhD

Concept Program Manager, OKBioStart

<https://www.okbiostart.com/>

ABOUT

Dr. Emi Kiyotake is the Concept Program Manager at OKBioStart within the University of Oklahoma's Vice President for Research and Partnerships office. She leads commercial due diligence and commercialization planning for early-stage life science and biotechnology innovations, working with researcher-founders across Oklahoma to evaluate promising technologies and develop clear paths toward further development, funding, licensing, or company formation.

Since joining OKBioStart in 2025, Dr. Kiyotake has led commercial due diligence and developed pitch decks and project plans for 21 participant concepts: 10 therapeutics, 4 medical devices, 2 combination drug-device products, and 5 software platforms. Her work assesses intellectual property and exclusivity, regulatory strategy, market opportunity, competition, business models, potential exit paths, and key scientific and commercial risks. She also leads customer discovery, incorporating stakeholder and reviewer insights into actionable scientific and business milestones.

Dr. Kiyotake previously spent more than nine years in translational biomaterials research, developing biomaterials for cartilage repair and spinal cord injury. She has authored 16 peer-reviewed publications, including eight as first author; served in professional society leadership; and mentored students and fellows in biomedical research and commercial due diligence. She earned her Ph.D. in Biomedical Engineering from the University of Oklahoma and her B.S. in Chemical and Biological Engineering from the University of Colorado Boulder. Her goal is to help researchers translate discoveries into commercial impact and strengthen Oklahoma's life science ecosystem.

OKBIOSTART

At OKBioStart, we believe that life science is critical to shaping the future of healthcare globally. We are committed to advancing biotech solutions by providing support and resources to researchers and entrepreneurs in the 10-county region of Oklahoma City.

Our program is designed to help translate scientific discoveries from the lab to the commercial marketplace, where they can improve people's lives and drive economic growth.

CONSULTING AGENCY OFFICE HOURS

LEVEL 1 – EAST/WEST BOARD ROOMS

1:00 PM – 4:00 PM

BOARD ROOM WEST



Martha Tsaliki, PhD

Launch Program Manager, OKBioStart

<https://www.okbiostart.com/>

ABOUT

Dr. Martha Tsaliki is the Launch Program Manager at OKBioStart, where she guides early-stage life science teams as they execute the most critical scientific and business milestones identified through the Concept Program. She works closely with founders to advance innovations toward meaningful development, funding, licensing, or company-formation opportunities.

Through the one-year Launch Program, Dr. Tsaliki helps teams develop and execute focused plans supported by up to \$70,000 in non-dilutive funding. Her role combines strategic guidance with hands-on project management, helping participants identify and engage key opinion leaders, consultants, and vendors; refine regulatory and intellectual property strategies; pursue follow-on funding; and develop go-to-market and incorporation plans. She also cultivates relationships with regional entrepreneurial-support programs, specialized regulatory and transaction advisors, and industry stakeholders to expand resources for participants and strengthen Oklahoma's life science ecosystem.

Before joining OKBioStart, Dr. Tsaliki gained experience in biotech investment and life science consulting through roles with Cortado Ventures and the Ronnie K. Irani Center for the Creation of Economic Wealth. Her doctoral research focused on pathogenic mediators and autoantibodies in Sjögren's disease. She has authored eight peer-reviewed journal articles and a book chapter, including four first-author publications, and has presented her work at national and international scientific conferences. Dr. Tsaliki earned her Ph.D. in Biomedical Sciences from the University of Oklahoma Health Sciences Center and her B.S. in Biological Sciences-Molecular Biology from McNeese State University.

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At OKBioStart, we believe that life science is critical to shaping the future of healthcare globally. We are committed to advancing biotech solutions by providing support and resources to researchers and entrepreneurs in the 10-county region of Oklahoma City.

Our program is designed to help translate scientific discoveries from the lab to the commercial marketplace, where they can improve people's lives and drive economic growth.

CONSULTING AGENCY OFFICE HOURS

LEVEL 1 – EAST/WEST BOARD ROOMS

1:00 PM – 4:00 PM

BOARD ROOM WEST



OFFICE OF TECHNOLOGY COMMERCIALIZATION
The UNIVERSITY of OKLAHOMA

Andrew Pollock, MBA

Managing Director, Office of Technology Commercialization

<https://www.ou.edu/otc>

ABOUT

Andrew Pollock is the Managing Director for the Office of Technology Commercialization, having joined the University of Oklahoma in 2014.

Andrew manages the overall day to day operations of OTC and serves as a technology manager for non-life science related inventions from the Norman and HSC campuses. In these roles Andrew works directly with faculty, other members of OTC and outside counsel to formulate protection strategies for intellectual property developed at the University, as well as engaging with industry to amplify the impact of these discoveries through commercialization. He has lead multiple initiatives to improve the outcomes of University IP including the recent launch of the upfront licensing of IP in research agreements.

Prior to joining the OTC Andrew was involved in an early-stage startup in the Oklahoma City area focused on external fixation prosthetics, where he built upon his background in Biomedical Engineering, and cultivated experience in grant writing, clinical study design, regulatory filings, venture financing and product development, leading to the launch of 4 new products. He is a proud alumnus of OU with a 2002 degree in Zoology.

OFFICE OF TECHNOLOGY AND COMMERCIALIZATION

The Office of Technology Commercialization, part of the Vice President for Research and Partnerships, helps innovation from the sharpest minds at OU transform into world-changing products. OTC serves as a resource for all faculty, staff, and graduate students interested in exploring the commercialization of intellectual property.

Additionally, new initiatives like the Growth Fund and the Post-Graduate Venture Fellows program allow us to further support campus inventors, as well as enhance the opportunities for the commercialization of university innovation.



ORAL COMPETITION

ORAL COMPETITION

Rainbolt Auditorium
3:00 pm – 3:58 pm

MODERATOR

Anne Kasus-Jacobi, PhD

*Associate Professor, The University of
Oklahoma Health Campus*



Snehal Patel, MD, PhD

3:00 pm – 3:08 pm

Development Of A First-In-Class Computational Pipeline For
Predicting Gain-Of-Function Variation In Oncogenes Using
Molecular Dynamic Simulations



Grant Chesbro, PhD

3:10 PM – 3:18 PM

Hallux Force Becomes More Variable After Neurotoxic
Chemotherapy: Application of a Proprietary Device for Screening
Motor Peripheral Neuropathy



Soumyabrata Munshi, MD, PhD

3:20 PM – 3:28 PM

Post-exposure Systemic Treatment with Nociceptin/Orphanin
FQ (N/OFFQ) Peptide (NOP) Receptor Antagonist, SB-612111,
Improves Behavioral Outcomes and Causes Neurocellular
Remodeling in a Preclinical Female Rat Model of Traumatic
Brain Injury

ORAL COMPETITION

Rainbolt Auditorium
3:00 pm – 3:58 pm

MODERATOR

Anne Kasus–Jacobi, PhD

*Associate Professor, The University of
Oklahoma Health Campus*



Kaili Liu, PhD

3:30 pm – 3:38 pm

Boundary-Organized Stroma In Human Pancreatic Cancer: A
Multi-Platform Spatial Atlas For Spatial Biomarker Development



David Miller, PhD

3:40 PM – 3:48 PM

Real-Time Detection Of Stroke During Neurosurgery Using
Laser Speckle Contrast Imaging And Machine Learning



Edriss Mirnia, PhD

3:50 PM – 3:58 PM

Label Free Optical Imaging For Oral Lesion Risk Stratification

ORAL COMPETITION

Rainbolt Auditorium
3:00 pm – 3:58 pm



Snehal Patel, MD, PhD

3:00 pm – 3:08 pm

Assistant Professor, Clinical Pathology, Molecular Pathology
Laboratory Medical Director, OU Health

Dr. Snehal Patel is a molecular pathologist and machine learning engineer with expertise in next-generation sequencing, cancer genomics, and artificial intelligence applications in pathology. He completed his MD/PhD at the University of Illinois, followed by AP/CP residency and MGP fellowship at Cedars-Sinai Medical Center and AI research fellowship at Michigan State University. Dr. Patel's research focuses on developing computational methods to improve cancer diagnosis and treatment selection, with a particular interest in integrating multi-modal data to enhance precision medicine approaches.

ABSTRACT OMITTED



Grant Chesbro, PhD

3:10 pm – 3:18 pm

Postdoctoral Research Fellow, Stephenson Cancer Center,
University of Oklahoma Health Sciences Center

Dr. Grant Chesbro is a Postdoctoral Research Fellow at Stephenson Cancer Center at the University of Oklahoma Health Sciences Center, where he works in the Cancer Rehabilitation Research Laboratory under the direction of Dr. Elizabeth Hile. His research focuses on neuromuscular function, motor control, fatigue, and human performance, with an emphasis on understanding and assessing functional impairments associated with neurological and clinical conditions. Dr. Chesbro earned his Ph.D. in Exercise Physiology from the University of Oklahoma, where his doctoral research examined training status differences in neuromuscular variability and complexity during fatiguing exercise. His work integrates exercise physiology, neuromuscular science, and translational research to better understand changes in motor function and develop clinically relevant approaches to the assessment and rehabilitation of neuromuscular dysfunction.

ORAL COMPETITION

Rainbolt Auditorium
3:00 pm – 3:58 pm

ABSTRACT

Hallux Force Becomes More Variable After Neurotoxic Chemotherapy: Application of a Proprietary Device for Screening Motor Peripheral Neuropathy

Authors: Grant Chesbro¹, Elizabeth S. Hile^{1,2}

¹Stephenson Cancer Center, University of Oklahoma Health Sciences Center

²Dept of Rehabilitation Sciences, College of Allied Health, OU Health Campus

Introduction: Peripheral neuropathy (PN) is the most common dose-limiting effect of neurotoxic chemotherapy. Research and practice focus on sensory symptoms, but motor impairments occur and may disrupt balance and gait. Because PN starts in the toes, experts recommend screening hallux extension Peak Force (PF). Rate of Force Development (RFD), Coefficient of Variation (CV), and Yank, force metrics that reflect motor control for standing activities, are not published in chemotoxicity. Hence, we examined changes in all four hallux force metrics using data collected with our proprietary dynamometer as women developed sensory PN during taxane/platinum chemotherapy. Methods: The right and left halluces of 8 women (67.4yrs±7.5) with gynecological (n=7) or breast (n=1) cancer were tested before and after 2 chemotherapy cycles. Using Repeated-Measures ANOVA (Bonferroni-adjusted), the highest of two 10-second maximal contractions were compared for the outcomes of PF, RFD, and the magnitude (CV) and rate (yank) of force variability in the final 5-sec hold. Results: While group change in PF was not significant (Mean Difference Right: -2.13N, $p=0.468$, $\eta^2=0.078$; Left: 0.73N, $p=0.794$, $\eta^2=0.010$), four participants declined from baseline (Right: -20.6%±6.2; Left: -11.8%±9.4). RFD also did not change significantly for the group (Right: -16.85 N/s, $p=0.591$, $\eta^2=0.043$; Left: -9.84 N/s, $p=0.571$, $\eta^2=0.043$). In contrast, force CV (Right: 6.07%, $p=0.005$, $\eta^2=0.707$; Left: 6.40%, $p=0.036$, $\eta^2=0.491$) and yank (Right: 0.09 N/s, $p=0.049$, $\eta^2=0.447$; Left: 0.05 N/s, $p=0.033$, $\eta^2=0.500$) significantly increased for the group.

Translational Impact: Hallux force variability increased in magnitude and rate as these women developed sensory PN, despite only 50% losing peak force after 2 cycles. We know of no other dynamometer that reports variability parameters. Results support continued engineering collaborations to optimize this device toward biomedical commercialization while simultaneously expanding studies of hallux force variability as an earlier indicator for screening, phenotyping, and monitoring cancer patients for chemotherapy-induced motor PN.

Funding: This research was funded by the Presbyterian Health Foundation (PHF) New Investigator Seed Grant Award to Elizabeth Hile, the Oklahoma Tobacco Settlement Endowment Trust (TSET) R23-03 and the National Cancer Institute Cancer Center Support Grant P30CA225520 awarded to the University of Oklahoma Stephenson Cancer Center. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health, Oklahoma TSET, or PHF. The APC was funded by the Oklahoma TSET.

ORAL COMPETITION

Rainbolt Auditorium
3:00 pm – 3:58 pm



Soumyabrata Munshi, MD, PhD

3:20 pm – 3:28 pm

Postdoctoral Research Fellow, University of Oklahoma Health Sciences Center

Dr. Soumyabrata Munshi is a medical scientist specializing in neuroscience and neuroimmunology. He holds M.B.B.S. and M.D. degrees from Calcutta, India, and a Ph.D. in neuroscience from Rosalind Franklin University of Medicine and Science, USA. He is currently a postdoctoral research fellow at the University of Oklahoma Health Sciences Center, with prior postdoctoral research appointments at Tulane University and Saint Francis Health System.

The overarching goal of Dr. Munshi's research is to uncover the underlying neuroimmune mechanisms of stress, trauma, neurodegeneration, and addiction, with the aim of identifying novel therapeutic targets for neurological and neuropsychiatric disorders.

Dr. Munshi has authored over 20 peer-reviewed journal articles, including prominent first-author publications in Molecular Psychiatry, The Journal of Neuroscience, Addiction Biology, Neuroscience, Behavioural Brain Research, and Brain, Behavior, and Immunity, among others. He serves as editorial board member and reviewer for a few major journals in his field. He had also been elected to membership in a few professional societies in recognition of his contributions to his field.

Dedicated to higher education excellence, Dr. Munshi also holds a certificate from the Preparing Future Faculty program, demonstrating his commitment to evidence-based pedagogy, professional mentorship, and academic leadership.

ABSTRACT

POST-EXPOSURE SYSTEMIC TREATMENT WITH NOCICEPTIN/ORPHANIN FQ (N/OFQ) PEPTIDE (NOP) RECEPTOR ANTAGONIST, SB-612111, IMPROVES BEHAVIORAL OUTCOMES AND CAUSES NEUROCELLULAR REMODELING IN A PRECLINICAL FEMALE RAT MODEL OF TRAUMATIC BRAIN INJURY

Soumyabrata Munshi¹, Megan Stiles², Kaylee Goodwin³, David Sherry², Kelly Standifer¹

¹ Department of Pharmaceutical Sciences, College of Pharmacy, University of Oklahoma Health Campus, Oklahoma City, OK

² Department of Cell Biology, College of Medicine, University of Oklahoma Health Campus, Oklahoma City, OK

³ Dodge Family College of Arts and Sciences, University of Oklahoma, Norman, OK

ORAL COMPETITION

Rainbolt Auditorium
3:00 pm – 3:58 pm

Introduction: Traumatic brain injury (TBI) is a major public health concern associated with persistent neurological, cognitive, and behavioral impairments, yet no FDA-approved pharmacological treatment exists. Despite evidence that biological sex influences neuroinflammation, neuronal injury, and functional recovery, females remain underrepresented in preclinical TBI research. Nociceptin/orphanin FQ (N/OFQ) peptide (NOP) receptor signaling regulates neuronal, inflammatory, and behavioral processes, and loss of it improves TBI outcomes. Therefore, we investigated whether NOP receptor antagonism promotes neurocellular remodeling and functional recovery after TBI.

Methods: Adult female Wistar Han rats underwent controlled cortical impact TBI or sham procedure (day 0), followed by daily i.p. treatment with the NOP receptor antagonist SB-612111 (5 mg/kg/day) or vehicle for 7 days (days 0 – 6). Neurological deficits, vestibulomotor performance, anxiety-like behavior, object recognition memory, and spatial retention memory were assessed through day 16. Ipsilateral neurocellular remodeling was evaluated using immunofluorescence markers for neurons (NeuN), neuronal dendrites (MAP2), microglia (Iba-1), astrocytes (GFAP), and myelinating oligodendrocytes (BCAS1).

Results: SB-612111 treatment attenuated neurological deficits by day 7, improved vestibulomotor performance by day 9, and reduced anxiety-like behavior, but did not improve memories, suggesting different mechanisms underlie deficits after TBI. SB-612111 restored TBI-induced reduction in cortical MAP2 expression and increased TBI-induced microglial arborization without altering cortical and thalamic Iba-1 expression, suggesting induction of positive brain remodeling and improved neuroplasticity by SB-612111 as well as structural reorganization toward a more homeostatic microglial state without affecting microglial population. MAP2 expression in the thalamus and NeuN, GFAP, and BCAS1 expression in both regions remained unchanged, indicating regional and cell-specific responses.

Translational Impact: NOP receptor antagonism differentially improved functional outcomes and promoted adaptive neurocellular remodeling after TBI in female rats. These findings identify NOP as a potential sex-informed therapeutic target with domain-specific effects. Future studies investigating dose-response relationships and sex-dependent responses will be required before clinical translation.

Funding: HT9425-23-1-0517 (U.S. Department of Defense)

ORAL COMPETITION

Rainbolt Auditorium
3:00 pm – 3:58 pm



Kaili Liu, PhD
3:30 pm – 3:38 pm

Research Assistant Professor, The University of Oklahoma

Kaili Liu, PhD, is a Research Assistant Professor at the Stephenson School of Biomedical Engineering at the University of Oklahoma. Dr. Liu's research integrates cancer immunotherapy, biomedical engineering, and single-cell and spatial multi-omics to understand how tumors and immune ecosystems respond to treatment. A major focus has been localized ablative immunotherapy, including single-cell transcriptomic studies of how treatment remodels tumor-infiltrating immune cells. This work also examines immunostimulatory biomaterials and nanoparticle-enabled approaches for cancer therapy and vaccine development. Building on this foundation, Dr. Liu's current research investigates human pancreatic cancer through multi-cohort single-cell and spatial transcriptomic analyses, with particular attention to the organization of malignant, stromal, and immune compartments at tumor boundaries. The long-term goal is to translate reproducible molecular, cellular and spatial features into testable biomarkers and strategies for patient stratification and therapeutic development. Dr. Liu received a PhD in Biochemistry and Molecular Biology from the Institute of Biophysics, Chinese Academy of Sciences, and completed postdoctoral training at the Oklahoma Medical Research Foundation and the University of Oklahoma.

ABSTRACT

BOUNDARY-ORGANIZED STROMA IN HUMAN PANCREATIC CANCER: A MULTI-PLATFORM SPATIAL ATLAS FOR SPATIAL BIOMARKER DEVELOPMENT

Kaili Liu¹, Hua Zhong¹, Rahhul S. Elangovan¹, Yuanhong Sun¹, Lin Wang¹, Abigae P. Williams¹, Trisha I. Valerio¹, Coline Furrer¹, Jacob P. Adams¹, Ashley R. Hoover¹, Wei R. Chen¹

¹Stephenson School of Biomedical Engineering, University of Oklahoma, Norman, OK

Introduction: Traumatic brain injury (TBI) is a major public health concern associated with persistent neurological, cognitive, and behavioral impairments, yet no FDA-approved pharmacological treatment exists. Despite evidence that biological sex influences neuroinflammation, neuronal injury, and functional recovery, females remain underrepresented in preclinical TBI research. Nociceptin/orphanin FQ (N/OFFQ) peptide (NOP) receptor signaling regulates neuronal, inflammatory, and behavioral processes, and loss of it improves TBI outcomes. Therefore, we investigated whether NOP receptor antagonism promotes neurocellular remodeling and functional recovery after TBI.

ORAL COMPETITION

Rainbolt Auditorium
3:00 pm – 3:58 pm

Methods. We assembled a single-cell reference atlas of 1,186,130 human pancreas cells from 19 published studies and used it to interpret 176 spatial gene-expression sections (458,877 tissue spots) spanning non-diseased pancreas, chronic pancreatitis, precancerous lesions, primary cancer, and metastasis. Malignant tissue was defined by a uniform lineage-marker criterion applied across all cohorts and platforms. All statistics were computed per sample or per patient, re-tested in six patient-matched normal-tumor pairs, and re-examined on two higher-resolution spatial platforms.

Results. Distance-resolved analysis revealed a reproducible three-layer organization: a malignant core, a band of fibroblasts and macrophages beginning precisely at the tumor boundary, and a lymphocyte-rich zone farther out — not the uniform narrow rim often depicted. Candidate matrix-mediated signaling (collagen, laminin, fibronectin) concentrated at this interface. After controlling for cell-type composition, fibroblast state rather than fibroblast number tracked position: matrix-depositing gene programs adjacent to tumor gave way to inflammatory programs with distance (19 patients, five studies; $P = 4.8e-05$). Higher-resolution platforms recovered the same architecture and showed that measurement resolution itself changes estimates of immune-cell adjacency.

Translational Impact. The tumor boundary is a recurrent, positionally organized structure — the interface, not bulk stromal content, is the candidate node for intervention. Validation in prospective surgical cohorts with linked outcomes — a natural cross-campus effort — would convert this structure into a testable spatial biomarker.



David Miller, PhD
3:40 pm – 3:48 pm
Postdoctoral Research Fellow

ABSTRACT OMITTED



Edriss Mirnia, PhD
3:50 pm – 3:58 pm
Postdoctoral Research Fellow

Seyed Edriss Mirnia, Ph.D., is a Software/AI Engineer at the University of Oklahoma, where his work focuses on biomedical imaging and machine learning for cancer detection. He develops image analysis methods for optical coherence tomography and fluorescence lifetime imaging, with a particular focus on identifying high-risk oral lesions. He earned his Ph.D. in Electrical Engineering from the Florida Institute of Technology in 2020 and completed postdoctoral research at the University at Buffalo, SUNY.

ABSTRACT OMITTED

ORAL COMPETITION

Rainbolt Auditorium
3:00 pm – 3:58 pm



MODERATOR

Anne Kasus-Jacobi, PhD

Associate Professor, The University of Oklahoma Health
Campus

Dr. Anne Kasus-Jacobi is an Associate Professor of Pharmaceutical Sciences at the University of Oklahoma Health Campus, where she leads a translational research program focused on the discovery and development of innovative peptide therapeutics for Alzheimer's disease and other neurodegenerative conditions.

Over the past decade, Dr. Kasus-Jacobi has secured sustained support from the National Institutes of Health and regional and local funding organizations to advance her research from fundamental laboratory discoveries toward therapeutic applications. Her work has contributed to the development and characterization of novel peptide drug candidates targeting mechanisms associated with amyloid-beta pathology, neuroinflammation, and neurotoxicity. Her research has also encompassed antimicrobial peptides and their potential applications in corneal wound healing and ocular disease.

In 2022, Dr. Kasus-Jacobi founded Alzarrok Bio to advance the development and commercialization of her patented therapeutic technology. The company is currently pursuing in vivo proof-of-concept for a multitarget peptide drug candidate for Alzheimer's disease, supported by a National Institutes of Health Small Business Technology Transfer (STTR) grant.

In addition to her research and entrepreneurial activities, Dr. Kasus-Jacobi is deeply committed to education and mentorship. She teaches pharmacology in the professional Doctor of Pharmacy program at the OU College of Pharmacy and mentors graduate students pursuing careers in pharmaceutical sciences and biomedical research.

Dr. Kasus-Jacobi earned her Ph.D. in Endocrinology from the University of Paris XI in France and completed postdoctoral training in molecular genetics at UT Southwestern Medical Center in Dallas, Texas. Since joining the University of Oklahoma, she has built a multidisciplinary research career spanning drug discovery and development, including antimicrobial peptides, antioxidant approaches for retinal degeneration, and, more recently, multitarget peptide therapeutics for neurodegenerative diseases.

POSTERS

SESSION 1

POSTER #	FIRST NAME	LAST NAME	ABSTRACT TITLE
1A ABSTRACT OMITTED	Mikail	Abbasov	EVERY CANCER CREATES ITS OWN DRUG TARGETS: 295,411 NEW LYSINES WRITTEN BY MUTATION, THE TECHNOLOGY TO MAP THEM, AND THE CHEMISTRY TO ENGAGE THEM
2A	Hassan	Abushukair	SINGLE-CELL AND SPATIAL TRANSCRIPTOMICS IDENTIFY AN IL-17/MAPK IMMUNE PROGRAM IN IMMUNE CHECKPOINT INHIBITOR-ASSOCIATED TRIPLE M OVERLAP SYNDROME
3A	Angelina	Akomea	FROM EXOSOMES TO ANTIBODIES: DEVELOPING TARGETED MONOCLONAL ANTIBODIES FOR PANCREATIC CANCER THERAPY
4A	Rasheeda	Ali	FROM FAMILY SILENCE TO SHARED RISK: A QUALITATIVE ANALYSIS OF GENETIC TESTING AND FAMILIAL RISK COMMUNICATION AMONG BLACK PROSTATE CANCER SURVIVORS
5A ABSTRACT OMITTED	Kiana	Amani	OPTIMIZING PARENCHYME INJECTION STRATEGIES FOR LOCALIZED DRUG DELIVERY IN GLIOBLASTOMA
6A ABSTRACT OMITTED	Nasim	Azari Torbat	SUPER-RESOLUTION IMAGING OF INTRACELLULAR LIPID AND POLYMERIC NANOPARTICLES FOR NEXT-GENERATION NANOMEDICINE DEVELOPMENT
7A	Babunageswararao	Kanuri	DISULFIRAM PROTECTS AGAINST DIET-INDUCED OBESITY BY REPROGRAMMING DIETARY LIPID FATE INDEPENDENT OF INFLAMMASOME SIGNALING
8A	Syed Saqib	Balkhi	BLOOD TESTS FOR TUMOR DNA IN KIDNEY CANCER: DETECTING RECURRENCE AND PREDICTING OUTCOMES ACROSS STAGES OF DISEASE
9A	Indranil	Biswas	TARGETING SUCCINATE RECEPTOR 1 TO MITIGATE NECROTIZING ENTEROCOLITIS
10A	Evie	Cameron-Mazin	DEVELOPMENT AND OPTIMIZATION OF BIODEGRADABLE LIPID NANOPARTICLES FOR MRNA DELIVERY TO THE LUNG ENDOTHELIUM
11A ABSTRACT OMITTED	Brototi	Chakrabarty	SODIUM THIOSULFATE NANOPARTICLES SHIELD SENSORY HAIR CELLS AGAINST CISPLATIN IN PRECLINICAL 3D COCHLEAR CULTURE AND ZEBRAFISH
12A	Siva Sai	Chandragiri	RADIATION-INDUCED ENDOTHELIAL SENESCENCE DRIVES CEREBROVASCULAR FRAGILITY AND MICROHEMORRHAGE: A SENOLYTIC-TARGETABLE MECHANISM OF ACCELERATED BRAIN VASCULAR AGING
13A ABSTRACT OMITTED	Kris	Dahlgren	ANCHOR-SPACER-LIGAND (ASL) CHEMISTRY ENABLES FUNCTIONAL AND REPRODUCIBLE PHYSICAL MODIFICATION OF EXOSOME MEMBRANES
14A	Akshaj	Dalavayi	INVESTIGATING THE EFFECTS OF METABOLISM MODULATING DRUG ON VIABILITY OF CANCER CELLS
15A	Ananya	Datta	EVALUATING CYTOKINE-MEDIATED NEUROVASCULAR INJURY IN HUMAN CEREBROVASCULAR CELL SPHEROIDS
16A	Somasekhara	Derangula	ATOVAQUONE ATTENUATES PANCREATIC CANCER-INDUCED CACHEXIA
17A ABSTRACT OMITTED	Morgan	Dyer	EFFECTS OF LONG-TERM AUDITORY DEPRIVATION ON INTER-ARTICULATOR SPEECH COORDINATION IN COCHLEAR IMPLANT RECIPIENTS
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Omitted abstracts were not included in the printed program at the authors' request.

ABSTRACT TITLE

SINGLE-CELL AND SPATIAL TRANSCRIPTOMICS IDENTIFY AN IL-17/MAPK IMMUNE PROGRAM IN IMMUNE CHECKPOINT INHIBITOR-ASSOCIATED TRIPLE M OVERLAP SYNDROME

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**Equal contribution and co-correspondence*

ABSTRACT TITLE

SINGLE-CELL AND SPATIAL TRANSCRIPTOMICS IDENTIFY AN IL-17/MAPK IMMUNE PROGRAM IN IMMUNE CHECKPOINT INHIBITOR-ASSOCIATED TRIPLE M OVERLAP SYNDROME

Abstract:

Introduction: Immune checkpoint inhibitor (ICI)-associated Triple M Overlap Syndrome (TMOS), concurrent myocarditis, myositis, and myasthenia gravis, represents the most severe and steroid-refractory form of ICI cardiotoxicity, with a fatality rate exceeding 38%. Its molecular basis remains undefined, and no biology-directed therapies exist.

Methods: We integrated cross-tissue single-cell RNA sequencing (scRNA-seq) from heart (n = 15), skeletal muscle (n = 6), and peripheral blood (n = 21) across two independent institutional cohorts with Xenium 5K spatial transcriptomics of TMOS cardiac and skeletal muscle tissue (n = 3) (Figure 1). Pseudobulk differential expression, pathway enrichment, spatial niche analysis, proximity-based cardiomyocyte stratification, and longitudinal peripheral blood module scoring were performed.

Results: TMOS cardiac T/NK cells exhibited a qualitatively distinct transcriptional program enriched for IL-17 signaling and downstream MAPK activation relative to myocarditis alone, consistent across both cardiac and skeletal muscle compartments. Spatial profiling revealed TMOS cardiac inflammation organized into four discrete cellular niches, with IL-17/MAPK signaling concentrated in an immune effector niche dominated by classical monocytes, intermediate monocytes, and Th1/Th17 cells. Cardiomyocytes adjacent to IL-17/MAPK-high immune hotspots underwent distance-dependent transcriptional reprogramming from contractile to inflammatory identity. A rising peripheral blood IL-17 trajectory in myocarditis overlap cases despite steroid treatment mirrored the spatial niche signature, suggesting persistent pathway activity contributing to steroid refractoriness. A pre-myocarditis four-gene peripheral blood signature showed preliminary evidence of distinguishing myocarditis overlap from myocarditis alone before clinical onset.

Translational Impact: These findings identify IL-17/MAPK as a spatially organized and potentially targetable axis in TMOS, with IL-17RA blockade and MAPK inhibition as candidate therapeutic strategies. An integrative spatial-peripheral framework is established for non-invasive monitoring of tissue-level inflammatory activity in ICI multi-organ autoimmunity.

Funding: This work was supported in part by start-up funding provided by the Department of Oncology Science, OUHC (TGO). This manuscript is supported in part by R01CA270234, R01CA163649, and U54CA274329 to PKS.

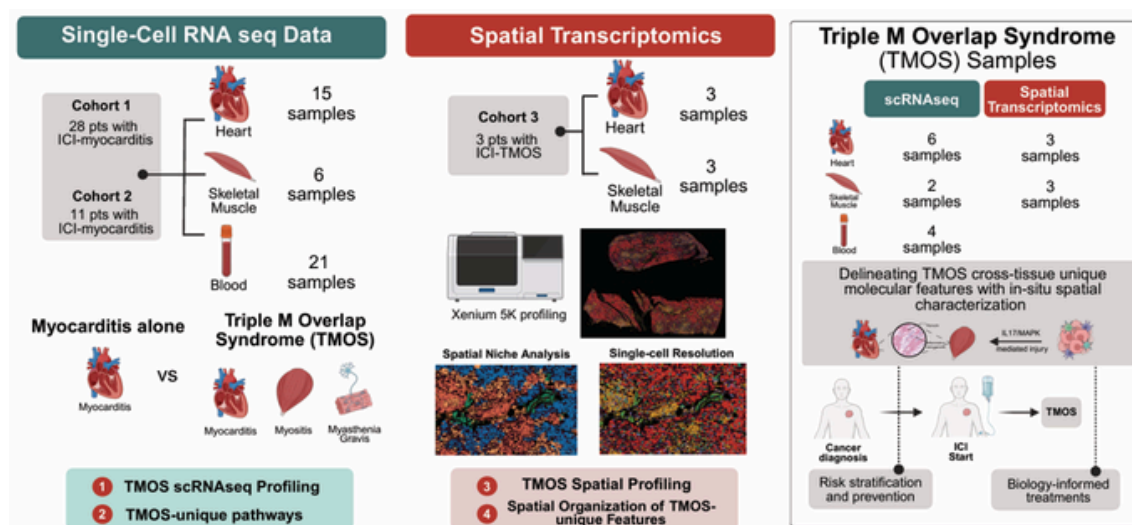


Figure 1: Study overview schematic showing cohort composition, analytical workflow, and TMOS sample distribution across scRNA-seq and spatial transcriptomics platforms

ABSTRACT TITLE

FROM EXOSOMES TO ANTIBODIES: DEVELOPING TARGETED MONOCLONAL ANTIBODIES FOR PANCREATIC CANCER THERAPY

Angelina Akomea^{1,2}, Maryam Firouzi¹, Dongin (Donoven) Kim¹

¹Department of Pharmaceutical Sciences, College of Pharmacy.

Abstract:

Introduction: Pancreatic ductal adenocarcinoma (PDAC) remains highly lethal because of late diagnosis, rapid progression and resistance to standard therapies. We propose an exosome-guided antibody discovery strategy in which small extracellular vesicles (sEVs) from pancreatic cancer cells are used as immunogens to generate monoclonal antibodies (mAbs) that retain high affinity for antigens displayed on the surface of the parent tumor cells.

Method: In this study, the size and concentration of exosomes isolated from Panc-1 human cell line were characterized by nanoparticle tracking analysis (NTA). The affinity tests were accomplished through immunochemistry technology. Mice were then immunized by three intraperitoneal (IP) injections of Panc-1 exosomes (5×10^9 particles/dose with complete and incomplete adjuvants) at 2-week intervals. Following administration, antibody production in mice serum were visualized by immunocytochemistry against Panc-1 cells. Hybridoma cells were then generated by fusing the splenic B-cells from the immunized mice with myeloma cells. Antibody screening was conducted by testing the supernatants from multiple hybridomas for binding affinities to Panc-1 cells. Single-cell cloning was subsequently used to isolate hybridomas capable of producing monoclonal antibodies for selective targeting of pancreatic cancer cells.

Results/Conclusions: The size and concentration of exosomes were an average size of ~100 nm and concentration of 1.6×10^{11} particles/ml respectively. The serum from vaccinated mice showed the parent pancreatic cancer cell affinity, proving the production of antibody within the body. After successful establishment of hybridoma cells, the antibody showing the highest affinity to the parent pancreatic cancer cell was screened. Our results shows that Panc-1 derived exosomes can be used to generate specific monoclonal antibodies that bind strongly to these pancreatic cancer cells. These antibodies will enable ligand discovery for future development of targeted therapeutics for PDAC. Future studies will focus on optimizing exosome formulations by using oxaliplatin-loaded CD8⁺ T-cell derived in Panc-1 tumor bearing mice.

POSTER PRESENTATIONS

SESSION 1 | 4A

ABSTRACT TITLE

FROM FAMILY SILENCE TO SHARED RISK: A QUALITATIVE ANALYSIS OF GENETIC TESTING AND FAMILIAL RISK COMMUNICATION AMONG BLACK PROSTATE CANCER SURVIVORS

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ABSTRACT TITLE

FROM FAMILY SILENCE TO SHARED RISK: A QUALITATIVE ANALYSIS OF GENETIC TESTING AND FAMILIAL RISK COMMUNICATION AMONG BLACK PROSTATE CANCER SURVIVORS

Introduction Black men (BM) are disproportionately affected by prostate cancer (CaP). Nationally, there are an estimated 333,830 new cases and 36,320 deaths in 2026. BM experience an incidence rate approximately 70% higher than White men. Communication about family cancer risk and genetic testing is therefore particularly important for Black CaP survivors and their families. This study aimed to identify communicative barriers, knowledge gaps, and educational needs related to family risk and genetic testing for CaP survivors and their families. **Methods:** We conducted a descriptive qualitative analysis of the transcript of a virtual community townhall held on June 25th, 2026, at the Stephenson Cancer Center, which lasted approximately 100 minutes, with a focus on genetic testing, family communication, and hereditary risk. There were 5 panelists speaking at this townhall, including two CaP survivors and advocates, a genetic counselor, a Urologist, and a Genitourinary Oncologist. The townhall was recorded and transcribed verbatim and analyzed using Braun and Clarke's thematic analysis approach to identify recurring themes.

Results Three themes emerged: (1) Family silence and fragmented health histories masked inherited cancer risk (incomplete family histories, masculinity, stigma, and taboos discouraged communication). (2) Gaps in awareness and understanding of genetic testing, its importance, and implications (limited awareness of genetic testing, confusion about types of tests and results), and (3) Genetic testing extended the meaning of a CaP diagnosis beyond the individual, motivating participants to inform their families, and support screening and preventative action (responsibility to relatives, communication and documentation of family risk). **Translational Impact:** Findings provide a foundation for the development of culturally responsive communication and intervention tools to promote family risk communication and genetic testing for Black CaP survivors. When implemented in survivorship care, genetic counselling, and community settings, these resources may support informed testing decisions and earlier risk-based screenings among family members.

Funding[MA2] : This work was supported by the Department of Defense, Congressionally Directed Medical Research Programs (CDMRP), Prostate Cancer Research Program, Award No. PC230551, this work was funded by the Oklahoma Tobacco Settlement Endowment Trust (TSET), contract #00003615, and the OU Health Stephenson Cancer Center via an NCI Cancer Center

ABSTRACT TITLE

DISULFIRAM PROTECTS AGAINST DIET-INDUCED OBESITY BY REPROGRAMMING DIETARY LIPID FATE INDEPENDENT OF INFLAMMASOME SIGNALING

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ABSTRACT TITLE

DISULFIRAM PROTECTS AGAINST DIET-INDUCED OBESITY BY REPROGRAMMING DIETARY LIPID FATE INDEPENDENT OF INFLAMMASOME SIGNALING

Introduction: Obesity is a major health challenge with limited durable pharmacotherapies. Disulfiram (DSF), an FDA-approved drug, protects against diet-induced obesity, but the underlying mechanism remains unclear. Because DSF inhibits gasdermin D (GSDMD), a mediator of inflammatory cell death, we investigated whether its metabolic benefits result from suppression of GSDMD-dependent inflammasome signaling.

Methods: Male mice were fed a high-fat diet (HFD) with or without DSF beginning at diet initiation. Genetic knockout and antisense approaches targeting GSDMD, together with NLRP3-deficient mice, were used to determine inflammasome dependence. Metabolic phenotyping was integrated with pair-feeding, indirect calorimetry, stable-isotope lipid tracing, lipid profiling, and adipose tissue transcriptomics to determine how DSF protects against obesity.

Results: DSF markedly reduced HFD-induced weight gain, adiposity, and insulin resistance without sustained reduction in caloric intake. In contrast, genetic deletion or inhibition of GSDMD failed to protect against obesity, while DSF remained effective in mice lacking either GSDMD or NLRP3. Mechanistically, DSF produced a striking context-dependent shift in lipid handling. Under steady-state conditions, DSF increased fecal fatty acid excretion while suppressing basal lipid oxidation. During an acute lipid challenge, however, DSF increased tracer-derived carbon flux to CO₂ and accelerated systemic lipid clearance. These metabolic effects occurred without increased basal energy expenditure.

Translational Impact: Our findings redefine how DSF protects against obesity. Rather than acting primarily through inflammasome inhibition or appetite suppression, DSF redirects the fate of dietary lipids away from storage. These findings identify lipid partitioning as a potential therapeutic target and provide a new mechanistic framework for repurposing an established drug for metabolic disease.

FUNDING

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POSTER PRESENTATIONS

SESSION 1 | 8A

ABSTRACT TITLE

BLOOD TESTS FOR TUMOR DNA IN KIDNEY CANCER: DETECTING RECURRENCE AND PREDICTING OUTCOMES ACROSS STAGES OF DISEASE

Syed Sagib Balkhi¹, Hanxia Li², Ranjit Jasara¹, Shamanth Manjunatha Reddy¹, Michael Cookson³, Kelly Stratton³, Sanjay Patel³, Brian Cross³, Jonathan Heinlen³, Daniel Zhao², Adanma Ayanambakkam¹

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ABSTRACT TITLE

BLOOD TESTS FOR TUMOR DNA IN KIDNEY CANCER: DETECTING RECURRENCE AND PREDICTING OUTCOMES ACROSS STAGES OF DISEASE

Introduction. Kidney cancer sheds very little tumor DNA into the blood, so blood tests tracking it are less established here than in other cancers. Two questions remain open: whether a newer test reading the entire tumor genome detects returning cancer better than an older, narrower test, and whether a single positive result predicts outcome.

Methods. We reviewed 204 patients with kidney cancer and 1,079 blood draws from 2022 to 2026, all analyzed with a test built from the patient's own tumor. The recurrence analysis included patients whose cancer had not spread at diagnosis, counting a patient positive if any draw before spread was detectable. The outcome analysis used the first test in each disease state.

Results. Ninety-four patients with localized disease (67 older test, 27 newer) contributed 455 draws; 71 later developed spread, only 16 with a qualifying earlier draw. The newer test was positive at or before the date spread was diagnosed in 4 of 4 patients and the older test in 7 of 12 (difference 41.7 points, $p=0.245$). False positives were rare with both (98.2% versus 95.7%, $p=0.505$). The groups were not comparable: older-test patients had more draws (median 6 versus 3), longer follow-up (648 versus 178 days), and higher stage (85% versus 59%). A first positive test occurred in 10% of patients without spread and 61% of those with spread or locally advanced disease, predicting shorter time to progression in both (Table); deaths were too few for precise survival estimates.

Translational Impact. A single positive blood test identified patients at high risk of progression in both early and advanced kidney cancer, helping decide who needs closer monitoring or earlier treatment. The newer test's apparent edge is not proven, since the groups differed in era and testing intensity; a direct comparison with fixed monitoring schedules is needed.

Funding
none

Footnotes

CI, confidence interval. Older test, a tumor-informed assay built from the protein-coding portion of the tumor genome; newer test, one built from the whole tumor genome. Retrospective, single-center analysis.

Disease state	Endpoint	Hazard ratio (95% CI)	p
No spread	Time to progression	13.56 (2.26-81.37)	0.004
No spread	Overall survival	3.55 (0.32-39.21)	0.30
Spread or locally advanced	Time to progression	5.36 (2.18-13.16)	<0.001
Spread or locally advanced	Overall survival	4.44 (0.98-20.16)	0.053

Table. Outcomes after a positive versus negative first blood test, unadjusted.

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Syed Saqib Balkhi¹, Hanxia Li², Ranjit Jasaraj¹, Shamanth Manjunatha Reddy¹, Michael Cookson³, Kelly Stratton³, Sanjay Patel³, Brian Cross³, Jonathan Heinlen³, Daniel Zhao², Adanma Ayanambakkam¹

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POSTER PRESENTATIONS

SESSION 1 | 9A

ABSTRACT TITLE

TARGETTING SUCCINATE RECEPTOR 1 TO MITIGATE NECROTIZING ENTEROCOLITIS

Indranil Biswas₁, Shirley Wang₁, Adam Wilson₁, Neeladri Roy₁, Addison Franca₁, Hala Chaaban₁, Kathryn Y. Burge₁

ABSTRACT TITLE

TARGETTING SUCCINATE RECEPTOR 1 TO MITIGATE NECROTIZING ENTEROCOLITIS

Background: Necrotizing enterocolitis (NEC) is a deadly gastrointestinal disease of the preterm intestine leading 25–30% mortality. NEC is characterized by bacterial infection, dysbiosis, profound inflammation and intestinal injury, associated with microbial succinate content. SUCNR1 activation in immune cells, augment proinflammatory signaling, whereas SUCNR1–signaling in intestinal tuft cells is uniquely associated with anti-inflammatory responses. However, the precise role of SUCNR1 in regulating NEC susceptibility and contributing to NEC pathogenesis remains obscure.

Aims: We aimed to analyze the SUCNR1 expression in surgically resected preterm ileum. Intracellular SUCNR1 signaling will be studied in preterm enteroids, with and without human monocyte–derived macrophages.

Methods: We first characterized SUCNR1 expression in preterm ileal enteroids and human monocyte–derived macrophages through immunofluorescence, flow cytometry and western blots. Intestinal enteroids were analyzed in multiple conformations, including basolateral-out, apical-out, enteroid–derived monolayers. Moreover, succinate and SUCNR1–specific agonists and antagonists were employed to explore SUCNR1– signaling in basolateral-out and apical-out preterm enteroid conformations both in the presence and absence of macrophages.

Results: Contrary to the prevailing view that SUCNR1 expression is restricted to tuft cells, immunostaining of preterm human intestinal sections revealed ubiquitous SUCNR1 expression within the intestinal epithelium. Flow cytometry employing human preterm enteroids confirmed that SUCNR1 expression is enriched within, but not exclusive to, doublecortin–like kinase 1 (DCLK1)⁺ tuft cells relative to DCLK1[−] epithelial populations and immune cells. Intracellular SUCNR1 signaling in intestinal enteroids suggested a critical role of succinate in cellular proliferation and differentiation. Heightened inflammatory phenotype in enteroid–macrophage cocultures were observed through synergistic activation of SUCNR1 and TLR4.

Conclusion: These findings demonstrate that physiological concentration of succinate is beneficial for intestinal development. However, synergistic activation of SUCNR1 and TLR4 may induce intestinal injury, leading to increased preterm intestinal permeability and diffusion of microbial succinate and LPS across the epithelium, purportedly perpetuating the inflammation in NEC.

Funding: This research was funded by the NIH (P20GM134973 [KYB]) and the Children’s Health Foundation (KYB and HC).

POSTER PRESENTATIONS

SESSION 1 | 10A

ABSTRACT TITLE

DEVELOPMENT AND OPTIMIZATION OF BIODEGRADABLE LIPID NANOPARTICLES FOR MRNA DELIVERY TO THE LUNG ENDOTHELIUM

Evie Cameron-Mazin¹, Luke Rhym², Georgina Stephanie², Houman Maftoon¹, Safwan R. Yakubu¹, Allen Yujie Jiang², Rajith S. Manan², Daniel G. Anderson², Idris O. Raji^{1}*

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ABSTRACT TITLE

DEVELOPMENT AND OPTIMIZATION OF BIODEGRADABLE LIPID NANOPARTICLES FOR MRNA DELIVERY TO THE LUNG ENDOTHELIUM

Introduction: Messenger RNA therapeutics could enable treatment of pulmonary vascular disease, but safe and efficient delivery to lung endothelial cells remains a major barrier. This study aimed to identify and optimize biodegradable lipid nanoparticles that selectively deliver mRNA to the lung endothelium after intravenous administration.

Methods: A chemically diverse library of 60 biodegradable ionizable lipids was synthesized and formulated into lipid nanoparticles carrying reporter mRNA. Pooled in vivo screening was used to identify lead materials, followed by systematic tail-length optimization and peptide barcoding to refine formulation composition. Lead nanoparticles were then evaluated for endothelial targeting, tolerability, and delivery of messenger RNA and gene-editing cargo in mice and cultured human cells.

Results: Screening identified IR-177-7 as a lead lipid for pulmonary delivery, and tail-length optimization revealed a sharp performance maximal at C15. Peptide barcoding further identified an optimized formulation, C15-21, which produced more than threefold higher lung protein expression than a benchmark lung-targeting nanoparticle and transfected up to 79.3 percent of lung endothelial cells at a 0.3 mg/kg dose, with minimal activity in epithelial and immune cells. C15-21 was well tolerated at doses up to 0.75 mg/kg based on serum chemistry markers. In addition, these nanoparticles enabled nearly 95 percent of reporter knockout in HEK293 cells in vitro and up to 7.5 percent PTEN editing in lung endothelial cells in vivo.

Translational Impact: These findings establish C15-21 as a biodegradable platform for selective delivery of mRNA and gene-editing cargo to lung endothelial cells. This approach could support future protein replacement and genome-editing therapies for pulmonary vascular disorders while reducing dependence on nonbiodegradable or permanently cationic delivery systems.

Funding:

Sanofi, National Institutes of Health grant HL162564-02 (D.G.A.), Koch Institute Support Grant 5P30-CA14051 (D.G.A), start-up funds from the University of Oklahoma, Norman (I.O.R.).

Footnotes:

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ABSTRACT TITLE

RADIATION-INDUCED ENDOTHELIAL SENESENCE DRIVES CEREBROVASCULAR FRAGILITY AND MICROHEMORRHAGE: A SENOLYTIC-TARGETABLE MECHANISM OF ACCELERATED BRAIN VASCULAR AGING

Siva Sai Chandragiri^{1,2}, Roland Patai^{1,2}, Dorina Nagy^{1,2}, Rafal Gulej^{1,2}, Raghavendra Y. Nagaraja^{1,2}, Santhya Shanmugarama^{1,2}, Kiana V. Kordestan^{1,2}, Shoba Ekambaram^{1,2}, Evelyn Brunner^{1,2}, Rebeka Kristof^{1,2}, Mark Nagykaldi^{1,2}, Zoltan Ungvari^{1,2,3}, Anna Csiszar^{1,2,3}

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ABSTRACT TITLE

RADIATION-INDUCED ENDOTHELIAL SENESENCE DRIVES CEREBROVASCULAR FRAGILITY AND MICROHEMORRHAGE: A SENOLYTIC-TARGETABLE MECHANISM OF ACCELERATED BRAIN VASCULAR AGING

Introduction: Whole brain radiotherapy (WBRT) is a cornerstone treatment for patients with multiple brain metastases and is routinely used to prevent recurrence after resection of cerebral lesions, yet nearly half of long-term survivors develop debilitating, progressive cognitive impairment. Growing evidence implicates radiation-driven vascular injury, particularly endothelial senescence within the neurovascular unit, though its role in destabilizing brain vasculature remains unresolved. We hypothesized that irradiation-induced endothelial senescence drives cerebrovascular fragility and cerebral microhemorrhages (CMHs) resembling premature vascular aging, and that clearing senescent cells with the senolytic agent navitoclax (ABT263) would limit this fragility and its neurological consequences.

Methods: Three-month-old mice underwent fractionated whole brain irradiation (5 Gy, twice weekly for four weeks) or sham exposure then received ABT263 or vehicle. At two months post-irradiation senescent endothelial and microglial populations were profiled by flow cytometry and single-cell RNA sequencing. Mice were challenged with angiotensin-II and L-NAME to provoke vascular fragility and resulting microhemorrhages were quantified histologically alongside lesion burden. Gait regularity was tracked as functional correlate of vascular injury.

Results: Irradiated mice showed markedly elevated endothelial senescence, earlier-onset gait abnormalities and greater microhemorrhage burden and lesion area than sham-irradiated, vehicle-treated controls. Survival analysis showed irradiated mice developed neurological deficits sooner and died earlier from deficit-related complications. ABT263 treatment reversed this trajectory, lowering endothelial senescence and microhemorrhage formation while extending deficit-free survival and preserving gait function.

Translational Impact: This work establishes endothelial senescence as mechanistic, therapeutically actionable driver of radiation-induced cerebrovascular aging, linking a hallmark of aging directly to microbleed-related injury seen in cerebral small vessel disease. Because senolytics reduced vascular fragility and neurological decline in this model these data provide preclinical rationale for testing senolytic co-therapy alongside cranial radiotherapy to protect long-term cognitive outcomes in brain cancer survivors and support developing endothelial senescence biomarkers to monitor radiation-induced vascular injury in future trials.

FUNDING: American Heart Association Predoctoral Fellowship (25PRE1378958), NIH/NIA (1R01AG068295-01)

POSTER PRESENTATIONS

SESSION 1 | 14A

ABSTRACT TITLE

INVESTIGATING THE EFFECTS OF METABOLISM MODULATING DRUG ON VIABILITY OF CANCER CELLS

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ABSTRACT TITLE

INVESTIGATING THE EFFECTS OF METABOLISM MODULATING DRUG ON VIABILITY OF CANCER CELLS

Introduction: Cancer cells undergo metabolic reprogramming to support rapid growth, proliferation, and survival, making metabolic pathways attractive therapeutic targets. Mitochondria are central to these processes through their roles in energy production, biosynthesis, redox regulation, and apoptosis. One mitochondrial enzyme of particular interest is dihydroorotate dehydrogenase (DHODH), which catalyzes a critical step in de novo pyrimidine biosynthesis. Because rapidly dividing cancer cells require large amounts of nucleotides for DNA and RNA synthesis, DHODH activity is often increased in malignant cells. Inhibition of DHODH disrupts pyrimidine production, impairs cellular metabolism, and can suppress cancer cell growth, making it a promising therapeutic target in several cancers, including bladder cancer.

Methods: Here we evaluated leflunomide, an FDA-approved DHODH inhibitor, for its effects on human bladder cancer cells as a potential drug-repurposing strategy. Human bladder cancer cells were cultured both as two-dimensional monolayers and three-dimensional spheroids and treated with increasing concentrations of leflunomide, alongside untreated controls. Cell viability, apoptosis, and spheroid growth were assessed at multiple time points following treatment. Experiments were performed with appropriate replicates, and statistical analyses were conducted to compare treatment groups.

Results: Leflunomide treatment produced a concentration-dependent reduction in bladder cancer cell viability and induced apoptosis in both monolayer and spheroid cultures. In addition, treated spheroids demonstrated reduced growth compared with untreated controls, indicating impaired tumor cell expansion. These findings suggest that inhibition of DHODH-mediated pyrimidine synthesis can adversely affect bladder cancer cell survival and growth.

Translational Impact: This study provides preliminary evidence supporting the investigation of DHODH and pyrimidine metabolism as therapeutic targets in bladder cancer and highlights the potential of leflunomide as a repurposed metabolic therapy for this disease.

Funding: Center for Cancer Prevention and Drug Development, Stephenson Cancer Center.

ABSTRACT TITLE

EVALUATING CYTOKINE-MEDIATED NEUROVASCULAR INJURY IN HUMAN CEREBROVASCULAR CELL SPHEROIDS

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ABSTRACT TITLE

EVALUATING CYTOKINE-MEDIATED NEUROVASCULAR INJURY IN HUMAN CEREBROVASCULAR CELL SPHEROIDS

Introduction: Blood brain barrier (BBB) is a semi-permeable membrane composed of endothelial cells, astrocytes, and pericytes. BBB dysfunction post-stroke is a hallmark of secondary neuroinflammation which worsens neuronal injury. BBB injury phenotype is exhibited by altered expression of endothelial tight junction proteins (zona-occludens-1 (ZO-1)) and astrocytic cytoskeletal protein (glial fibrillary acidic protein (GFAP)); in response to inflammatory cytokines- interleukin-1 beta (IL-1 β) and tumor necrosis factor-alpha (TNF- α). Existing endothelial monoculture models fail to capture neurovascular unit interactions essential for barrier integrity and recovery. Addressing this technical gap, we evaluated a human multicellular 3D BBB organoid for modelling cytokine-mediated human stroke injury.

Methods: Organoids were generated using a 1:1:1 ratio of human-derived BBB cells. Post 48h of formation, spheroids were treated for 24h with IL-1 β (10-25ng/ml) and TNF- α (20-50ng/ml) to model stroke-like conditions. Organoids were fixed and immunostained for ZO-1 and GFAP. Confocal microscopy and ImageJ quantification assessed neurovascular cell marker expression, and cytokine-dependent changes in mean fluorescence intensity (MFI).

Results: Organoids formed reproducibly and expressed CD31⁺ endothelial cells, GFAP⁺ astrocytes, and NG2⁺ pericytes; supporting formation of human multicellular BBB organoids. IL-1 β -treated organoids showed variable ZO-1 expression whereas TNF- α treated organoids showed a slight reduction in MFI from 44.81 (20ng/ml) to 27.40 (50ng/ml), corresponding to a ~40% decrease; however, this trend was not statistically significant. In contrast, IL-1 β (25ng/ml) exhibited a statistically significant increase in GFAP expression as compared to untreated controls. Together, these data support our hypothesis that post-stroke elevation of TNF-alpha and IL-1beta decreases tight junction integrity and astrogliosis in the human BBB.

Translational Impact: BBB spheroids will close a key technical gap in therapeutic development, an area that currently relies on rodent models. Data generated from resolving inflammatory damage to BBB spheroids will guide the selection of neurovascular repair treatments post-stroke, and potentially uncover molecular mechanisms involved in stroke-related neurovascular injury.

Funding: Support is gratefully acknowledged from the NIGMS of NIH (R35GM150970, to JRC) and the OU Undergraduate Research Opportunities Program (to IA). Confocal microscopy was performed in the COBRE Medical Imaging Technology Development Core (MITDC), funded by the NIGMS of NIH (P20GM135009)

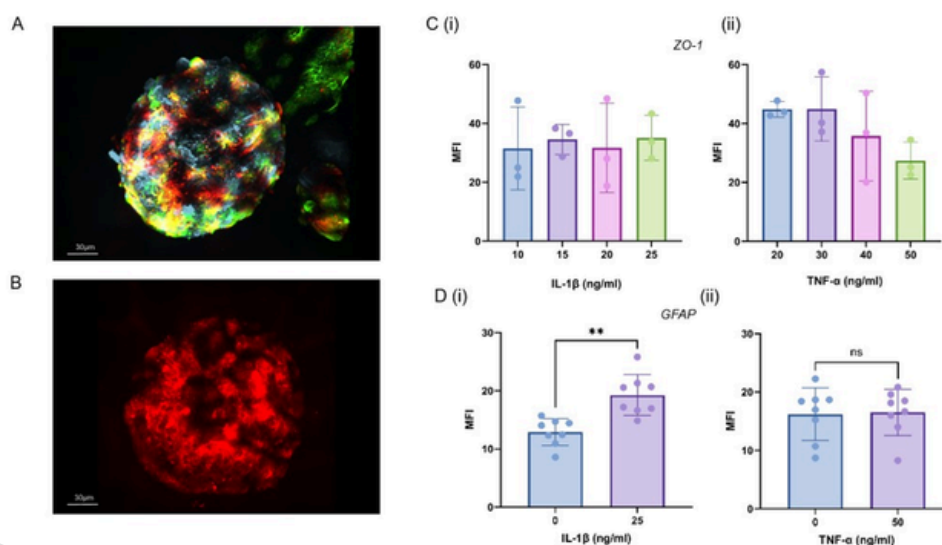


Fig. Establishment and cytokine-mediated injury screening of 3D human BBB organoids. (A) Workflow for (i) organoid generation and (ii) Immunofluorescence staining. (A) Organoids showing CD31⁺ endothelial cells (blue), GFAP⁺ astrocytes (red) and NG2⁺ pericytes (green). (B) Confocal ZO-1 staining (red) 24h after TNF- α treatment (30ng/ml). MFI Quantification of (C) ZO-1 and (D) GFAP following IL-1 β and TNF- α treatment across tested cytokine concentrations.

POSTER PRESENTATIONS

SESSION 1 | 16A

ABSTRACT TITLE

ATOVAQUONE ATTENUATES PANCREATIC CANCER–INDUCED CACHEXIA

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ABSTRACT TITLE

ATOVAQUONE ATTENUATES PANCREATIC CANCER–INDUCED CACHEXIA

Abstract

Cancer-associated cachexia is a multifactorial metabolic syndrome characterized by systemic inflammation, progressive skeletal muscle wasting, and poor therapeutic outcomes. Pancreatic cancer is among the malignancies most frequently associated with cachexia, contributing significantly to patient mortality. Given the lack of FDA-approved therapies to counteract cachexia, drug repurposing offers a promising alternative strategy. Atovaquone, an FDA-approved antimalarial compound with potent anti-inflammatory properties, has previously been shown to inhibit STAT3 activation—a key driver of cancer-induced wasting.

In this study, we evaluated the anti-cachectic potential of atovaquone in pancreatic cancer–induced cachexia using both in vitro and in vivo models. Treatment of myotubes with S2013 pancreatic cancer cell–conditioned media (CM) induced classical cachectic responses, including upregulation of atrogin-1 and MuRF1 and downregulation of myosin heavy chain. Co-treatment with atovaquone reversed these effects, as confirmed by real-time PCR and Western blot analysis, indicating attenuation of muscle degradation. Mechanistically, atovaquone inhibited mitochondrial oxidative phosphorylation, leading to a reduced oxygen consumption rate (OCR) and activation of a compensatory glycolytic shift. This was evidenced by an increase in extracellular acidification rate (ECAR), glucose uptake, and lactate production. Enhanced glycolysis restored cellular energy balance and suppressed catabolic signaling, thereby protecting against CM-induced myotube atrophy. Inhibition of glycolysis with 2-deoxy-D-glucose (2-DG) abrogated the protective effects of atovaquone, underscoring the importance of glycolytic compensation in its mechanism of action.

Collectively, our findings demonstrate that atovaquone mitigates cancer-induced muscle wasting by reprogramming myotube metabolism from oxidative phosphorylation to glycolysis, thus preserving muscle mass and function. These results highlight atovaquone as a promising metabolic intervention for managing pancreatic cancer–associated cachexia.

Keywords: Atovaquone • Cancer cachexia • Mitochondrial oxidative phosphorylation • Glycolytic shift • Muscle wasting

Acknowledgment of Funding:

This work was supported by the Department of Oncology Science, The University of Oklahoma

POSTER PRESENTATIONS

SESSION 1 | 19A

ABSTRACT TITLE

TARGETED ANNEXIN A5 MERTANSINE CONJUGATE FOR LEUKEMIA THERAPY

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ABSTRACT TITLE

TARGETED ANNEXIN A5 MERTANSINE CONJUGATE FOR LEUKEMIA THERAPY

Introduction: Leukemia treatment remains limited by relapse, resistance, and systemic toxicity from insufficient tumor selectivity. Mertansine (DM1) is a potent microtubule inhibitor, but its hydrophobicity and off-target toxicity restrict systemic use. We developed an annexin A5-DM1 conjugate (ANXA5-DM1) to target phosphatidylserine, which is externalized on malignant cells.

Methods: ANXA5 was conjugated to DM1 using the heterobifunctional crosslinker sulfo-SMCC, producing a noncleavable thioether linkage. Binding and cytotoxicity were evaluated in three murine leukemia cell lines: C1498 (myeloid acute myeloid leukemia), P388D1 (monocytic/lymphoid), and L1210 (lymphoid acute lymphoblastic leukemia). Calcium-dependent binding was evaluated, and apparent dissociation constants (K_d) were estimated. Viability was measured by a resazurin reduction assay after 72 hours of exposure (100 μ M to 1 pM).

Results: ANXA5-DM1 showed subnanomolar binding to C1498, P388D1, and L1210 cells, with apparent K_d values of 0.37, 0.93, and 0.32 nM, respectively. Conjugate IC_{50} values were 1.03, 1.20, and 0.26 nM, respectively, compared with 4.5, 264, and 93 nM for free DM1. Thus, conjugation increased in vitro potency approximately 4- to 360-fold across the models.

Translational Impact: By coupling DM1 to ANXA5, this strategy combines the potency of a microtubule inhibitor with calcium-dependent targeting of cell-surface phosphatidylserine. Because phosphatidylserine is a lipid target shared across malignant phenotypes rather than a single protein antigen, ANXA5-DM1 may act across heterogeneous leukemic populations and may be less vulnerable to antigen-loss escape than receptor-targeted therapies. The noncleavable linker is also designed to limit premature DM1 release. These properties support evaluation in models of relapsed and treatment-resistant leukemia.

Funding: University of Oklahoma Foundation.

POSTER PRESENTATIONS

SESSION 1 | 20A

ABSTRACT TITLE

AN INTEGRATIVE FRAMEWORK FOR IDENTIFYING REGULATORY VULNERABILITIES IN MYC-DRIVEN ALL

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ABSTRACT TITLE

AN INTEGRATIVE FRAMEWORK FOR IDENTIFYING REGULATORY VULNERABILITIES IN MYC-DRIVEN ALL

INTRODUCTION: Acute lymphoblastic leukemia (ALL) is the most common and deadliest pediatric malignancy, accounting for ~20% of childhood cancer diagnoses and related deaths. Relapse remains a major clinical challenge, and outcomes for high-risk, refractory and relapsed disease are poor. MYC is a central oncogenic driver in ALL, but its essential roles in transcription, DNA replication, and epigenomic organization complicate efforts to therapeutically target the oncoprotein. Defining the downstream regulatory systems through which MYC drives aggressive disease is therefore critical. To address this, we developed a transgenic zebrafish model overexpressing human MYC that recapitulates human ALL, enabling comparison of healthy, preleukemic, and leukemic cells.

METHOD: We generated transcriptomic, DNA replication timing, and MYC chromatin binding profiles across healthy, preleukemic, and leukemic populations. This multi-modal approach serves two complementary purposes. First, genes supported by concordant trends across all three datasets are high-confidence, cross-validated therapeutic targets, while the union of the datasets substantially expands the pool of candidate regulatory features. Second, because transcription and replication are normally tightly coupled, directly comparing these modalities reveals sites where coordination breaks down due to aberrant MYC activity.

RESULTS: Our approach yielded a core set of high-confidence candidates corroborated across all three datasets and a broader set of candidates unique to individual modalities, which may have been missed by any single approach. Several candidates show strong prognostic associations in pediatric ALL patient data. Functional validation is currently underway in both our zebrafish model and human ALL cell lines.

TRANSLATIONAL IMPACT: This multi-modal strategy offers a generalizable framework for identifying therapeutic vulnerabilities downstream of essential oncoproteins like MYC, both by expanding the pool of candidate regulatory features and by revealing where MYC's normally coordinated functions become decoupled during malignant transformation. These candidates represent potential biomarkers and therapeutic targets for improving risk stratification and treatment of high-risk ALL.

FUNDING: Presbyterian Health Foundation Team Science Award, Hyundai Hope on Wheels Hope Scholar Grant, Hyundai Hope on Wheels Impact Award, American Cancer Society STRONG Program, W.J. Jones Family Foundation

ABSTRACT TITLE

TRANSLATIONAL STRATEGIES FOR ENGINEERING PROTECTIVE IMMUNITY AGAINST CANCER AND RESPIRATORY VIRAL INFECTIONS

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ABSTRACT TITLE

TRANSLATIONAL STRATEGIES FOR ENGINEERING PROTECTIVE IMMUNITY AGAINST CANCER AND RESPIRATORY VIRAL INFECTIONS

Abstract

Introduction: Protective immunity is essential for preventing cancer recurrence and infectious diseases, yet current strategies are often limited by antigenic heterogeneity, immune escape, and rapidly evolving pathogens. Host-directed immunomodulation offers a promising approach to enhance immune protection independently of specific antigenic variations. This study evaluates host-directed immunomodulatory strategies that promote protective immunity against cancer and viral infections, and explores mechanisms of durable immune protection.

Methods: Galactated chitosan (GC) was evaluated as an immunostimulant in two disease models. In pancreatic cancer, GC was incorporated into a graphene oxide-based photothermal therapy (GO/GC + PTT) and evaluated for therapeutic efficacy, tumor rechallenge survival, and immune activation. In respiratory viral models, intranasal GC was administered as a prophylactic or vaccine adjuvant, and survival, clinical outcomes, and immune responses were assessed. Mechanistic studies were performed using primary mouse dendritic cells and macrophages.

Results: Treatment with GO/GC + PTT eliminated primary tumors and provided long-term protection, with 85% of mice surviving tumor-free for 100 days. Following tumor-rechallenge, 40% of mice showed durable protection against cancer recurrence. Prophylactic intranasal administration of GC protected 67% of mice against lethal SARS-CoV-2 challenge and protected infected lungs from tissue damage. Intranasal vaccination with GC + SARS-CoV-2 antigens allowed 90% of mice to survive lethal challenge. Protection in both disease models was associated with enhanced immune activation characterized by type I interferons, inflammatory mediators, antigen-presenting cell maturation, and humoral immunity. These findings demonstrate that immune stimulation can generate durable protection across multiple disease settings.

Translational Impact: This study supports host-directed immunomodulation as a promising and versatile translational strategy promoting protective immunity against cancer and respiratory viral infections, while identifying type I interferons as promising candidates for future translational immune monitoring. Therefore, this work lays the foundation for the clinical development of immunotherapies, prophylactic interventions, and vaccines.

Funding

National Cancer Institute (R01CA205348 and R01CA269897), National Institute of General Medical Sciences of the National Institutes of Health (P20GM162339), Oklahoma Center for the Advancement of Science and Technology (HF20-019 and HR23-069), IBEST-OUHSC Funding for Interdisciplinary Research of the University of Oklahoma, OKBioStart (08-79-05678).

Keywords: Protective immunity; Antitumor immunity; Antiviral immunity; Biopolymers; Tumor ablation; Immunotherapy; Prophylaxis; Vaccination; Pancreatic cancer; Respiratory viruses; Prolonged survival; Type I interferons.

POSTER PRESENTATIONS

SESSION 1 | 24A

ABSTRACT TITLE

OSTRINIA FURNACALIS PHEROMONE-BINDING PROTEIN 2: ROLE OF ARG70 IN LIGAND RELEASE

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ABSTRACT TITLE

OSTRINIA FURNACALIS PHEROMONE-BINDING PROTEIN 2: ROLE OF ARG70 IN LIGAND RELEASE

Olfaction is the sense of smell by which organisms detect chemical signals in the environment. In moths, males rely on olfaction to detect female sex pheromones and locate mates. Pheromone-binding proteins (PBPs) in the male antennae mediate the transport of hydrophobic pheromones through the aqueous sensillar lymph to olfactory receptor neurons.

The Asian corn borer, *Ostrinia furnacalis* (Ofur), is a major agricultural pest responsible for significant crop damage and is difficult to control due to insecticide resistance. Consequently, understanding the molecular basis of pheromone binding and release may facilitate the development of environmentally sustainable alternatives to insecticides. OfurPBP2, a key olfactory protein, contains two regulatory features: a histidine-arginine gate and a C-terminal gate that controls ligand binding and release. Unlike other common lepidopteran PBPs, which possess a conserved His70–His95 histidine gate, OfurPBP2 contains an Arg70 residue in place of His70. Structural studies suggest that Arg70 forms a novel Arg70–His88 gate that replaces the classical histidine gate. Whether the Arg70–His88 gate plays any role in regulating the pH-dependent pheromone binding and release is not known. Furthermore, it has also been reported that OfurPBP2 may release ligand through a molten globule-like state rather than the canonical pH-dependent conformational switch observed in other lepidopteran PBPs.

To investigate the functional role of Arg70, an R70A mutant was generated, expressed in *Escherichia coli* using both IPTG induction as well as autoinduction media. While IPTG induction produced inclusion bodies requiring refolding, autoinduction yielded soluble recombinant protein. Both the refolded and soluble proteins were purified using anion-exchange chromatography and size-exclusion chromatography. Circular-dichroism, fluorescence spectroscopy, and nuclear magnetic resonance spectroscopy will be used to characterize the effect of mutation on protein function. These studies will provide mechanistic insight into OfurPBP2 function and would support the development of environmentally sustainable strategies to limit use of insecticides.

Our research was financially supported by NSF Award CHE-2402694 to Dr. Smita Mohanty.

ABSTRACT TITLE

COMPOSITION DEFINED PAYLOAD FREE NANOGEL DRUG CARRIERS FOR MACROPHAGE DIRECTED IMMUNOMODULATION

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ABSTRACT TITLE

COMPOSITION DEFINED PAYLOAD FREE NANOGEL DRUG CARRIERS FOR MACROPHAGE DIRECTED IMMUNOMODULATION

Introduction: Immune dysregulation drives cancer and chronic inflammatory disease, yet drug carrier design often treats the carrier as a passive payload vehicle. We hypothesize that composition defined hydrogel nanoparticles can be engineered as payload free immunomodulatory platforms because their bulk composition, crosslinking, and surface chemistry can be independently tuned.

Methods: We synthesized a 20 member library of PEG shelled, disulfide crosslinked, pH responsive cationic nanogels by ARGET ATRP, varying tertiary amine identity, hydrophobe identity, and cationic to hydrophobic ratio. Nanogels were characterized by FT IR, NMR, quantitative NMR, DLS, and zeta potential analysis. Macrophage responses were evaluated in RAW 264.7 cells and primary murine bone marrow derived macrophages using viability, cytokine, flow cytometry, and targeted transcriptomic assays. The platform is being expanded across cationic, anionic, and zwitterionic polymer libraries.

Results: Nanogels showed composition dependent colloidal behavior, with many formulations in the 150 to 250 nm range and zeta potential transitions from about +30 mV at acidic pH toward near neutral values by pH 8.5. DEAEMA rich formulations maintained greater than 60% viability at high dose yet were strongly immunoactive, inducing significant IL 6 and TNF alpha secretion, including IFN gamma primed IL 6 levels of approximately 80 to 90 pg/mL, and upregulating iNOS and CD206. Targeted transcriptomics showed that DEAEMA-rich nanogels resembled IFN-gamma/LPS-like inflammatory programs in RAW 264.7 macrophages while inducing a more selective inflammatory-remodeling state in primary macrophages, including increased Clec4e, Slc7a11, Gdf15, and Il1rn.

Translational Impact: These findings support nanogel composition, including charge identity, charge density, hydrophobe identity, crosslinking, and headgroup chemistry, as a programmable design space for self therapeutic or adjuvant like drug carrier materials. Lead formulations will advance toward in vivo studies of tumor associated macrophage reprogramming and tumor immune microenvironment remodeling.

Funding: National Institute of General Medical Sciences R35GM150970, Harold Hamm Diabetes Center, National Cancer Institute Cancer Center Support Grant P30CA225520, National Institute of General Medical Sciences P30GM154635

POSTER PRESENTATIONS

SESSION 1 | 27A

ABSTRACT TITLE

**STATE-RESOLVED REGULATORY PROFILING IDENTIFIES PRODUCT-SPECIFIC
TRANSCRIPTION FACTOR AND PATHWAY ACTIVITY IN AXI-CEL AND TISA-CEL INFUSION
PRODUCTS**

Trenton Gibson¹, Taha Al-Juhaishi¹

¹University of Oklahoma College of Medicine

ABSTRACT TITLE

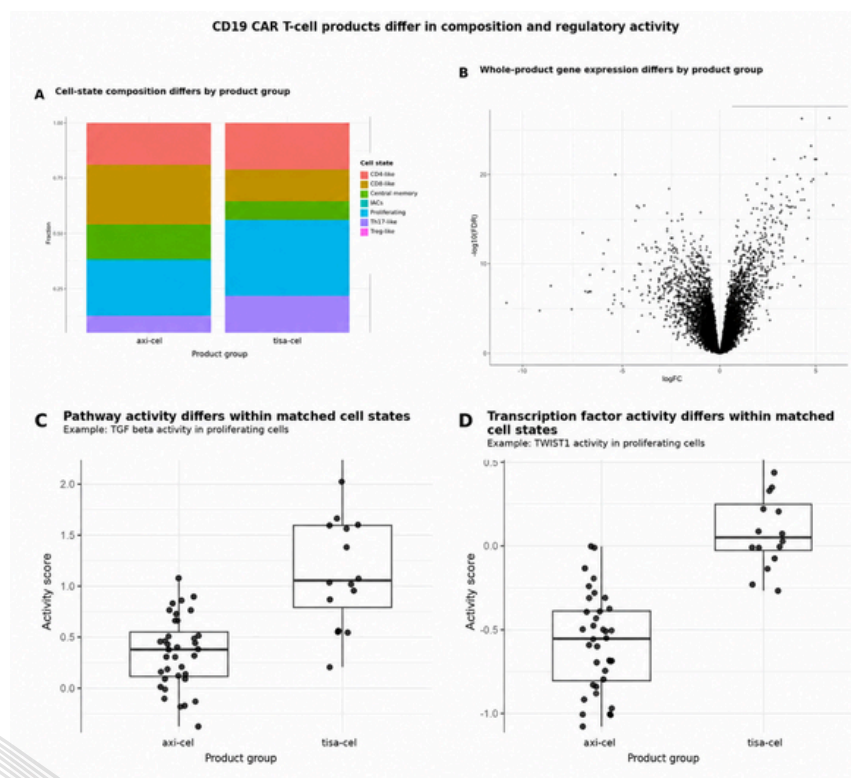
STATE-RESOLVED REGULATORY PROFILING IDENTIFIES PRODUCT-SPECIFIC TRANSCRIPTION FACTOR AND PATHWAY ACTIVITY IN AXI-CEL AND TISA-CEL INFUSION PRODUCTS

Introduction: Axicabtagene ciloleucel (axi-cel) and tisagenlecleucel (tisa-cel) are approved CD19 CAR T-cell therapies for relapsed/refractory large B-cell lymphoma. A prior single-cell study found axicabtagene ciloleucel products enriched for central-memory/effector-like T-cell states and tisagenlecleucel products enriched for proliferative states, with broad product-associated gene-expression differences. Whether these differences reflect cell-state abundance alone or state-intrinsic regulatory programs remains incompletely defined. We sought to determine whether these products differ in inferred pathway and transcription factor activity within matched cell states.

Methods: We analyzed a published single-cell RNA-sequencing dataset of 57 pre-infusion CAR T-cell products, including 39 axi-cel and 18 tisa-cel products. After quality control, 92,191 cells were retained and harmonized into CD4-like, CD8-like, central-memory-like, proliferating, Th17-like, Treg-like, and IAC states. Products were compared by state composition, whole-product pseudobulk differential expression, and state-resolved mechanistic modeling. Sample-by-state pseudobulks were generated, pathway activity was inferred with PROGENy, transcription factor activity with DoRothEA, and product effects were modeled with and without response-group adjustment.

Results: Axi-cel products had higher CD8-like and central-memory-like fractions, while tisa-cel products had higher proliferating and Th17-like fractions. Whole-product analysis showed stronger product-associated transcriptional signal than pooled responder-versus-non-responder comparisons. PROGENy identified differences in TGF beta, NF kappa B, estrogen, TNF alpha, hypoxia, JAK-STAT, PI3K, and WNT-related pathways. DoRothEA identified state-specific transcription factor differences involving NFIC, ZBED1, TWIST1, E2F-family factors, MYC/MYCN, JUND, HOXA9, RXRA, and HOXB7. Proliferating cells showed the strongest DoRothEA signal, with 119 robust associations after response adjustment. Across states, 442 DoRothEA and 40 PROGENy associations met robust same-direction criteria.

Translational Impact: These products differ in cell-state abundance and stable within-state regulatory programs. State-resolved profiling may support product quality assessment, biomarker development, and manufacturing optimization to improve CAR T-cell expansion, persistence, efficacy, and toxicity risk stratification.



POSTER PRESENTATIONS

SESSION 1 | 29A

ABSTRACT TITLE

EPIGENETIC REGULATION OF TROPHOBLAST STEM CELL DIFFERENTIATION: THE ROLE OF PRMT5 IN PLACENTAL DEVELOPMENT

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ABSTRACT TITLE

EPIGENETIC REGULATION OF TROPHOBLAST STEM CELL DIFFERENTIATION: THE ROLE OF PRMT5 IN PLACENTAL DEVELOPMENT

Introduction: Defects in trophoblast and placental development are associated with pregnancy-related complications such as preterm labor, fetal growth restriction, and preeclampsia, which remain poorly understood at molecular and genetic levels. Trophoblast cells from placenta can differentiate into (1) Extravillous trophoblasts (EVT) and (2) Syncytiotrophoblasts (STB). Our study shows the role of protein arginine methyltransferase PRMT5 which is a master epigenetic regulator in TS cell differentiations.

Methods: We used an in vitro model of human trophoblast cells (TS cells), inducible to differentiate into various placental lineages such as EVT and STB. This model provides an ideal system to study the effects of gene manipulation on TS cell development and differentiation. Furthermore, we used lentiviral-mediated short hairpin RNAs (shRNA) and guide RNA (gRNA) to knock down and knockout the expression of PRMT5 in TS cell.

Results: Preliminary single-cell and bulk RNA-seq analyses, as well as qPCR of hTS cells, demonstrated that PRMT5 transcripts are highly expressed in hTS cells, a finding further supported by Western blot analysis. We successfully knocked down PRMT5 transcript expression in hTS cells, confirmed by qPCR and Western blot analysis. We investigated the role of PRMT5 in the proliferation, morphology, and potential to maintain the stem cell state of hTS cells through various assays. Knockdown of PRMT5 did not impact hTS cell proliferation or morphology. However, PRMT5 knockdown affected the differentiation of hTS cells into both STB and EVT lineages. To validate our results, we conducted qPCR analysis on transcripts specific to STB and EVT lineages.

Translational Impact: By elucidating the role of PRMT5 as master regulator in TS cell differentiation, a foundation for prenatal diagnostics and epigenetic therapeutics can be established that can help in preventing pregnancy associated complications.

POSTER PRESENTATIONS

SESSION 1 | 31A

ABSTRACT TITLE

TOGARAM1 LOSS DISRUPTS THE CILIARY PROTEOME, CONTRIBUTING TO CELLULAR PATHOPHYSIOLOGY

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ABSTRACT TITLE

TOGARAM1 LOSS DISRUPTS THE CILIARY PROTEOME, CONTRIBUTING TO CELLULAR PATHOPHYSIOLOGY

Introduction: Cilia are signaling organelles required for life. Despite being partially membrane-enclosed, cilia maintain distinct, dynamically-regulated protein compositions. Multiple ciliary proteome regulators are implicated in ciliopathies, suggesting aberrant proteomes drive disease. However, pathogenic proteomic disruptions remain understudied in ciliopathies like the neurodevelopmental disorder Joubert syndrome (JS). Defects in >40 genes cause JS, including TOGARAM1. TOGARAM1's function is emerging, but its role in ciliary proteome regulation is unknown. To address this gap, we probed for proteomic perturbations across cilia types.

Methods: Using differential data-independent acquisition mass spectrometry, we quantified changes in isolated TOGARAM1 knockout human primary cilia and SHF1 (TOGARAM1 ortholog) knockout *Chlamydomonas* motile cilia compared to controls. Bioinformatic analysis predicted affected functions and pathways for future study.

Results: First, we compared TOGARAM1-knockout and wild-type HEK293T primary cilia. 367 proteins were significantly altered in mutant cilia versus controls. Ingenuity Pathway Analysis predicted disruptions of cellular processes and signaling pathways, including expected and novel cascades like Hedgehog and Ephrin signaling, respectively. We next investigated SHF1 loss in *Chlamydomonas* motile cilia. We identified 309 significant differentially abundant proteins. DAVID bioinformatics detected functional trends among perturbed proteins, highlighting distinct processes aberrant in mutant motile cilia, such as metabolism and ion transport. Finally, we searched for shared proteomic defects across models. Lipid metabolism and metal-binding proteins were accumulated in mutant cilia, whereas calcium-binding and ankyrin repeat proteins were depleted across models. We uncovered 49 shared proteins that overlapped with published TOGARAM1 interactors. Five proteins were depleted across models, indicating TOGARAM1 may promote their ciliary localization.

Translational Impact: This novel comparative study illuminates how loss of ciliary proteins can discompose the ciliary proteome, leading to functional consequences. Our approach can be applied to probe proteomic changes in other ciliopathies, bolstering our understanding of proteomic disruption in pathogenesis, identifying key relevant pathways, and illuminating new therapeutic targets.

This research was funded by grant #R00HD100554 to JCV

POSTER PRESENTATIONS

SESSION 1 | 33A

ABSTRACT TITLE

EFFECTS OF TRANSCRANIAL PHOTOBIMODULATION ON MARKERS OF NEUROVASCULAR COUPLING AND COGNITIVE FUNCTION: A PILOT RANDOMIZED DOUBLE-BLINDED CLINICAL TRIAL

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ABSTRACT TITLE

EFFECTS OF TRANSCRANIAL PHOTOBIMODULATION ON MARKERS OF NEUROVASCULAR COUPLING AND COGNITIVE FUNCTION: A PILOT RANDOMIZED DOUBLE-BLINDED CLINICAL TRIAL

Introduction: Over 14 million Americans aged 65 and older experience cognitive impairment, which can progress to vascular dementia. This is facilitated by age-related impairment of neurovascular coupling (NVC) which refers to insufficient upregulation of local cerebral blood flow to active brain regions. Transcranial photobiomodulation (tPBM) is a non-invasive neuromodulation technique targeting age-related mitochondrial dysfunction in brain cells via infrared light and may lead to cognitive benefits by improving NVC. This randomized, sham-controlled double-blinded study investigates whether 4-weeks of tPBM intervention can improve NVC and cognitive outcomes and if age plays a role in such effects ([clinicaltrials.gov: NCT07209683](https://clinicaltrials.gov/ct2/show/study/NCT07209683)).

Methods: This ongoing project will have 40 participants (20 male, 20 female) of five age groups (21–85 years) randomized in a 1:1 ratio to receive either active tPBM or sham therapy. Participants complete a baseline visit, a 4-week follow-up visit, and 12–14 self-administered 20-minute sessions every other day. The NIH Toolbox and n-back tasks are used to assess cognitive function. NVC is measured with functional near-infrared spectroscopy (fNIRS), transcranial doppler, and dynamic vessel analysis.

Results: To date, 19 participants have completed the full protocol, out of these, 16 individuals matched within the same age and gender strata: 4 pairs in the young group (2 female pairs), 3 in the middle-aged group (1 female pairs) and 1 pair of older women. Preliminary analyses of these strata revealed clinically meaningful improvement in cognition fluid composite score obtained by NIH toolbox. Markers of NVC did not show a significant variation.

Translational Impact: Data collection is expected to finish by November 30. Analyses will continue through 2027. We hypothesize that 4 weeks of tPBM will improve NVC, particularly in older adults, supporting cognitive function. These findings could establish tPBM as a non-invasive strategy for mitigating age-related cognitive impairment.

Funding: P20GM125528, U54GM10493

ABSTRACT TITLE

LONGITUDINAL CHANGES IN INTER-ARTICULATOR SPEECH COORDINATION FOLLOWING PARTIAL GLOSSECTOMY AND LINGUAL RADIATION

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ABSTRACT TITLE

LONGITUDINAL CHANGES IN INTER-ARTICULATOR SPEECH COORDINATION FOLLOWING PARTIAL GLOSSECTOMY AND LINGUAL RADIATION

Abstract

Introduction: Oral and oropharyngeal cancers (OC) and their treatments frequently disrupt speech motor function by injuring key articulatory structures, particularly the tongue. Surgery and radiotherapy (RT) can alter efferent motor commands, afferent sensory feedback, and tissue elasticity, resulting in persistent motor speech impairments. However, the physiological mechanisms linking treatment-related structural changes to speech impairment remain poorly understood. This ongoing longitudinal study investigates how treatment-induced alterations in lingual sensorimotor function contribute to speech impairment and recovery following treatment for lingual tumors.

Methods: We here use electromagnetic articulography (EMA) to quantify real-time tongue and jaw kinematics before treatment and for up to three years following treatment in patients with tongue cancer, alongside age-matched healthy controls. During production of vowel-consonant-vowel sequences varying in vowel identity (/a/, /ε/), consonant (/t/, /d/), production rate (normal vs. fast), and syllable stress (stressed vs. unstressed), EMA is used to track and record articulatory movements and quantify inter-articulator coordination. CT-based radiation dosimetry is integrated to identify candidate dose-sensitive functional subregions of the tongue and characterize relationships among regional radiation exposure, tissue remodeling, and speech motor performance.

Results: Guided by preliminary results demonstrating diminished precision of inter-articulator timing following surgery and/or radiotherapy, we hypothesize that treatment-induced structural and sensorimotor changes disrupt speech motor coordination. We further hypothesize that the extent of radiation-induced fibrosis, the presence of insensate reconstructive tissue, and greater radiation dose to functionally critical tongue subregions will predict the severity of speech motor impairment.

Translational Impact: Study outcomes will establish a mechanistic framework linking treatment-related changes in tongue structure and sensorimotor function to speech motor impairment and recovery. The findings will provide foundational evidence for developing objective biomarkers of treatment-related dysfunction, identifying candidate dose-sensitive tongue regions, and guiding future optimization of surgical, radiation, and rehabilitation strategies to improve long-term speech outcomes and quality of life.

Funding: This work was supported by research funds from the University of Oklahoma (OU) College of Allied Health and by a seed grant from the OU Health Stephenson Cancer Center (M. Masapollo, PI).

ABSTRACT TITLE

COMPUTED TOMOGRAPHY BASED FLAP SHAPE AND VOLUME ESTIMATION FOR DIEP FLAP BREAST RECONSTRUCTION

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ABSTRACT TITLE

COMPUTED TOMOGRAPHY BASED FLAP SHAPE AND VOLUME ESTIMATION FOR DIEP FLAP BREAST RECONSTRUCTION

Abstract

Introduction: The deep inferior epigastric perforator (DIEP) flap is the gold standard for autologous breast reconstruction [1], [2], transferring abdominal tissue to recreate the breast mound. Accurate lower-abdominal subcutaneous adipose tissue (SAT) estimation supports planning. Current assessment relies on subjective examination or manual flap delineation on computed tomography angiography (CTA), limiting reproducible tissue-volume quantification [3]. This study developed an automated framework for flap modelling and SAT volume estimation using routine CTA.

Methods: CTA images and intraoperative DIEP flap weights from two patients were included. Pelvic and lumbar segmentations [4] defined the umbilicus search region. Candidates were ranked by anterior-surface depression, midline proximity, compactness, and sagittal-profile agreement; a point 2 cm cranial to the umbilicus defined the upper flap reference. The patient-specific midplane used the umbilicus, pelvis centroid, and anterior pubic-symphysis point. Pelvic Principal Component Analysis (PCA) defined orientation; lumbar vertebrae guided pubic-symphysis localization. A lip-shaped outline (Figure 1), 13 cm high and 35–40 cm wide [5], within available width, was projected onto the abdominal surface. Lip-shaped, diamond, and elliptical profiles accommodated surgeon preference and anatomy. Anterior SAT within the 3D footprint was segmented [6], volume calculated from voxel dimensions, and mass estimated at 1.0 g/mL [7].

Results: The lip-shaped design was evaluated in two patients with untrimmed whole-flap weights of 1,973 and 1,788 g. CTA-derived SAT volumes were 1,765.9 and 2,263.3 mL; estimated masses were 1,765.9 and 2,263.3 g. Absolute percentage errors were 10.5% and 26.6%. Findings support feasibility but require validation against clinical ground truth using matched harvested-flap SAT, size, and weight measurements.

Translational Impact: This framework could enable DIEP flap volume estimation from CTA without specialized volumetric systems. Future work will evaluate larger cohorts and develop machine-learning methods for landmark detection and 3D flap-shape prediction, integrating surface projection and automatic SAT volume estimation into a one-step workflow.

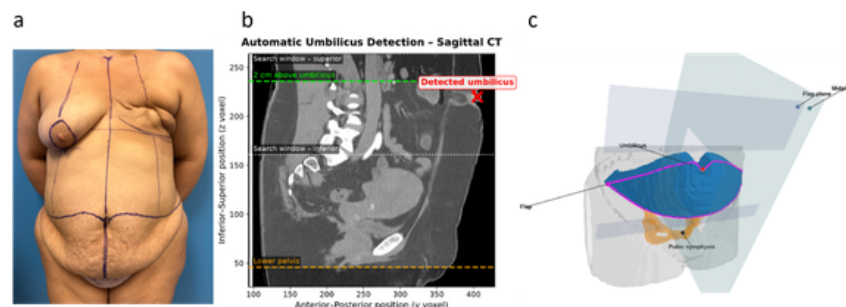


Figure 1. Flap planning workflow. (a) Patient 2D image showing preoperative surgical markings. (b) Sagittal CT view of automated umbilicus localization, showing the superior and inferior search limits, detected umbilicus, and the 2-cm cranial reference point. (c) Three-dimensional view of the constructed flap and its spatial relationship to the anatomical references and geometric planes. Pelvic PCA was used to establish orientation, with lumbar guidance used for localization of the anterior pubic symphysis reference. The umbilicus, pubic symphysis, flap, flap plane, and midplane are labelled.

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ABSTRACT TITLE

ENGINEERING ORALLY DELIVERED LIPID NANOPARTICLES FOR GASTROINTESTINAL DISEASE THERAPEUTICS

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ABSTRACT TITLE

ENGINEERING ORALLY DELIVERED LIPID NANOPARTICLES FOR GASTROINTESTINAL DISEASE THERAPEUTICS

Body of Abstract:

Introduction: Lipid nanoparticles (LNPs) have revolutionized nucleic acid delivery; however, all FDA-approved LNP therapeutics are administered by injection, limiting patient compliance and localized treatment of gastrointestinal (GI) diseases. Oral delivery offers a promising alternative but remains challenging due to gastric acidity, digestive enzymes, and the intestinal mucus barrier. Here, we developed a library of novel ionizable lipids to identify LNP formulations capable of enhancing oral mRNA delivery using physiologically relevant intestinal models.

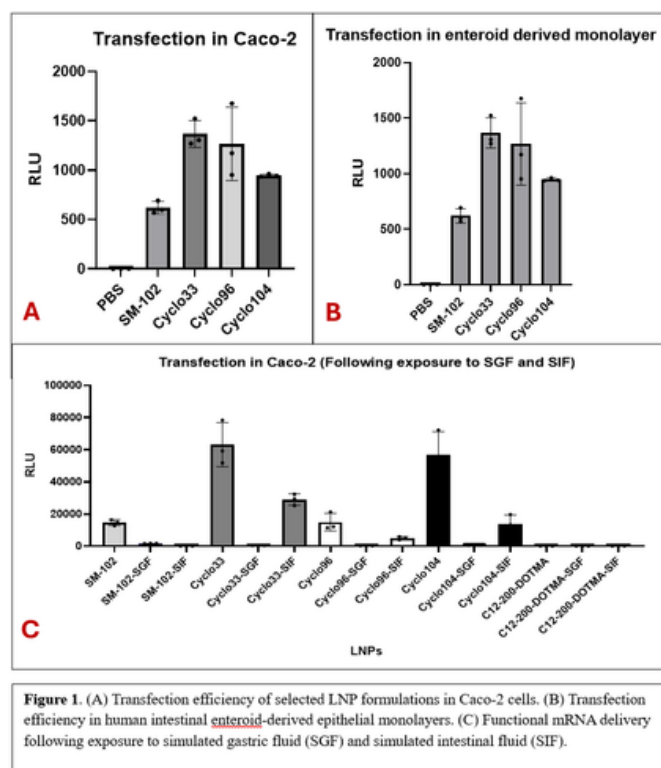
Methods: A library of 120 ionizable lipids encapsulating firefly luciferase (Fluc) mRNA was formulated by manual mixing. Initial screening was performed in HEK293 cells, and lead formulations were subsequently evaluated in Caco-2 intestinal epithelial cells using FDA-approved SM-102 and MC3 as benchmark controls. Lead candidates were further assessed following exposure to simulated gastric fluid (SGF) and simulated intestinal fluid (SIF). Human preterm intestinal organoid-derived epithelial monolayers were used to evaluate transfection efficiency and cell viability. The lead formulation was also evaluated following oral gavage in mice.

Results: Initial screening identified multiple ionizable lipids that outperformed FDA-approved benchmark formulations. In Caco-2 cells, Cyclo33, Cyclo96, and Cyclo104 demonstrated substantially greater luciferase expression than SM-102 and MC3. Although exposure to SGF markedly reduced transfection across all formulations, lead candidates retained measurable activity following SIF exposure. In human intestinal organoid-derived epithelial monolayers, all three lead formulations exceeded SM-102, with Cyclo33 exhibiting the highest transfection efficiency while maintaining cell viability. Oral delivery studies are ongoing to evaluate gastrointestinal transgene expression in vivo.

Translational Impact: These findings identify novel ionizable lipid formulations that improve mRNA delivery to GI tract and establish a promising platform for the development of orally administered nucleic acid therapeutics targeting gastrointestinal diseases.

Funding:

Start-up funds from the University of Oklahoma



ABSTRACT TITLE

PROTRACTED DEVELOPMENT OF PRECISE ARTICULATORY GESTURE COORDINATION IN CHILDREN AGED 7–12 YEARS: EVIDENCE FROM JAW AND LINGUAL KINEMATICS

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ABSTRACT TITLE

PROTRACTED DEVELOPMENT OF PRECISE ARTICULATORY GESTURE COORDINATION IN CHILDREN AGED 7–12 YEARS: EVIDENCE FROM JAW AND LINGUAL KINEMATICS

Abstract

Introduction: Speech motor development continues beyond early childhood, with school-age children exhibiting differences from adults in articulatory gesture organization. However, prior studies have relied primarily on indirect acoustic measures that cannot reveal the underlying articulatory movements. This study addresses this gap by using electromagnetic articulography (EMA) to directly quantify jaw–tongue coordination in children aged 7–12 years and determine whether articulatory timing precision has reached adult-like levels.

Methods: Forty healthy participants were recruited, including 20 children (7–12 years) and 20 young adults. EMA was used to track tongue–tip and jaw movements during production of vowel–consonant–vowel sequences containing varied vowels (/a/, /ε/) and alveolar stop consonants (/t/, /d/). Speech was elicited under different production rate (fast vs. normal) and syllable stress (first-syllable stressed vs. unstressed) conditions. Temporal coordination was quantified as the latency between the onset of vowel-related jaw-lowering movements and consonant-related tongue-tip-raising movements.

Results: In adults, consonant-related tongue–tip movement onset latencies, referenced to vowel-related jaw onsets, varied systematically as a function of production rate and syllable stress. In contrast, children exhibited greater kinematic variability and less systematic modulation of tongue–tip onset timing across speaking conditions, indicating reduced precision of inter-articulator coordination. Findings further suggest that children more consistently modulate articulatory timing in response to changes in rate than to changes in stress, suggesting that global control of utterance-level timing matures earlier than the capacity to selectively regulate the timing of individual articulatory gestures.

Translational Impact: By directly observing articulatory movements, this study provides physiological evidence that speech motor control continues to mature well beyond age 7. These findings advance mechanistic models of typical speech development, establish normative benchmarks for articulatory coordination, and provide a framework for investigating atypical speech development in pediatric populations, including children with repaired cleft palate and deaf children who receive cochlear implants.

Funding: This work was supported by research funds from the University of Oklahoma (OU) College of Allied Health and by a seed grant from the Presbyterian Health Foundation (M. Masapollo, PI).

ABSTRACT TITLE

BONE MARROW ADIPOCYTE LIPOLYSIS SUPPORTS THE OSTEOANABOLIC ACTIONS OF INTERMITTANT PARATHYROID HORMONE (PTH)

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ABSTRACT TITLE

BONE MARROW ADIPOCYTE LIPOLYSIS SUPPORTS THE OSTEOANABOLIC ACTIONS OF INTERMITTANT PARATHYROID HORMONE (PTH)

Parathyroid hormone (PTH, 1–34; teriparatide) is a therapeutic option for severely osteoporotic patients that stimulates bone formation. However, a considerable proportion of patients are unresponsive to PTH therapy. Therefore, continued research is needed to define the molecular mechanisms by which PTH exerts its osteoanabolic effects, and likewise, why some patients do not respond to treatment. Previous research showed that fatty acid metabolism is required for PTH-stimulated bone formation, and that bone marrow adipocytes (BMAdipo) are a potential source of fatty acids. The current project hypothesizes that PTH's osteoanabolic actions directly involve stimulation of lipolysis in BMAdipo to enhance fatty acid oxidation in osteoblasts. To test this, we utilized the novel Flippase-activated Adipoq-Cre (FAC) mouse model crossed with *Atgl* (*Pnpla2*)^{fl/fl} mice to selectively block lipolysis in BMAdipo. At 16 weeks of age, female FAC.*Atgl*^{fl/fl} mice, along with littermate controls (Con), were ovariectomized (OVX) and then allowed to lose bone while expanding marrow adiposity for 6 weeks. Next, PTH (100 µg/kg bodyweight) or vehicle control (Veh) were administered 5 days/week for 6 weeks. We discovered that Con OVX mice, and not FAC.*Atgl*^{fl/fl} OVX mice, responded to PTH treatment as evidenced by a significantly higher whole body bone mineral density and trabecular bone parameters. In addition, we quantified long chain fatty acids in circulating and bone marrow sera. Unexpectedly, we detected more oleic acid (C18:1) in FAC.*Atgl*^{fl/fl} OVX-Veh compared to Con OVX-Veh in both serum types. Within the bone marrow space, PTH lowered oleic acid in both Con OVX and FAC.*Atgl*^{fl/fl} OVX mice. These data support the tenet that PTH treatment requires fatty acids from BMAdipo to support bone formation. Moreover, these findings offer a novel approach to manage osteoporosis and suggest that responsiveness along with the efficacy of PTH treatment can be regulated by modulation of metabolic processes within the bone niche.

Funding: National Institute of Aging (R01AG069795)

ABSTRACT TITLE

TUMOR IMMUNE LANDSCAPE IN MOUSE SPLEEN FOLLOWING LOCALIZED ABLATIVE IMMUNOTHERAPY IN PANCREATIC CANCER MODEL

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ABSTRACT TITLE

TUMOR IMMUNE LANDSCAPE IN MOUSE SPLEEN FOLLOWING LOCALIZED ABLATIVE IMMUNOTHERAPY IN PANCREATIC CANCER MODEL

1 Abstract

Introduction: Pancreatic ductal adenocarcinoma (PDAC) is highly resistant to immunotherapy due to its immunosuppressive tumor microenvironment (TME), characterized by dense fibrosis and limited effector T-cell infiltration [1,2]. Photothermal therapy (PTT) combined with the immunoadjuvant glycated chitosan (GC) represents a promising strategy to enhance anti-tumor immunity [3]. Here, we evaluated the immunological effects of PTT+GC in a metastatic pancreatic cancer mouse model using single-cell RNA sequencing (scRNA-seq) of tumor and spleen tissues.

Methods: C57BL/6J mice bearing subcutaneous Panc02H7 tumors were treated with PTT (980 nm, 0.85 W/cm², 10 min) followed by intratumoral administration of 100 µL GC. Tumors and spleens were harvested 13 days post-treatment, dissociated into single-cell suspensions, and analyzed using the 10x Genomics 5' Gene Expression assay.

Results: scRNA-seq analyses identified diverse immune and stromal populations with distinct treatment-associated responses. Two B-cell populations exhibited antigen-presentation features, with one showing a more activated phenotype marked by Cd40 and Icosl. T cells displayed effector/cytotoxic and TCR-signaling features (Nkg7, Ccl5, Zap70) alongside Il17r/Tcf7-associated differentiation. Myeloid cells exhibited antigen-processing/phagolysosomal features (Cd68, Lgals3, Ctss, Tyrobp) and a C1q-expressing population (C1qa-c), while tumor-associated macrophages showed increased Apoe. Stress-associated genes (Hspa8, Dnaja1, Dnajb1, Hsph1, Ddit4, Fkbp4) were globally upregulated. Random Forest analysis of module-scored programs identified stress response as the strongest predictor of treatment, alongside myeloid/neutrophil, antigen-presentation, B-cell receptor, helper T-cell, cytotoxic, activation, and degranulation programs. GO enrichment supported myeloid/leukocyte activation, neutrophil responses, and immune effector functions.

Translational Impact: PTT+GC induces coordinated innate and adaptive immune activity alongside a prominent cellular stress response, characterized by myeloid/neutrophil activation, antigen-presentation programs, and T-cell effector activity. These findings provide mechanistic insight into the immune response associated with PTT+GC and support further investigation of this approach as an immunomodulatory strategy for PDAC, including its potential integration with immunotherapies.

2 Funding

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ABSTRACT TITLE

MACROPHAGES UPTAKE SYNTHETIC NANOPARTICLE CYTOKINES (SYNK) PRIMARILY BY CLATHRIN-MEDIATED ENDOCYTOSIS

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ABSTRACT TITLE

MACROPHAGES UPTAKE SYNTHETIC NANOPARTICLE CYTOKINES (SYNK) PRIMARILY BY CLATHRIN-MEDIATED ENDOCYTOSIS

Our laboratory has developed a novel P(AAm-co-MAA) nanogel (NG) that may be modified with covalently bound cytokines to generate Synthetic Nanoparticle Cytokines (SyNKs)^{1,2}. Both therapeutics alter macrophage phenotype in vitro^{1,2}. NGs are thought to influence cellular uptake and trafficking through their tunable physiochemical properties, while SyNKs are designed to function through binding to cytokine receptors^{1,2}. To optimize these therapeutics, it is critical to understand the mechanisms of these interactions and how those processes may compete with one another in relation to dose and activation state.

RAW macrophages (RAWs) were treated with RhoB-NGs or RhoB-IL-10- or -IFN- γ -SyNKs. Fluorescence microscopy and flow cytometry confirmed cellular uptake by quantifying mean fluorescence intensity (MFI). MFI measurement of RAWs treated with increasing doses of RhoB-NGs determined uptake kinetics. Live-cell imaging of AF488-NG-dosed RAWs with Lysotracker Red was used to observe intracellular trafficking. NG uptake mechanisms were determined by treating RAWs with pharmaceuticals known to inhibit select endocytosis mechanisms (Clathrin-mediated endocytosis (CME), Caveolin-mediated endocytosis (CaME), and micropinocytosis), then dosing with RhoB-NGs. The MFI was quantified.

NGs were uptaken via endo-lysosomal trafficking. SyNKs promoted enhanced NG uptake; IL-10 promoted a 25% increase and IFN- γ promoted a 3-fold increase in MFI. RAWs internalized RhoB-NGs in a dose-dependent manner up to 100 μ g/mL, indicating saturable, receptor-mediated uptake. CME was determined to contribute most to NG uptake, followed by CaME. Micropinocytosis inhibitors did not reduce uptake. These findings suggest NGs and SyNKs most readily interact with CME cargo receptors.

Cytokine therapies have been increasingly explored as a tool for treating diseases driven by immune dysfunction. Significant barriers to clinical translation include harmful off-target effects and short half-life in systemic circulation. The NG and SyNK platforms provide a promising solution for these challenges, offering no cytotoxicity, prolonged circulation, and highly tunable material properties that may enhance tissue targeting and therapeutic efficacy.

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POSTER PRESENTATIONS

SESSION 1 | 45A

ABSTRACT TITLE

MULTIMODAL OCULAR AI MODELS FOR PREDICTING DISABILITY IN MULTIPLE SCLEROSIS

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ABSTRACT TITLE

MULTIMODAL OCULAR AI MODELS FOR PREDICTING DISABILITY IN MULTIPLE SCLEROSIS

Introduction: Multiple sclerosis (MS) exerts focal and diffuse damage to the central nervous system (CNS) which accrues before it becomes clinically visible. The eye offers non-invasive access to neuronal, vascular, and functional features of the CNS. Although optical coherence tomography (OCT) measures of inner retinal damage are standard practice in MS, whether ocular imaging can predict broader neurological disability remains unclear. Another open question is how to best combine ocular scans with complementary clinical measures. We explore how new approaches using artificial intelligence (AI) models, pre-trained on generic datasets to learn general patterns, can be adapted to MS to develop markers of neurodegeneration and integrate multiple data types. Methods: We adapt the RETFound OCT model trained on >800,000 unique scans to our local MS cohort (n = 50 train, 20 test) by using “transfer learning”, where we add new layers to the original frozen neural network and optimize them for a new task. We integrated RETFound’s OCT image features with timed 25-foot walk scores, age, and cognitive performance to predict a person’s Expanded Disability Status Scale (EDSS) score, the most broadly used disability measure in MS. Results: Transfer learning on RETFound predicted EDSS scores (0–10 range, 0.5 increments) with a mean absolute error of 1.2 points. Combining these OCT features with the other clinical measurements reduced the error to 0.875 points. Translational Impact: Our findings suggest that AI models of OCT data may capture relevant information beyond the traditional thickness measurements currently used. When integrating these models with other clinical measures, they may provide an objective approach to estimating MS-associated disability.

Funding: NIH Grants T32EY023202, P20GM156711, P30AG050911

POSTER PRESENTATIONS

SESSION 1 | 47A

ABSTRACT TITLE

DISRUPTING THE GLIOBLASTOMA MICROENVIRONMENT: NON-INVASIVE MAPPING WITH THERAPEUTIC MICROGLIAL ACTIVATION VIA ULTRASOUND

Vishaak Rajaram¹, Rakesh Rudraboina^{2,3}, Madison Milan^{2,3}, Aleksandra Kosmider^{2,3}, Sharon Negri^{2,3}, Jennifer Ihuoma^{2,3}, Stefano Tarantini^{2,3}

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ABSTRACT TITLE

DISRUPTING THE GLIOBLASTOMA MICROENVIRONMENT: NON-INVASIVE MAPPING WITH THERAPEUTIC MICROGLIAL ACTIVATION VIA ULTRASOUND

Introduction

Glioblastoma (GBM), the most aggressive glioma, is the most common brain tumor amongst male adults with previous exposure to ionized radiation, as well as individuals with rare inherited genetic syndromes. GBM migrates through the whole brain, hijacking immune cells and using them to suppress immune responses for cancer expansion. Current treatment plans include invasive biopsies, surgical resectioning, and radiochemotherapy, providing a 15-month median survival rate regardless of the treatment, showing an urgent clinical need for alternate diagnostic and therapeutic strategies. This study utilizes functional ultrasound (fUS) imaging to non-invasively map tumor progression of mice with GBM, treated with focused ultrasound (FUS) to stimulate an immune response to the tumor.

Methods

Mouse models of GBM were monitored using a localized fUS imaging system and validated against an In Vivo Imaging System (IVIS) to track tumor growth and vascular remodeling. Mice underwent chronic FUS therapy at high and low intensities. Tissues were then analyzed via immunohistochemistry (IHC) using Iba-1 and CD-68 biomarkers to measure microglial activation and morphological quantifications, alongside CD-31 and endomucin to evaluate vascular changes.

Results

The fUS imaging correlated with the IVIS tumor progression, by mapping tissue disorganization and hemodynamic changes, which were confirmed by vascular IHC staining. The ramification of microglia decreased while somal area increased in mice that underwent FUS treatment at both high and low settings compared to the controls, indicating robust activation.

Translational Impact

This work shows potential for fUS imaging to provide a high-resolution, non-invasive alternative to traditional invasive biopsies for monitoring GBM progression. Furthermore, demonstrating that localized FUS therapy successfully stimulates host microglial activation establishes a powerful foundation for utilizing ultrasound as a non-invasive adjuvant immunotherapy to disrupt the immunosuppressive GBM microenvironment and improve patient outcomes.

This research was supported by the American Association for Cancer Research (AACR), Cancer Research Education for Success(CARES) Grant (AACR-CARES5464-OKLA).

POSTER PRESENTATIONS

SESSION 1 | 50A

ABSTRACT TITLE

PLASMA SOLUBLE HLA CLASS I IMMUNOPEPTIDOMICS AS A WINDOW INTO SARS-COV-2 INFECTION

Silvano Ross¹, Saghar Kaabinejadian², Hooman Yari², William Hildebrand³

The University of Oklahoma¹

ABSTRACT TITLE

PLASMA SOLUBLE HLA CLASS I IMMUNOPEPTIDOMICS AS A WINDOW INTO SARS-COV-2 INFECTION

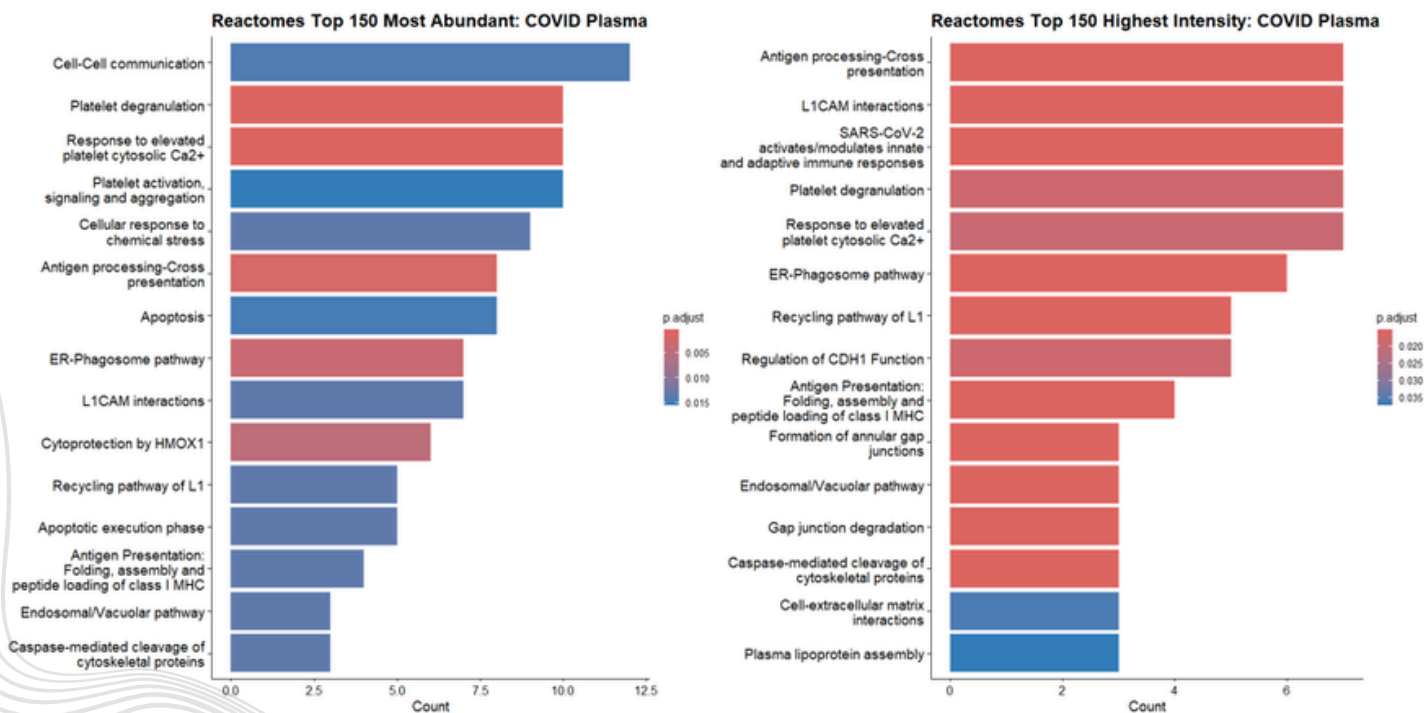
Introduction: Plasma is minimally invasive to collect and can be archived long-term. Our laboratory has shown that cell-free HLA (soluble HLA, sHLA) can be immunoaffinity-purified from plasma. Because HLA class I presents peptides from a broad intracellular proteome, the plasma sHLA ligandome may report the systemic biological state. We hypothesized that SARS-CoV-2-derived peptides, or host peptides consistent with infection, could be detected among HLA class I peptides from COVID-positive plasma, supporting sHLA immunopectidomics as a potential diagnostic.

Methods: Three plasma samples from patients with acute COVID-19 were obtained from the Oklahoma Clinical and Translational Sciences Institute (OCTSI). After centrifugation, 5 ml was passed over a W6/32 immunoaffinity column and class I HLA were eluted in 10% acetic acid to release bound peptides. Peptides were HPLC-fractionated and analyzed by ZenoTOF mass spectrometry. Spectra were processed in PEAKS Studio against SwissProt Human + SwissProt SARS-CoV-2 (reviewed); SwissProt Human + TrEMBL SARS-CoV-2 (reviewed + unreviewed); and TrEMBL SARS-CoV-2 alone. Candidate viral peptides were assessed by database search and DeepNovo, with pathway enrichment (KEGG, GO, Reactome) in clusterProfiler.

Results: No SARS-CoV-2 peptides were identified with the reviewed SwissProt databases. Searches including unreviewed TrEMBL SARS-CoV-2 entries yielded candidate virus-derived peptide assignments by database search and DeepNovo, and pathway analysis recovered viral infection-related signatures. These preliminary assignments require orthogonal validation and COVID-negative plasma controls.

Translational Impact: These proof-of-concept data show that plasma sHLA class I peptides are recoverable from small clinical volumes by high-resolution mass spectrometry. If candidate viral ligands and infection-associated signatures are confirmed, plasma sHLA immunopectidomics could complement existing diagnostics as a minimally invasive readout of intracellular antigen presentation during infection.

Funding: Supported by NIH T32GM154645



POSTER PRESENTATIONS

SESSION 1 | 53A

ABSTRACT TITLE

**AN ENHANCED VALVEFIT PIPELINE FOR PATIENT-SPECIFIC COMPLETE ATRIOVENTRICULAR
CANAL VALVE RECONSTRUCTION, MESH OPTIMIZATION, AND COMPUTATIONAL MODELING**

Hamid Shahsavari¹, Harold M. Burkhart¹, Arshid Mir¹, Jonathan D. Ball¹, Chung-Hao Lee²

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ABSTRACT TITLE

AN ENHANCED VALVEFIT PIPELINE FOR PATIENT-SPECIFIC COMPLETE ATRIOVENTRICULAR CANAL VALVE RECONSTRUCTION, MESH OPTIMIZATION, AND COMPUTATIONAL MODELING

Introduction: Complete atrioventricular canal (CAVC) is a congenital heart defect characterized by a single common atrioventricular valve (AVV). AVV regurgitation is frequent, yet the relationship between CAVC valve geometry, mechanics, and regurgitation remains poorly understood. This study presents an in vivo mechanistic framework for translating three-dimensional echocardiographic (echo) imaging data into computational models requiring accurate reconstruction and high-quality discretization. We enhanced the recently published ValveFit framework to establish a patient-specific image-to-analysis pipeline for CAVC valve modeling.

Methods: Patient-specific CAVC valve geometries were obtained from segmented three-dimensional echo point clouds. Unlike the standard-template geometry used in the original ValveFit framework, a patient-specific template derived from each subject's segmented valve anatomy initialized reconstruction to better preserve individual leaflet morphology. A differentiable B-spline representation and control-point optimization fitted the segmented data and generated analysis-ready surfaces. A mesh-optimization algorithm improved element quality by optimizing skewness, aspect ratio, element angles, and Jacobian quality while preserving valve topology.

Results: For one representative CAVC case, patient-specific initialization improved reconstruction accuracy, reducing mean Nearest Neighbor Distance (NND) by 20%, from 0.60 mm using standard-template initialization to 0.48 mm. Mesh optimization reduced maximum aspect ratio from 23.99 to 6.28 and maximum skewness from 0.89 to 0.65, while increasing minimum Jacobian from 0.16 to 0.53. Minimum and maximum element angles improved from 11.74° and 170.29° to 20.57° and 148.06°, respectively, while preserving topology. The optimized geometry was exported as a continuous and discretized surface for computational analysis.

Translational Impact: This image-to-analysis pipeline converts echo data into patient-specific computational models of congenital AVV disease. Improved reconstruction and mesh quality may facilitate reliable biomechanical simulations and virtual evaluation of repair strategies. Patient-specific geometry may also enable more accurate 3D-printed valve models for anatomical visualization and preoperative planning. Together, these capabilities may support patient-specific surgical planning and improved CAVC treatment strategies.

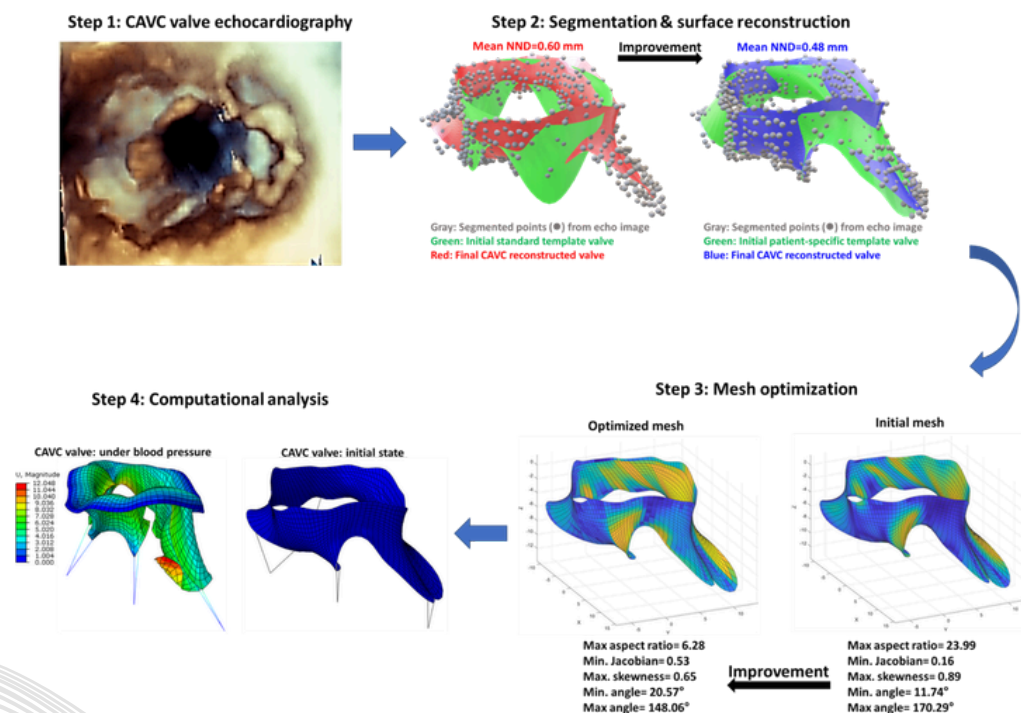


Figure 1. Overview of the presented study including 4 steps.

ABSTRACT TITLE

QUANTIFYING PHYSICAL RESILIENCE AS A DYNAMIC BIOMARKER OF BIOLOGICAL AGING IN MICE

Santhya Shanmugarama^{1,2}, Evelyn Brunner^{1,2}, Mark Nagykaldi^{1,2}, Roland Patai^{1,2}, Dorina Nagy^{1,2,4}, Shoba Ekambaram^{1,2}, Kiana Vali Kordestan^{1,2}, Rebeka Kristof^{1,2,3}, Parikshat Sirpal^{1,2}, Basit Khan^{1,2}, Stefano Tarantini^{1,2,3,5}, Anna Csiszar^{1,2,3,4}

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ABSTRACT TITLE

QUANTIFYING PHYSICAL RESILIENCE AS A DYNAMIC BIOMARKER OF BIOLOGICAL AGING IN MICE

Introduction: Aging progressively reduces the body's ability to withstand and recover from physiological stress. However, conventional measures of aging and frailty often identify functional decline relatively late. We investigated whether longitudinal measures of physical resilience could provide dynamic indicators of biological aging in mice.

Methods: Aging mice were followed longitudinally across control (n=53), fusogenic liposome (FL; n=77), apolipoprotein E-functionalized FL (ApoE-FL; n=89), and polyethylene glycol (PEG; n=50) cohorts. Physical resilience was assessed using body-weight responses, recovery following isoflurane anesthesia, grip strength, body condition, kyphosis, fur and skin condition. Repeated measurements were used to characterize changes in functional status and recovery capacity across the lifespan alongside survival.

Results: Longitudinal assessment revealed progressive age-associated changes across multiple dimensions of physical resilience. Grip performance declined with advancing age, while recovery following anesthesia became prolonged and increasingly variable. Clinical assessments showed progressive deterioration in posture, reflected by increasing kyphosis, and worsening fur and skin condition, accompanied by changes in body condition. Body-weight responses following physiological challenge also became more variable with aging. Collectively, these longitudinal patterns demonstrate multidimensional deterioration in functional capacity and physiological recovery during aging.

Translational Impact: Dynamic assessment of how an organism responds to and recovers from physiological stress may complement conventional measures of frailty by capturing changes in resilience across the lifespan. This multidimensional preclinical framework provides a foundation for validating physical resilience as a biomarker of biological aging and for evaluating interventions designed to preserve healthspan. Future validation against lifespan and molecular aging markers may support development of analogous resilience-based assessments for earlier identification of functional decline in humans.

Funding: RF1AG072295, R01AG055395, R01AG068295, R01AG070915, K01AG073614, R01NS100782, R01CA255840, AHA916225, and Presbyterian Health Foundation.

POSTER PRESENTATIONS

SESSION 1 | 55A

ABSTRACT TITLE

ROLE OF BACH1 IN PANCREATIC CANCER PATHOGENESIS AND THERAPY EFFICACY

Surendra Kumar Shukla

Department of Oncology Science, OUHSC, OKC, OK, USA

ABSTRACT TITLE

ROLE OF BACH1 IN PANCREATIC CANCER PATHOGENESIS AND THERAPY EFFICACY

The BACH1 (BTB domain and CNC homolog 1) transcription factor plays a very important role in the regulation of different aspects of cellular physiology. It is overexpressed in various cancers and regulates different aspects of cancer pathogenesis. However, its role in pancreatic cancer progression and therapeutic response is not well understood. In the present study, we have explored the effect of BACH1 modulation on pancreatic cancer growth and therapeutic response by utilizing different in vitro as well as in vivo model systems. We observed that BACH1 expression is significantly upregulated in pancreatic cancer patients, which significantly correlates with poor survival of PDAC patients. BACH1 knockdown leads to reduced cellular growth in 2D as well as 3D model systems. Further, we have demonstrated that BACH1 knockdown leads to the upregulation of the unfolded protein response (UPR) pathway, mainly through the downregulation of the PDIA6 protein. Similar effects were observed when pancreatic cancer cells were treated with Hemin, an FDA-approved pharmacological agent targeting BACH1. Further, we have demonstrated that BACH1 transcriptionally regulates PDIA6 expression, which plays a significant role in BACH1-mediated cellular growth. We also observed that BACH1 knockdown leads to improved sensitivity towards FOLFIRINOX, the current standard of care for pancreatic cancer patients. Similarly, hemin treatment improved the efficacy of FOLFIRINOX in cell culture as well as an orthotopic implantation mouse model of pancreatic cancer. Overall, we have demonstrated the novel role of BACH1 in UPR regulation and therapy response. Targeting BACH1 with hemin in combination with FOLFIRINOX can improve the therapeutic efficacy of current chemotherapeutics.

Funding:

Stephenson Cancer Centre–Department of Oncology Science
ACS-IRG, PHF, NIH-NCI

POSTER PRESENTATIONS

SESSION 1 | 56A

ABSTRACT TITLE

**DEVELOPMENT OF FIRST -IN CLASS- IN VIVO SAFE AND EFFECTIVE NOVEL EEF2-KINASE
INHIBITORS FOR TRIPLE-NEGATIVE BREAST CANCER**

Merve Sikik¹, Sayra Dilmac¹, Bulent Ozpolat¹

¹*Stephenson School of Biomedical Engineering, University of Oklahoma, Norman, OK, USA*

ABSTRACT TITLE

DEVELOPMENT OF FIRST -IN CLASS- IN VIVO SAFE AND EFFECTIVE NOVEL EEF2-KINASE INHIBITORS FOR TRIPLE-NEGATIVE BREAST CANCER

Introduction: Triple-negative breast cancer (TNBC) accounts for 15–20% of breast cancers, lacks ER, PR, HER2 expression, and has the poorest survival of all subtypes. Current therapies lack durable benefit, and new targets are urgently needed. We demonstrated that eukaryotic elongation factor 2 kinase (eEF2K), an atypical serine/threonine kinase, is a poor prognostic factor driving TNBC proliferation, invasion, tumor growth and drug resistance, and validated it as a novel molecular target in clinically relevant models (1–5). However, no eEF2K inhibitor is FDA-approved due to limited potency, selectivity, and safety of published compounds. We developed small molecule eEF2K inhibitors and investigated three novel leads.

Methods: eEF2K expression and patient survival were analyzed using TNMplot and KM Plotter (NCI-TCGA). In silico docking, molecular dynamics, and ADME analyses targeted the eEF2K ATP-binding site (PDB: 8GM5). Cell proliferation was assessed by CCK8 and clonogenic assays. Target inhibition was confirmed at 4, 24 and 48 hours by Western blotting to evaluate the direct downstream target p-eEF2 (Thr56), GAPDH as loading control. Synergy was evaluated by isobologram analysis. Apoptosis was evaluated by Annexin V/PI staining and flow cytometry. Tumor growth was monitored in MDA-MB-231 xenografts.

Results: eEF2K was overexpressed in TNBC tumors versus normal breast tissue and correlated with poor survival. Docking showed higher ATP-pocket affinity than NH125 or A-484954. In TNBC cells, the inhibitors dose-dependently suppressed p-eEF2 at nanomolar concentrations, inhibited proliferation and colony formation, and induced apoptosis. Binding constants (K_d) are being determined by surface plasmon resonance and isothermal titration calorimetry to confirm docking predictions. In vivo, two lead inhibitors significantly inhibited MDA-MB-231 tumor growth at 20 mg/kg with no observed toxicity.

Translational Impact: These inhibitors block eEF2K signaling and suppress TNBC tumor growth without toxicity. A water-soluble lead compound is suitable for systemic administration, establishing eEF2K inhibition as a viable therapeutic strategy for TNBC.

Funding: NIH-NCI (R01) – 1R01CA244344-01A1.

Presenting author: Merve Sikik. **Corresponding author:** Bulent Ozpolat.

1. Hamurcu Z, Ashour A, Kahraman N, Ozpolat B. *Oncotarget*. 2016;7:16619–16635.
2. Bayraktar R, Ivan C, Bayraktar E, et al., Ozpolat B. *Clin Cancer Res*. 2018;24:4225–4241.
3. Karakas D, Ozpolat B. *J Mol Med*. 2020;98:775–787.
4. Karakas D, Ashour A, Mokhlis HA, et al., Ozpolat B. *Cell Death Dis*. 2025. doi:10.1038/s41419-025-07803-w
5. Biltekin E, Gul OA, Kahraman N, et al., Ozpolat B. *Int J Mol Sci*. 2025;26:6792.

POSTER PRESENTATIONS

SESSION 1 | 57A

ABSTRACT TITLE

LIPOPROTEIN(A) INDUCED VASCULAR DYSFUNCTION IN DIABETIC RETINOPATHY

Md Sohel¹, Xue Cai¹, Amina Jamil¹, Tim F. Dorweiler⁴, Nicole El-Darzi², Rui Cheng⁵, Jian-Xing Ma⁵, Irina Pikuleva², George S. Abela³, Maria Grant⁶, Julia V. Busik¹.

¹*Stephenson School of Biomedical Engineering, University of Oklahoma, Norman, OK, USA*

ABSTRACT TITLE

LIPOPROTEIN(A) INDUCED VASCULAR DYSFUNCTION IN DIABETIC RETINOPATHY

Introduction: Triple-negative breast cancer (TNBC) accounts for 15–20% of breast cancers, lacks ER, PR, HER2 expression, and has the poorest survival of all subtypes. Current therapies lack durable benefit, and new targets are urgently needed. We demonstrated that eukaryotic elongation factor 2 kinase (eEF2K), an atypical serine/threonine kinase, is a poor prognostic factor driving TNBC proliferation, invasion, tumor growth and drug resistance, and validated it as a novel molecular target in clinically relevant models (1–5). However, no eEF2K inhibitor is FDA-approved due to limited potency, selectivity, and safety of published compounds. We developed small molecule eEF2K inhibitors and investigated three novel leads.

Methods: eEF2K expression and patient survival were analyzed using TNMplot and KM Plotter (NCI-TCGA). In silico docking, molecular dynamics, and ADME analyses targeted the eEF2K ATP-binding site (PDB: 8GM5). Cell proliferation was assessed by CCK8 and clonogenic assays. Target inhibition was confirmed at 4, 24 and 48 hours by Western blotting to evaluate the direct downstream target p-eEF2 (Thr56), GAPDH as loading control. Synergy was evaluated by isobologram analysis. Apoptosis was evaluated by Annexin V/PI staining and flow cytometry. Tumor growth was monitored in MDA-MB-231 xenografts.

Results: eEF2K was overexpressed in TNBC tumors versus normal breast tissue and correlated with poor survival. Docking showed higher ATP-pocket affinity than NH125 or A-484954. In TNBC cells, the inhibitors dose-dependently suppressed p-eEF2 at nanomolar concentrations, inhibited proliferation and colony formation, and induced apoptosis. Binding constants (K_d) are being determined by surface plasmon resonance and isothermal titration calorimetry to confirm docking predictions. In vivo, two lead inhibitors significantly inhibited MDA-MB-231 tumor growth at 20 mg/kg with no observed toxicity.

Translational Impact: These inhibitors block eEF2K signaling and suppress TNBC tumor growth without toxicity. A water-soluble lead compound is suitable for systemic administration, establishing eEF2K inhibition as a viable therapeutic strategy for TNBC.

Funding: NIH-NCI (R01) – 1R01CA244344-01A1.

Presenting author: Merve Sikik. **Corresponding author:** Bulent Ozpolat.

1. Hamurcu Z, Ashour A, Kahraman N, Ozpolat B. *Oncotarget*. 2016;7:16619–16635.
2. Bayraktar R, Ivan C, Bayraktar E, et al., Ozpolat B. *Clin Cancer Res*. 2018;24:4225–4241.
3. Karakas D, Ozpolat B. *J Mol Med*. 2020;98:775–787.
4. Karakas D, Ashour A, Mokhlis HA, et al., Ozpolat B. *Cell Death Dis*. 2025. doi:10.1038/s41419-025-07803-w
5. Biltekin E, Gul OA, Kahraman N, et al., Ozpolat B. *Int J Mol Sci*. 2025;26:6792.

POSTER PRESENTATIONS

SESSION 1 | 58A

ABSTRACT TITLE

TARGETING STING MITIGATES OBESITY-EXACERBATED NEUROINFLAMMATION

Melissa Testut^{1,2}, Kaylea Massey², Gaurav Kumar², Gabriel Pardo², Robert C. Axtell^{1,2}

¹University of Oklahoma Health Sciences Center, ²Oklahoma Medical Research Facility

ABSTRACT TITLE

TARGETING STING MITIGATES OBESITY-EXACERBATED NEUROINFLAMMATION

Introduction

Obesity is associated with worse outcomes and reduced treatment response in women with multiple sclerosis (MS), but the biological mechanisms contributing to these effects remain poorly understood. We hypothesized that obesity-induced mitochondrial stress activates STING, an immune pathway that detects cellular damage, to elevate type I interferon signaling and worsen neuroinflammatory disease in women with MS.

Methods

Serum proteomic signatures were analyzed to identify obesity-associated inflammatory pathways in obese and nonobese women with MS. In parallel, female mice were fed control or high-fat diets and assessed for mitochondrial dysfunction and interferon production and response. STING was then pharmacologically inhibited to determine the downstream effects on interferon signaling and neuroinflammatory disease severity.

Results

Women with MS and obesity exhibited altered circulating inflammatory proteins, including an enrichment of proteins associated with inflammation and type I interferon signaling. In mice, spleens exhibited elevated interferon-stimulated gene expression, and adipose immune cells displayed increased mitochondrial dysfunction. Inhibition of STING reduced interferon-responsive gene expression in obese female mice and reduced the severity of neuroinflammatory disease.

Translational Impact

These findings identify mitochondrial stress and STING signaling as potentially modifiable pathways linking obesity with neuroinflammatory disease severity in women. Further studies could establish STING-associated biomarkers and support development of targeted therapies for this high-risk population.

Funding

OCASCR, NIH NIDA, NIH NIGMS T32GM154645

POSTER PRESENTATIONS

SESSION 1 | 60A

ABSTRACT TITLE

FROM EXTREME GENETIC HETEROGENEITY TO SHARED TARGETS: CONVERGENT WNT AND HEDGEHOG DYSREGULATION IN JOUBERT SYNDROME

Julie Craft Van De Weghe^{1,2}, Justyna Szymczak¹, Rose Johnson¹, Felix Asare¹, Zuri Martinez¹, Arianna Gomez², Angela Christman², Ryan Kittle³, Rich Johnson⁴, Jea Park⁴, Michael MacCoss⁴, Dan Doherty²;

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ABSTRACT TITLE

FROM EXTREME GENETIC HETEROGENEITY TO SHARED TARGETS: CONVERGENT WNT AND HEDGEHOG DYSREGULATION IN JOUBERT SYNDROME

Ciliopathies rob vision and intellect, deteriorate organs, and truncate life spans. They range from embryonic lethality to syndromic, multi-organ conditions. Despite affecting ~1 in 1000 individuals, no curative therapies exist, underscoring the need to define the pathomechanisms. Ciliopathies arise from dysfunctional cilia, organelles that orchestrate signaling. Strict regulation of their proteome is essential, and its disruption likely drives disease, as regulators are implicated in ciliopathies.

Joubert syndrome (JS), a neurodevelopmental ciliopathy, exemplifies the complexity of genetically heterogeneous conditions. Defects in >40 cilia-related genes cause the pathognomonic JS 'molar tooth sign' on brain MRI and often progressive degeneration of the retina, kidney, and liver. Prior gene-centric studies revealed diverse defects but no shared, disease-driving pathways. Critically, no analysis of the ciliary proteome in JS has been conducted, hindering our understanding of convergent disease mechanisms.

Here, we bridge this gap by performing the first JS proteomics screen. We genome-engineered a library of loss-of-function cell lines spanning 11 JS genes and developed a robust protocol for isolating primary cilia. Differential data-independent acquisition mass spectrometry of isolated cilia identified proteins with altered abundance in JS mutant cilia versus controls. Bioinformatics analysis of the datasets (adjusted p-value ≤ 0.05) predicted significant dampening of Ephrin and other signaling pathways. Across the datasets, the top shared features were the predicted bidirectional dysregulation of Wnt and Hedgehog signaling. We generated a second cell library to alter the abundance of our top 16 shared differentially abundant proteins and assessed the impact on these pathways. We confirmed that Wnt and Hedgehog signaling are functionally impaired, indicating the JS pathomechanism involves combinatorial signaling dysfunction. We identified several previously unrecognized regulators of these signaling pathways. Our unparalleled cross-genetic approach defines a shared JS proteomic pathogenic landscape, validates a common dysregulated Wnt and Hedgehog signaling axis, and offers new pathway targets for future therapies.

Funding: NIH NICHD R00HD100554 to JCV

POSTER PRESENTATIONS

SESSION 1 | 62A

ABSTRACT TITLE

DEVELOPMENT OF A NASAL SPRAY FOR RESPIRATORY VIRUS PROPHYLAXIS – FROM BENCHTOP TO MARKET

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²Stephenson Cancer Center, The University of Oklahoma Health Campus, Oklahoma City, OK 73104, USA

ABSTRACT TITLE

DEVELOPMENT OF A NASAL SPRAY FOR RESPIRATORY VIRUS PROPHYLAXIS – FROM BENCHTOP TO MARKET

Abstract

Introduction: Respiratory viruses, such as influenza and coronaviruses, remain a major global health burden. Vaccines are effective but may lag behind emerging or rapidly evolving viruses, highlighting the need for rapidly deployable, broad-spectrum prophylaxis. We are developing N-dihydrogalactochitosan (GC), an immunostimulatory biopolymer, as a needle-free prophylactic nasal spray through an integrated bench-to-market translational pathway.

Methods: GC and related chitosan derivatives were evaluated for candidate optimization and prophylactic efficacy in mouse respiratory virus infection models. A differentiated human airway epithelial air–liquid interface model was established to evaluate GC uptake, tolerability, and immune responses. Nasal spray prototypes are under development in collaboration with Aptar Pharma. ClinicalTrials.gov analysis, feasibility interviews, and preliminary TAM/SAM/SOM analysis were conducted to evaluate clinical positioning and commercial potential.

Results: Intranasal GC demonstrated substantial prophylactic efficacy in a lethal SARS-CoV-2 mouse model, reducing morbidity and mortality by approximately 75%. In a validated human airway epithelial model, GC was internalized by epithelial cells and modulated immune signaling without detectable cytotoxicity at concentrations up to 0.1%. Comparative evaluation showed that GC, fucosylated chitosan, and mannosylated chitosan induced comparable pro-inflammatory activation and reduced influenza-associated lung pathology, broadening the prophylactic candidate pool. Human nasal spray devices from Aptar Pharma were identified and are being evaluated for prototype development. ClinicalTrials.gov analysis identified 188 trials of intranasal interventions against respiratory infections, including 17 evaluating non-vaccine immunomodulatory approaches, defining the competitive space for product positioning. Feasibility interviews with 20 stakeholders showed positive interest, while preliminary market analysis supported commercialization potential.

Translational Impact: This work establishes an integrated bench-to-market pathway combining animal efficacy, human-relevant validation, candidate optimization, product development, and commercial assessment. By bridging preclinical efficacy with human airway testing while addressing delivery, market, and regulatory considerations, this study provides a translational foundation for developing a broad-spectrum intranasal prophylactic against respiratory viral infections.

Funding

OKBioStart (08-79-05678), Oklahoma Center for the Advancement of Science and Technology (HF20-019 and HR23-069)

POSTER PRESENTATIONS

SESSION 1 | 63A

ABSTRACT TITLE

THE 22-POINT CHECK LIST FOR BRIDGING THE GAP BETWEEN ACADEMIA AND INDUSTRY IN PRECLINICAL CANCER DRUG DEVELOPMENT

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ABSTRACT TITLE

THE 22-POINT CHECK LIST FOR BRIDGING THE GAP BETWEEN ACADEMIA AND INDUSTRY IN PRECLINICAL CANCER DRUG DEVELOPMENT

Introduction: The Center for Cancer Prevention and Drug Development (CCPDD), directed by Dr. Chinthalapally V. Rao, investigates carcinogenesis, identifies prevention and intervention targets, and evaluates candidate agents for toxicity and efficacy. The CCPDD contributes to NCI program projects focused on organ specific cancer models and, as an NCI contract research organization, have tested seven of the eleven compounds advancing toward clinical trials through the NCI PREVENT program. Thus, with this unique combination of academic and industry aligned expertise the CCPDD is among the most productive preclinical rodent based cancer research programs regionally and nationally. Cancer remains the most frequently cited disease relevance in basic and medical biology research. Yet the path from academic discovery to clinical application is often poorly understood, and significant gaps persist between academia and industry in both mindset and knowledge of the drug development process.

Methods: This article outlines the essential components of contemporary preclinical cancer drug development to better equip academic researchers and strengthen the competitiveness of cancer-focused proposals.

Results: Educational material: The 22-point checklist for academic researchers preparing for a proposal with cancer as disease relevance

Translational Impact: Our goal is to help bridge the translational divide, streamline the progression of academic innovations toward industry, and ultimately improve the success rate of candidate therapeutics by reducing attrition in the preclinical-to-clinical transition.

Category: Preclinical, Biological

Funding: Not applicable

SESSION 2

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SESSION 2

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Omitted abstracts were not included in the printed program at the authors' request.

ABSTRACT TITLE

MULTI-MODAL ANALYSES OF B CELLS IN FOLLICLES AND GERMINAL CENTERS DURING THYMIC INVOLUTION

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ABSTRACT TITLE

MULTI-MODAL ANALYSES OF B CELLS IN FOLLICLES AND GERMINAL CENTERS DURING THYMIC INVOLUTION

Introduction: Thymic involution, which peaks during puberty, is the atrophy of thymus driven by reduced T cell production and adipose replacement. Involution is the first major step of immunosenescence – age-related immune function decline. Beyond diminished T cell output, involution also affects thymic B cells. Intriguingly, pediatric thymic B and T cell cancers peak during this same window, yet cellular mechanism of involution and its relationship to thymic cancers are poorly understood. Here, we aim to define changes in thymic B cells during involution through their protein and gene expression profiles.

Methods: We collected 35 thymic specimens (infant n=9, toddler n=5, pre-pubertal n=9, peri-pubertal n=12). We performed H&E and IHC (CD19, CD21, BCL6) stains to identify B-cell follicles. We profiled 21 proteins in 52 regions of interest (ROI) from 13 thymi by Imaging Mass Cytometry (IMC) and ~5000 RNAs in 32 ROI across age groups by Xenium™ Spatial Transcriptomics (ST). For both, tonsil sections served as controls.

Results: Unexpectedly, we identified B cell follicles (CD19/21⁺ foci) in pre-pubertal thymi, found exclusively in medulla. However, during involution (i.e., peri-pubertal thymi), we detected GC (CD19/CD21/BCL6⁺ foci). IMC corroborated these patterns, revealing medullary follicles and GC comprising unique cellular neighborhoods. Our findings were further validated by ST, which showed cell clusters with different gene expression in and surrounding follicles and GC. Moreover, tonsillar vs thymic GC comparisons demonstrated distinct transcriptional signatures.

Translational impact: We have established thymic B cell follicles develop as puberty approaches, and GC form coincident with puberty and involution – and these structures reside in distinct medullary cellular neighborhoods. We hypothesize follicle and GC formation may play a role in involution, and that involution perturbations may spawn thymic lymphoid malignancies. Future work will investigate the antigenic targets that trigger the genesis of thymic follicles and GC, and genetic pathways underlying their formation.

Funding: W. J. Jones Family Foundation, Presbyterian Health Foundation (PHF) Seed Grant, Hyundai Hope On Wheels 2024 Impact Award

ABSTRACT TITLE

CILIARY PROTEOMICS REVEALS MULTI-PATHWAY SIGNALING DISRUPTION AND CANDIDATE THERAPEUTIC TARGETS IN ARMC9-RELATED JOUBERT SYNDROME

Felix Asare^{1,2}, Rose Johnson^{1,2}, Justyna Szymczak^{1,2}, Julie Van De Weghe^{1,2}

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ABSTRACT TITLE

CILIARY PROTEOMICS REVEALS MULTI-PATHWAY SIGNALING DISRUPTION AND CANDIDATE THERAPEUTIC TARGETS IN ARMC9-RELATED JOUBERT SYNDROME

Introduction: Primary and motile cilia are antenna-like organelles essential for signaling and development. These functions depend on strict regulation of their protein composition. Their dysfunction causes ciliopathies, including Joubert syndrome (JS), a neurodevelopmental disorder defined by a specific brain malformation, lifelong disability, and multi-organ disease, with no curative therapy. Although >40 JS genes are known, their loss has largely been examined using targeted assays, leaving the broader effects on the ciliary proteome unknown. We identified ARMC9 as a JS causal gene whose loss shortens cilia and reduces stabilizing modifications on the axonemal microtubules that support signaling. Because other ARMC proteins directly regulate ciliary protein composition, we hypothesized that ARMC9 loss remodels the ciliary proteome and disrupts signaling.

Methods: We isolated human primary cilia from ARMC9-knockout and control HEK293T cells and motile cilia from ARMC9-mutant and wild-type *Chlamydomonas*, a unicellular alga, then compared proteomes by data-independent acquisition mass spectrometry.

Results: ARMC9 loss remodeled both proteomes. In primary cilia, 1,433 proteins were altered (adjusted $P < 0.05$), and pathway enrichment analysis predicted dampened Ephrin, GPCR, WNT, and Hedgehog signaling and impaired cilium assembly. Protein-interaction analysis linked the top 30 altered proteins to WNT, Hedgehog, and other JS-associated proteins. In motile cilia, 119 proteins were altered, with signaling proteins predominantly depleted and uncharacterized ciliary proteins accumulated. Both datasets converged on disrupted signaling machinery. qPCR validated blunted induction of Hedgehog and WNT target genes after individual pathway stimulation in human knockout cells.

Translational Impact: Our data support coordinated disruption of multiple signaling pathways as a candidate mechanism of ARMC9-related JS, with Ephrin changes providing a candidate link to the abnormal axon guidance underlying the JS brain malformation. Using patient-derived cells and neuronal models, we will determine whether restoring ARMC9 or pharmacologically targeting these pathways rescues ciliary, signaling, and axon-guidance phenotypes, prioritizing targets for mechanism-based JS therapy.

Funding

NICHD R00HD100554

ABSTRACT TITLE

WIDE-FIELD MULTISPECTRAL FD-FLIM DERMOSCOPE WITH DUAL EXCITATION FOR MACHINE-LEARNING-ASSISTED SKIN CANCER ASSESSMENT

Lutfun Nahar Asha¹, Gabriel Tortorelli¹, Rodrigo Cuenca¹, Luis Rosales-Casas¹, Eveling Castro-Gutierrez², Marco Aedo-Lopez², Milagros Zegarra-Mejía², Edwin Llamoca², Karla Ancí-Paredes², Claudia Mares², Mónica Ruiz-Ballón², Patricia Rivera², Mauricio Postigo², Angel Choquehuanca-Peraltilla², Christian Suca-Velando², Valentina Chambilla-Perca², Michael Sucle-Suca², Javier A. Jo¹

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ABSTRACT TITLE

WIDE-FIELD MULTISPECTRAL FD-FLIM DERMOSCOPE WITH DUAL EXCITATION FOR MACHINE-LEARNING-ASSISTED SKIN CANCER ASSESSMENT

Abstract:

Introduction: Accurate, noninvasive assessment of suspicious skin lesions remains challenging because conventional visual inspection and dermoscopy rely primarily on morphological features and clinician expertise. Autofluorescence lifetime imaging provides complementary biochemical and structural contrast that may improve lesion characterization. We developed a wide-field frequency-domain fluorescence lifetime imaging (FD-FLIM) dermoscope incorporating dual-wavelength excitation and multispectral emission detection for label-free assessment of cutaneous lesions.

Methods: The system uses excitation at 375 and 473 nm with four emission channels centered at 405, 442, 525, and 637 nm to capture autofluorescence associated with endogenous fluorophores including NADH, FAD, collagen, elastin, protoporphyrin IX, and lipofuscin. System lifetime measurements were validated using fluorescence lifetime standards against calibrated time-correlated single-photon counting (TCSPC) measurements.

Results: FD-FLIM measurements demonstrated close agreement with TCSPC reference measurements, with fluorescence lifetime differences below 5%. The system provides a field of view of approximately 295 mm² with an imaging resolution of approximately 200 μ m. Fluorescence lifetime measurements of the evaluated fluorophores were reproducible across repeated measurements, supporting the stability of the system for quantitative lifetime imaging.

Translational Impact: The dermoscope is designed for clinical evaluation of suspicious skin lesions and will be deployed at a hospital in Arequipa, Peru. FLIM images will be linked with clinical assessments and histopathological diagnoses to establish a clinically annotated dataset. Autofluorescence intensity and lifetime features will subsequently be evaluated using machine-learning models to classify benign, premalignant, and malignant lesions. This label-free approach could provide objective biochemical and structural information complementary to conventional dermoscopy and ultimately support noninvasive assessment of suspicious skin lesions.

POSTER PRESENTATIONS

SESSION 2 | 5B

ABSTRACT TITLE

MECHANISM OF OSTRINIA FURNACALIS PBP2 FUNCTION: ROLE OF THE C-TERMINAL TAIL

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ABSTRACT TITLE

MECHANISM OF OSTRINIA FURNACALIS PBP2 FUNCTION: ROLE OF THE C-TERMINAL TAIL

Introduction

Ostrinia furnacalis, also known as Asian Corn Borer, a lepidopteran moth, is an agricultural pest found across Asia, Australia, Africa, and some regions of the United States. It is responsible for the destruction of over one-third of the total crops and food stored globally. The common use of pesticides is detrimental to the crops as well as the environment. Many produces contain as many as 30 different pesticide residues that are harmful to human health. The *O. furnacalis* pheromone-binding protein 2 (OfurPBP2) located in the male antennae is involved in detecting female-emitted pheromones, a crucial step in the mating process. The binding of pheromone to OfurPBP2 is the first step in the signaling events where a chemical signal translates into an electrical signal resulting in the behavioral response in the male.

Methods

Two biological gates (His70–His95 and C-terminus) have been reported to be involved in the ligand release in several well-studied lepidopteran PBPs. In contrast, OfurPBP2 contains an Arg70–His88 gate and a C-terminus containing additional charged residues. To investigate the role of the C-terminus in pheromone binding and release, we have deleted the C-terminal tail of OfurPBP2 generating the OfurPBP Δ 129–Q144 truncated mutant.

Results

We will present the recombinant protein expression, refolding, purification and the impact of the truncation on protein folding.

Translational Impact

Understanding the mechanism of pheromone binding and release would ultimately help in the development of sustainable pest management strategy.

Funding: This research is financially supported by NSF Awards CHE-1807722 (S.M.), CHE-2402694 (S.M.).

POSTER PRESENTATIONS

SESSION 2 | 7B

ABSTRACT TITLE

SELF-ASSEMBLING PEPTIDE-BASED THERAPEUTIC STRATEGY FOR INDUCING PROGRAMMED CELL DEATH IN BREAST CANCER MODELS

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ABSTRACT TITLE

SELF-ASSEMBLING PEPTIDE-BASED THERAPEUTIC STRATEGY FOR INDUCING PROGRAMMED CELL DEATH IN BREAST CANCER MODELS

Introduction

Breast cancer remains a major therapeutic challenge due to tumor heterogeneity and limited treatment effectiveness across diverse disease subtypes. Developing novel therapeutic platforms that effectively induce cancer cell death while providing mechanistic understanding remains a significant challenge in cancer treatment. Self-assembling peptides represent a promising class of biomaterials capable of forming functional nanostructures and interacting with cellular components. Previously, we demonstrated that bovine serum albumin (BSA) can modulate the aggregation behavior of an oppositely charged self-assembling peptide system, [II], enabling control over peptide assembly and biological activity. In this study, we investigated BSA-regulated [II] peptide assemblies for their ability to induce programmed cell death in human and murine breast cancer models and explored cellular mechanisms regulating therapeutic responses.

Methods

Multiple breast cancer cell models were treated with self-assembling [II] peptides under controlled aggregation conditions regulated by BSA. Cellular responses were evaluated using viability assays, membrane integrity analysis, and apoptotic cell death assays. Biological responses and cellular stress changes following peptide treatment were analyzed to identify factors influencing therapeutic outcomes.

Results

Self-assembling [II] peptides induced significant cancer cell death across multiple breast cancer models, with variations in sensitivity and cellular response patterns observed among models. Peptide treatment resulted in increased membrane damage and activation of intracellular stress pathways associated with programmed cell death. Modulation of stress-related mechanisms altered peptide-induced cytotoxic responses, demonstrating involvement of cellular stress regulation in therapeutic activity. These findings provide mechanistic insight into how self-assembling peptide structures interact with cancer cells to promote cell death.

Translational Impact

This study investigates a self-assembling peptide-based therapeutic platform for breast cancer treatment and provides mechanistic insights to guide optimization. By identifying cellular pathways involved in peptide-mediated cancer cell death, this approach may support the development of targeted biomaterial therapies and combination strategies for improving treatment outcomes.

Funding

This work was supported by the Peggy and Charles Stephenson Professorship #2 Endowment at the Stephenson School of Biomedical Engineering, University of Oklahoma, and the National Institute of General Medical Sciences (NIGMS) of NIH under award R35GM150970.

Footnotes

The authors acknowledge the support and contributions of all individuals and facilities involved in this research.

POSTER PRESENTATIONS

SESSION 2 | 8B

ABSTRACT TITLE

ACCELERATING ACQUISITION OF DEVICE ASSISTED CRAWLING THROUGH INCREASED FREQUENCY OF INTERVENTION

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ABSTRACT TITLE

ACCELERATING ACQUISITION OF DEVICE ASSISTED CRAWLING THROUGH INCREASED FREQUENCY OF INTERVENTION

Abstract

Introduction: Task-specific training is essential for enhancing motor skill acquisition. Assistive therapeutic devices are increasingly used in rehabilitation. Determining optimal intervention dosing is important for movement learning and efficient and effective care for children with neuromotor disorders. Prone locomotion, the earliest mobility skill, improves cognitive and spatial development, motor memory, and parent-child attachment.

Evidence supports infants learning to use an assistive device for crawling. However, optimal training frequency on prone locomotor skill acquisition using robotic-assisted training has not been established. This study examined the effect of training dose on skill acquisition in infants learning prone locomotion using the Self-Initiated Prone Progressive Crawler (SIPPC3). We hypothesized that infants training at higher frequencies would demonstrate earlier and more proficient prone locomotor skills than at lower frequencies.

Methods: Seven typically developing infants (4–7 months) participated in a nonconcurrent multiple-baseline single-case design. After a two-week baseline, infants trained on the SIPPC3 four times/week (Group A), three times/week (Group B), or two times/week (Group C). Outcomes included goal-directed prone mobility proficiency measured with the Movement Observation Coding Scheme, Kinematic characteristics generated by the SIPPC3, and the Bayley Scales of Infant and Toddler Development, Third Edition (Bayley-III) gross motor scale.

Results: Higher-frequency training was associated with earlier attainment of goal-directed prone mobility, larger effect sizes for prone mobility proficiency, greater mean distance traveled, and higher Bayley-III gross motor scores compared with lower-frequency groups.

Translational Impact: These findings inform dosing of early task-specific interventions using assistive therapeutic devices. Preliminary findings suggest higher-frequency task-specific training using a robotic device is associated with earlier acquisition and greater proficiency of prone locomotion in infants at low risk for CP. Higher-frequency training increases practice repetitions, a key component of motor learning. Findings provide preliminary dosing parameters that can be investigated in larger samples and infants at risk for cerebral palsy.

Funding details: Funding for this study was partially supported by funds from the Office of Special Education training grant # H325K120310 and the National Science Foundation NRI #1208639.

Disclosure statement: The authors report there are no competing interests to declare.

ABSTRACT TITLE

PRECLINICAL EVALUATION OF BIPHASIC HYDROGEL FOR LOCAL MULTI-AGENT CHEMOTHERAPEUTIC DELIVERY TO GLIOBLASTOMA

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ABSTRACT TITLE

PRECLINICAL EVALUATION OF BIPHASIC HYDROGEL FOR LOCAL MULTI-AGENT CHEMOTHERAPEUTIC DELIVERY TO GLIOBLASTOMA

Introduction: Glioblastoma (GBM) is among the most aggressive primary brain tumors, affecting approximately 12,000 patients annually in the United States with a 7% average 5-year survival[1]. The standard of care combines surgical resection with adjuvant chemoradiotherapy, but efficacy remains limited by blood–brain barrier (BBB) transport. To bypass the BBB, we developed an injectable, biphasic hydrogel platform containing drug-loaded polymeric nanoparticles. We hypothesize that this system can enable long-term release of synergistic drug combinations in GBM.

Methods: Athymic mice (n=5) bearing orthotopic G55 human gliomas were treated with saline, unloaded hydrogel, or doxorubicin-loaded hydrogel. Mice were monitored for up to 60 days with one group sacrificed at 14 days to analyze doxorubicin retention. In vitro degradation of hydrogel discs (n=3) was quantified by disc thickness, stiffness, and mass for up to 42 days. Hydrogel biocompatibility was evaluated using L929 murine fibroblasts (n=6) exposed to the hydrogel's individual components for 24 hours. Biocompatibility was measured by lactate dehydrogenase (LDH) release and metabolic assays. Drug synergy was evaluated in G55 human glioma cells (n=6) following 72-hour exposure to combinations of doxorubicin, temozolomide, SN38, and fasudil. The Bliss Independence model was used to quantify synergistic effects.

Results: Doxorubicin remained detectable by fluorescence microscopy at 14 days and 50% of mice treated with doxorubicin-loaded hydrogels survived 60 days compared with 20% and 0% of saline and unloaded gel groups, respectively. Degradation studies showed a 50.1% reduction in mass with no significant change in gel stiffness or thickness, suggesting the evacuation of nanoparticles. Biocompatibility testing showed no cytotoxicity to L929 fibroblasts. In synergy experiments, fasudil enhanced chemotherapeutic efficacy, with the strongest effect observed with doxorubicin.

Translational Impact: Our biphasic hydrogel provides a safe and effective strategy for bypassing the BBB to deliver synergistic therapy combinations directly to peritumoral tissue improving GBM patient long-term survival.

Funding Acknowledgement: Support is gratefully acknowledged from the American Cancer Society Institutional Research Grant (ACS-IRG-23-01), the National Science Foundation (ART InTRO, NSF Award #2331409) and the US EDA (via OKBioStart) (08-79-05678).

[1] D. Eisenbarth and Y. A. Wang, "Glioblastoma heterogeneity at single cell resolution," *Oncogene*, vol. 42, no. 27, pp. 2155–2165, Jun. 2023, doi: 10.1038/s41388-023-02738-y.

POSTER PRESENTATIONS

SESSION 2 | 12B

ABSTRACT TITLE

PEPTIDE THERAPY TARGETING AMYLOID-B AND INFLAMMATORY PATHWAYS IN ALZHEIMER'S DISEASE: IN PHARMACOKINETICS AND TARGET ENGAGEMENT OUTCOMES IN MOUSE.

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ABSTRACT TITLE

PEPTIDE THERAPY TARGETING AMYLOID- β AND INFLAMMATORY PATHWAYS IN ALZHEIMER'S DISEASE: IN PHARMACOKINETICS AND TARGET ENGAGEMENT OUTCOMES IN MOUSE.

Introduction: Alzheimer's disease (AD) is an age-related neurodegenerative disorder marked by progressive cognitive and memory decline, amyloid- β (A β) accumulation, and neuroinflammation. Current FDA-approved therapies are largely mono-targeted and do not effectively address disease causation or halt progression. Our research aims to develop a multitarget peptide drug simultaneously targeting A β and key neuroinflammatory mediators, potentially improving therapeutic efficacy.

Methods: To evaluate peptide half-life, diffusion, and brain distribution, wild-type (WT) mice received a single stereotaxic-guided bolus injection of ALZ100 or ALZ300 (1 μ L, 5 mM in saline) into the right lateral ventricle. Cortex and hippocampus were collected at 0–180 minutes and analyzed by LC–MS/MS. For therapeutic studies, osmotic pumps delivering saline, ALZ100, or ALZ300 (1 mM; 0.25 μ L/h) were implanted for 13 days in WT and APP/PS1(TG) mice. Brain tissues were subsequently analyzed for A β oligomers and inflammatory markers, including S100A9, IL-6, TNF- α , and IL-1 β , using biochemical and immunohistochemical methods.

Results: At 45 min post bolus injection, peptide was detectable and quantified in ipsilateral cortex and hippocampus but not in the contralateral cortex suggesting limited diffusion. Half-life was estimated at 20 min, suggesting rapid degradation. In TG mice, intracerebroventricular infusion of ALZ300 induced IL-1 β in the ipsilateral cortex. A β oligomers, A β plaques and activated immune cells were observed in AD mice brains. Elevation of S100A9 was observed with aging, both in the WT and TG mice. ALZ100 and ALZ300 did not show any effect on A β oligomers or S100A9 at 1mM, when infused for 13 days.

Translational Impact: Taken together, our results suggest that our peptide candidates should not be used naked. A liposomal formulation could increase diffusion, protect against degradation, prevent induction of IL-1 β , and allow for less invasive and longer treatment using intranasal delivery. Our next goal is to develop and optimize liposomal formulation for these peptides.

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09/20/2024–08/31/2025, under NCE until 08/31/2026
Categories: b (commercial translational) and c (preclinical).

POSTER PRESENTATIONS

SESSION 2 | 15B

ABSTRACT TITLE

ENHANCING THE ACTIVITY OF THERAPEUTIC COMPOUNDS WITH FORMULATIONS OPTIMIZED BY QBD

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ABSTRACT TITLE

ENHANCING THE ACTIVITY OF THERAPEUTIC COMPOUNDS WITH FORMULATIONS OPTIMIZED BY QBD

Introduction. Many promising therapeutic compounds remain in the discovery phase due to limitations of their physicochemical properties such as poor aqueous solubility and limited in vivo efficacy. Methods. My lab employs the Quality by Design (QbD) approach recommended by the FDA to reduce the time required to develop a target product so that it will meet its desired clinical performance. The steps of this approach are: (1) Define desired product performance and identify product critical quality attributes (CQAs); (2) Design formulation and process to meet product CQAs. (3) Understand impact of material and process parameters on product critical quality. Using QbD methodology, we have optimized the following formulations to improve the solubility, dissolution and efficacy of SHetA2 (OK1), a promising compound with anti-cancer and anti-tuberculosis activity: microparticles for inhalation as well as vaginal suppositories and capsules for oral delivery, currently in clinical trials. Results. SHetA2 microparticles dissolved much faster and to a greater extent ($21.19 \pm 4.40\%$) than the unprocessed drug ($3.51 \pm 0.9\%$). Vaginal administration of a 15-mg/kg dose achieved an area under the cervix concentration versus time curve that was ~120 times larger than that after a 60 mg/kg administered orally. The solubility of SHetA2 in the capsule formulations was $6 \mu\text{g/ml}$ compared to $0.02 \pm 0.01 \mu\text{g/ml}$ for unprocessed drug. Translational Impact. Inclusion of promising therapeutic compounds into optimized pharmaceutical formulations can enhance their therapeutic effect by several fold and produce a dosage form that can be used in clinical trials.

ABSTRACT TITLE

SYNERGISTIC Fe^{3+} CHELATION AND RADICAL SCAVENGING BY DOPAMINE-BASED SCHIFF BASES TO IMPROVE MYOGLOBIN REDOX STABILITY

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ABSTRACT TITLE

SYNERGISTIC Fe^{3+} CHELATION AND RADICAL SCAVENGING BY DOPAMINE-BASED SCHIFF BASES TO IMPROVE MYOGLOBIN REDOX STABILITY

Meat discoloration is primarily associated with myoglobin oxidation, which negatively affects shelf life and consumer acceptance. During storage, free ferric iron (Fe^{3+}) can promote oxidative reactions, enhance reactive oxygen species (ROS) generation, and accelerate the conversion of oxymyoglobin to metmyoglobin. Therefore, antioxidants possessing multiple functional properties, including metal-chelating, radical-scavenging, and myoglobin-stabilizing activities, may offer an effective strategy for improving meat color stability. This study aimed to synthesize dopamine-based Schiff base antioxidants and evaluate their Fe^{3+} -chelating capacity, free-radical-scavenging activity, and ability to inhibit oxidative transformations of myoglobin.

Schiff base compounds were designed and synthesized through imine reaction and characterized through ^1H NMR. Their Fe^{3+} -chelating ability was investigated using UV–visible spectroscopy, while antioxidant capacity was assessed using DPPH and ABTS radical-scavenging assays. The protective effects of the compounds against myoglobin oxidation were evaluated at pH 5.6 and 7.4, representing conditions relevant to meat and physiological systems, respectively.

The Schiff bases exhibited substantial radical-scavenging activity and significantly reduced oxidative changes in myoglobin. They inhibited metmyoglobin formation compared with untreated systems at both pH conditions, indicating improved myoglobin redox stability. Protection against Fe^{3+} -mediated oxidation further suggested that metal chelation contributed to the observed antioxidant effects ($P < 0.05$). Suppression of ferrylmyoglobin formation would additionally support interruption of oxidative propagation pathways.

Overall, dopamine-based Schiff base antioxidants represent promising multifunctional compounds capable of protecting myoglobin through combined radical scavenging, Fe^{3+} chelation, and inhibition of higher oxidation-state myoglobin formation. Future studies should evaluate their effectiveness in fresh meat systems for improving color stability and extending shelf life.

POSTER PRESENTATIONS

SESSION 2 | 19B

ABSTRACT TITLE

QUANTITATIVE SURFACE DCLK1 ANTIGEN DENSITY ANALYSIS PREDICTS TARGETABILITY OF CHEMORESISTANT OVARIAN CANCER BY CAR T CELLS

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ABSTRACT TITLE

QUANTITATIVE SURFACE DCLK1 ANTIGEN DENSITY ANALYSIS PREDICTS TARGETABILITY OF CHEMORESISTANT OVARIAN CANCER BY CAR T CELLS

Introduction: High-grade serous ovarian carcinoma (HGSOC) is the most common and lethal subtype of ovarian cancer. Although most patients initially respond to platinum-based chemotherapy, nearly 80% ultimately develop resistant disease. Chimeric antigen receptor (CAR) T cell therapy is a promising therapeutic strategy; however, its efficacy in solid tumors is limited by tumor antigen scarcity and heterogeneity, poor tumor penetration, and immunosuppression. The diffuse intraperitoneal dissemination characteristic of HGSOC may nevertheless provide a particularly favorable setting for this approach. We have established that doublecortin-like kinase 1 (DCLK1) is elevated in HGSOC cell lines, is associated with poor prognosis, and promotes tumor cell stemness, chemoresistance, epithelial-to-mesenchymal transition, invasion, and metastasis. DCLK1 exists as four major isoforms, of which isoforms 2 and 4 contain extracellular domains accessible to antibody-based targeting. Our preliminary studies show elevated DCLK1 isoforms in malignant ascites-derived tumor cells, with further enrichment in platinum- and poly(ADP-ribose) polymerase inhibitor (PARPi)-resistant derivatives, while prior studies show limited expression in most normal adult tissues. In this study we sought to define the surface expression and antigen density of DCLK1 in HGSOC patients and determine the therapeutic efficacy of DCLK1-targeted CAR T cells in patient-derived tumor models.

Methods: DCLK1 isoforms 2 and 4 were quantified in HGSOC tissues (N=127) using immunohistochemistry with quantitative image analysis using HALO software. DCLK1 antigen density was measured in parental and drug-resistant ovarian cancer cell lines using fluorescently labeled anti-DCLK1 antibodies and quantitative flow cytometry calibrated with fluorescence reference beads. We then evaluated DCLK1-targeted CAR T-cell efficacy and safety using three-dimensional spheroids derived from patients.

Results: HGSOC tumors displayed elevated expression of DCLK1 isoforms 2 and 4, with a mean of 65.6% of tumor cells staining per case and predominantly moderate-to-strong (2+ to 3+) staining intensity. Drug-resistant ovarian cancer models demonstrated increased surface DCLK1 antigen density compared with parental cells. DCLK1-targeted CAR T cells selectively recognized and effectively eliminated DCLK1-expressing chemoresistant ovarian cancer cells in vivo and ex vivo, supporting DCLK1 as an actionable target for cellular immunotherapy.

Translational Impact: These findings support DCLK1-targeted CAR T-cell therapy as a promising therapeutic strategy for chemoresistant HGSOC, including its potential use in combination with existing therapies. Ongoing studies will integrate quantitative DCLK1 antigen-density measurements with functional assays in patient-derived ascites models and xenografts to define the relationships between target abundance and therapeutic efficacy and establish an antigen-density threshold to guide future patient selection.

Funding: Ovarian Cancer Research Alliance (CRDG-2024-3-1550)

POSTER PRESENTATIONS

SESSION 2 | 20B

ABSTRACT TITLE

LIVER-DERIVED FGF21 REGULATES HEPATIC GLUCOSE, LIPID AND MITOCHONDRIAL ADAPTATION TO BRANCHED-CHAIN AMINO ACID RESTRICTION

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ABSTRACT TITLE

LIVER-DERIVED FGF21 REGULATES HEPATIC GLUCOSE, LIPID AND MITOCHONDRIAL ADAPTATION TO BRANCHED-CHAIN AMINO ACID RESTRICTION

Introduction. Dietary branched-chain amino acid (BCAA) restriction enhances hepatic insulin sensitivity and glucose metabolism and induces fibroblast growth factor 21 (FGF21), which is known for improving glucose and lipid metabolism. The objective was to determine whether liver-derived FGF21 mediates the beneficial effect of low-BCAA diets in HepG2 cells.

Methods. A pLKO.1 vector carrying shRNA against human FGF21, or a non-targeting scramble shRNA (shSCR), was packaged into lentivirus in HEK-293T cells using psPAX2 and pMD2.G. HepG2 cells were transduced and stable knockdown (KD) lines selected with puromycin, with knockdown efficiency confirmed by qPCR and immunoblotting. After overnight serum starvation, KD and Scr cells were incubated in control (CON) or BCAA-depleted, i.e., (–) BCAA media for 48 h, then harvested to assess expression of protein and genes involved in glycogenesis, lipolysis, and mitochondrial biogenesis.

Results. In shSCR (control) cells, (–) BCAA increased pGSK3 β (Ser9) protein expression, an effect absent in KD cells, indicating that FGF21 is required for BCAA depletion-induced glycogenesis. (–) BCAA also increased mRNA abundance ACOX1, LPL, CPT1 α and HSL in shSCR cells, whereas in KD cells this effect either was absent (for ACOX1 and LPL) or diminished (for CPT1 α and HSL) suggesting that FGF21 fully or partially mediates lipolysis in response to BCAA depletion. Similarly, PGC1 β , SOD1, SOD2, NRF1, NRF2 and AMPL1 α gene expression were increased with (–) BCAA in shSCR cells, with all these genes showing either no upregulation or decreased expression in KD cells indicative of the role of FGF21 in mitochondrial biogenesis in liver.

Translational Impact. Hepatic FGF21 partly or fully mediates BCAA depletion-induced glycogenesis, lipolysis and mitochondrial biogenesis in HepG2 cells. This finding may lead to identifying dietary and molecular targets for preventing and treating diabetes and related metabolic disorders. Future preclinical work will be required to confirm these findings.
Keywords: BCAA depletion, FGF21, Glycogenolysis, Lipolysis, Mitochondrial Biogenesis.

Funding Sources: Hatch project accession no. 1012889 from the USDA National Institute of Food and Agriculture (NIFA)

Footnotes: Findings are based on an immortalized human hepatocyte line and require confirmation in preclinical models.

POSTER PRESENTATIONS

SESSION 2 | 23B

ABSTRACT TITLE

HEPATOCYTE MLKL DRIVES OBESITY-DRIVEN HEPATOCELLULAR CARCINOMA PROGRESSION VIA MITOCHONDRIAL DYSFUNCTION INDEPENDENT OF NECROPTOSIS IN MASLD

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ABSTRACT TITLE

HEPATOCTYTE MLKL DRIVES OBESITY-DRIVEN HEPATOCELLULAR CARCINOMA PROGRESSION VIA MITOCHONDRIAL DYSFUNCTION INDEPENDENT OF NECROPTOSIS IN MASLD

Introduction:

Metabolic dysfunction–associated steatotic liver disease (MASLD) is a leading cause of HCC, particularly in obesity, yet mechanisms linking hepatocyte dysfunction to tumorigenesis remain unclear. Mixed lineage kinase domain–like protein (MLKL), the effector of necroptosis, is elevated in MASLD, but its hepatocyte–intrinsic role in obesity–driven MASLD–HCC is unknown.

Methods and Results:

Using a long-term western diet (WD)–induced MASLD–HCC model in hepatocyte–specific MLKL knockout (MklHepKO) mice, we defined MLKL’s hepatocyte–intrinsic function. WD increased hepatocyte MLKL protein expression without detectable necroptosis activation, indicating a necroptosis–independent role. MLKL deficiency did not alter WD–induced inflammation, fibrosis, or liver injury but increased hepatic lipid accumulation while reducing lipotoxic lipid species and preserving mitochondrial function. WD–fed MklHepKO mice developed fewer and smaller tumors with reduced incidence, multiplicity, proliferation, and stemness. Transcriptomic analysis revealed upregulation of mitochondrial oxidative phosphorylation pathways in MklHepKO livers. WD suppressed the mitochondrial fusion protein and tumor suppressor mitofusin 2 (MFN2), whereas MLKL deficiency restored MFN2 expression post–translationally. In HCC cells, MLKL deletion reduced proliferation, improved mitochondrial respiration, and decreased glycolysis; these effects were reversed by MFN2 deletion. MLKL is localized to nuclear and mitochondrial compartments, consistent with organelle–intrinsic functions. The human MLKL inhibitor necrosulfonamide (NSA) suppressed HepG2 xenograft growth, and elevated MLKL expression in human HCC correlated with poorer overall survival.

Translational Impact:

This study identifies hepatocyte MLKL as a druggable, necroptosis–independent driver of obesity–associated MASLD–HCC. By linking MLKL–mediated mitochondrial dysfunction and MFN2 suppression to liver tumorigenesis, these findings support the MLKL–MFN2 axis as a novel therapeutic target and biomarker for MASLD–associated HCC.

FUNDING

R01AG059718, R03 CA262044, Harold Hamm Diabetes Center–Stephenson Cancer Center Team Science Grant, Oklahoma Center for Adult Stem Cell Research (OCASCR) grant to SSD.

POSTER PRESENTATIONS

SESSION 2 | 26B

ABSTRACT TITLE

ROLE OF GATING AND C-TERMINAL REGIONS IN OFURPBP2

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ABSTRACT TITLE

ROLE OF GATING AND C-TERMINAL REGIONS IN OFURPBP2

Introduction: Olfaction allows moths to detect signals, enabling males to sense pheromones and locate mates. Disrupting this communication could provide an alternative to insecticides, which raise health and environmental concerns. OfurPBP2, a pheromone-binding protein from the Asian corn borer, *Ostrinia furnacalis*, shows pH-dependent changes associated with ligand release. In *Antheraea polyphemus* PBP1 (ApolPBP1), the histidine gate and C-terminal region contribute to ligand binding and release. OfurPBP2 differs from ApolPBP1 in these regions, including Arg70 and its C-terminal sequence. We generated a chimeric OfurPBP2 mutant containing R70H and ApolPBP1 C-terminal region to examine their structural contribution.

Methods: The mutant was expressed in *E. coli* and purified by DEAE ion-exchange and size-exclusion chromatography. SDS-PAGE confirmed purity. CD with SELCON3 and ¹H-¹⁵N HSQC NMR characterized the mutant at pH 6.5, 5.5, and 4.5, then returned to pH 6.5.

Results: The mutant showed a dispersed HSQC spectrum at pH 6.5, consistent with a folded protein. At pH 4.5, peak loss occurred. CD analysis showed α -helical content decreased from 61.8% at pH 6.5 to 52.0% at pH 4.5, with near-UV CD changes between 250 and 320 nm, suggesting changes in the local environment and packing of aromatic residues. Compared with wild-type OfurPBP2, which showed a more extensive low-pH transition, the mutant showed a less pronounced response while retaining substantial secondary and tertiary structure. As in wild-type OfurPBP2, the HSQC spectrum recovered its well-dispersed pattern after returning to pH 6.5, demonstrating reversibility. These findings suggest that R70 and the C-terminal region contribute to the extent of the acidic-pH conformational transition in OfurPBP2.

Table 1. pH-dependent structural changes of OfurPBP2 R70H/C-terminal swap mutant

pH condition	α -Helix (%)	β -Sheet (%)	HSQC observation	Structural interpretation
6.5	61.8	2.7	Well dispersed	Folded state
5.5	—	—	Slight spectral change	Early conformational change
4.5	52.0	6.1	Substantial peak loss	pH-dependent structural rearrangement
6.5 (return)	—	—	Well dispersed again	Reversible recovery

Translational Impact: Defining OfurPBP2 structural features may help identify targets to disrupt pheromone detection. This work may support selective inhibitors that interfere with male moths' ability to locate females and provide an alternative to broad-spectrum insecticide control.

Funding: This research was supported by the National Science Foundation under Award CHE-2402694 to Dr. Smita Mohanty.

POSTER PRESENTATIONS

SESSION 2 | 28B

ABSTRACT TITLE

PRELIMINARY INVESTIGATION OF RAMAN SPECTRAL SIGNATURES FOLLOWING CISPLATIN TREATMENT IN OVARIAN CANCER CELL LINES

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ABSTRACT TITLE

PRELIMINARY INVESTIGATION OF RAMAN SPECTRAL SIGNATURES FOLLOWING CISPLATIN TREATMENT IN OVARIAN CANCER CELL LINES

Introduction

Chemotherapy resistance remains a major challenge in the treatment of ovarian cancer, contributing to disease recurrence and poor patient outcomes. Identifying biochemical signatures that are associated with treatment response could improve patient monitoring and support the development of non-invasive methods for assessing the effectiveness of current treatment methods. This study investigated whether Raman spectroscopy combined with computational analysis could distinguish treatment-sensitive and treatment-resistant ovarian cancer cell lines following cisplatin exposure.

Methods

Raman Spectra were collected from OVCAR4 and OVCAR8 cell lines at baseline and 24 and 48 hours following exposure to cisplatin. Spectra were then baseline corrected, normalized and smoothed before analysis. Principal Component Analysis (PCA) was used to evaluate treatment associated spectral variation and identify Raman shifts contributing to group separation. Welch's t-test was performed on selected Raman bands to assess statistical significance.

Results

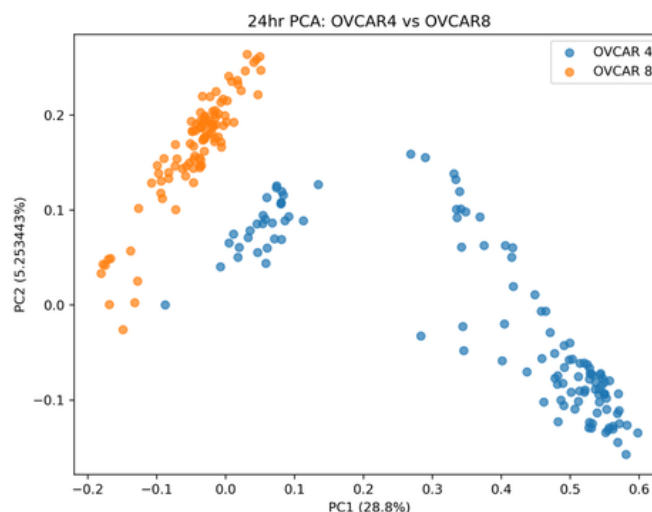
PCA demonstrated a clear separation between baseline, 24-hours and 48 hours treatment groups in both cell lines. Direct comparison of OVCAR4 and OVCAR8 showed distinct spectral signatures at both post treatment time points. Separation at 24 hours was primarily driven by the bands near 1092, 1298, 1440 and 1660 cm^{-1} , while separation at 48 hours was primarily driven by bands near 961, 1004 and 1450 cm^{-1} . These regions are associated with DNA, protein and lipid vibrations. Welch's t-test confirmed statistical significant differences between treatment sensitive and treatment resistant cell lines ($p < 0.001$).

Translational Impact

These findings demonstrate the potential of Raman spectroscopy combined with computational tools to identify biochemical signatures associated with cisplatin response in ovarian cancer. This work provides a foundation for future investigation of Raman spectroscopy as a non-invasive technique for monitoring chemotherapy response and supporting personalized treatment strategies.

Funding

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ABSTRACT TITLE

MINIMALLY INVASIVE MICRONEEDLE SENSORS FOR MOLECULAR DIAGNOSTICS: A TRANSLATIONAL WEARABLE PLATFORM

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ABSTRACT TITLE

MINIMALLY INVASIVE MICRONEEDLE SENSORS FOR MOLECULAR DIAGNOSTICS: A TRANSLATIONAL WEARABLE PLATFORM

Continuous physiological monitoring provides unprecedented opportunities for early disease detection, personalized treatment, and autonomous healthcare delivery. The dermal interstitial fluid (ISF) can be accessed in a minimally invasive manner by wearable microneedle-based electrochemical biosensors, one of the point-of-care technologies, thus enabling the real-time detection of clinical biomarkers without frequent phlebotomy. Here we present a high-performance microneedle sensor for detection of metabolic targets in artificial interstitial fluid (AISF). We show that by using glucose as a model biomarker, polymer transport-gated enzymatic electrochemical interfaces (MN/CP/PB/GOx-CHI-PEG-PEGDE), combining a prussian blue (PB) transducer for low potential hydrogen peroxidase (H_2O_2) reduction with glucose oxidase (GOx) immobilized in a chitosan-poly(ethylene glycol) network crosslinked with poly(ethylene glycol) diglycidyl ether (PEGDE), restricting substrate diffusion without altering intrinsic enzyme kinetics to deliver excellent sensitivity, linear range, rapid response times and strong biofouling resistance over long operation times. Its microarchitecture minimizes signal drift and bio-interfacial attenuation, enabling accurate real-time tracking of dynamic concentration fluctuations under physiological conditions. with a LOD of 0.1 mM in AISF, our system produces a linear range of 0–30 mM glucose, which falls within the physiological range of ISF. Also demonstrating consistent outcomes even when interfering molecules are present. Importantly, the modular electroanalytical platform and surface modification strategies developed in this glucose model system offer a direct translational pathway for the detection of cancer biomarkers. In validating this robust wearable architecture, our work closes the gap between fundamental electrochemical sensing and scalable continuous clinical diagnostics towards next-generation autonomous disease monitoring platforms.

ABSTRACT TITLE

STATIC COMPRESSION OPTICAL COHERENCE ELASTOGRAPHY FOR QUANTITATIVE ASSESSMENT OF BIOLOGICAL TISSUE ELASTICITY

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ABSTRACT TITLE

STATIC COMPRESSION OPTICAL COHERENCE ELASTOGRAPHY FOR QUANTITATIVE ASSESSMENT OF BIOLOGICAL TISSUE ELASTICITY

Abstract:

Introduction: Tissue stiffness can provide useful information about disease, but many existing elasticity measurement methods require complex equipment or external vibration. Static compression optical coherence elastography is a light-based imaging approach that measures how tissue changes during gentle compression and may provide a simple method for quantitative elasticity assessment.

Methods: Optical images were acquired before and after applying controlled compression to biological tissue. Small changes in the optical signal were calculated to determine tissue deformation. Samples were measured under the same loading and imaging conditions as reference materials with known mechanical properties. The tissue response was then compared with the reference materials to estimate quantitative elasticity. The approach was evaluated for tissue elasticity measurement, tissue classification, and identification of tumor margins.

Results: Static compression imaging generated spatially resolved measurements of tissue deformation and elasticity. Comparison with reference materials enabled quantitative assessment of mechanical properties. The method produced promising results for distinguishing tissues with different mechanical characteristics, classifying tissue types, and identifying regions associated with tumor margins.

Translational Impact: This approach does not require a separate vibration or excitation device and can be integrated with optical imaging systems. By providing structural images and quantitative mechanical information from the same measurement, it may support more objective tissue evaluation during diagnosis, surgery, and pathology assessment. Further validation in larger and clinically representative studies is needed before routine clinical use.

Acknowledgement of Funding: University of Oklahoma Health Sciences Center (3P30CA225520), National Science Foundation (OIA-2132161, 2238648, 2331409), National Institute of Health (R01DK133717), Oklahoma Center for the Advancement of Science and Technology (HR23-071), the medical imaging COBRE (P20 GM135009)

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POSTER PRESENTATIONS

SESSION 2 | 31B

ABSTRACT TITLE

OPTICAL COHERENCE TOMOGRAPHY DETECTS BILIARY MICROSTRUCTURAL ALTERATIONS FOR EVALUATING BILE DUCT VIABILITY IN LIVER TRANSPLANTATION

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ABSTRACT TITLE

OPTICAL COHERENCE TOMOGRAPHY DETECTS BILIARY MICROSTRUCTURAL ALTERATIONS FOR EVALUATING BILE DUCT VIABILITY IN LIVER TRANSPLANTATION

Purpose:

Biliary complications remain a major cause of graft dysfunction after liver transplantation. Current clinical imaging primarily detects late luminal abnormalities and cannot assess donor bile duct microstructure at graft evaluation. This study evaluates whether OCT can non-invasively quantify epithelial injury, stromal edema, and duct-wall structural context in human bile ducts.

Methods:

Human bile duct samples from accepted donor grafts, discarded donor grafts, and native bile ducts were imaged ex vivo using polarization-sensitive OCT. Custom 3D analysis quantified five OCT biomarker families: epithelial denudation, edema, wall thickness, luminal corrugation, and periductal tissue coverage. Representative OCT findings were compared with matched H&E histology.

Results:

OCT resolved a two-layer viability framework. Epithelial denudation and edema represented direct-injury biomarkers, whereas wall thickness, corrugation, and periductal tissue coverage represented structural/contextual descriptors. Epithelial denudation was the dominant injury signal and increased along the proximal-to-distal axis in discarded bile ducts. Edema showed heterogeneous intramural distribution and was most prominent in the middle wall compartment. Discarded ducts were descriptively thicker and more heterogeneous than accepted donor or native ducts. Exploratory analyses linked epithelial denudation with cold ischemia time and edema burden with warm ischemia time.

Conclusions:

OCT provides depth-resolved, label-free mapping of donor bile duct microstructure and supports a candidate five-biomarker framework for bile duct viability assessment. This approach may complement histology by identifying epithelial injury, stromal-fluid injury, and structural remodeling before transplantation. Future validation should incorporate PBG/PVP injury, OCTA vascular imaging, and post-transplant biliary outcomes.

POSTER PRESENTATIONS

SESSION 2 | 33B

ABSTRACT TITLE

HUMAN MYC OVEREXPRESSION IN ZEBRAFISH LYMPHOBLASTS TRIGGERS DIVERSE ACUTE LYMPHOBLASTIC LEUKEMIA SUBTYPES.

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ABSTRACT TITLE

HUMAN MYC OVEREXPRESSION IN ZEBRAFISH LYMPHOBLASTS TRIGGERS DIVERSE ACUTE LYMPHOBLASTIC LEUKEMIA SUBTYPES.

Introduction: Acute Lymphoblastic Leukemia (ALL) is a blood cancer arising from immature immune cells, with more than 40 known subtypes that differ in their genetic causes and behavior. Most subtypes share high levels of a cancer-driving gene called MYC. Using a zebrafish model that overexpresses human MYC in developing immune cells, we found that these fish unexpectedly develop at least five distinct types of leukemia, with many fish developing more than one type at once. We hypothesize that, as in humans, these leukemia types arise from different stages of immune cell development and depend on different genetic changes, which may explain why they grow, spread, and behave differently.

Methods: We used advanced sequencing methods to study these zebrafish leukemias, including sequencing of RNA from single cells to identify the developmental origin of each leukemia, and sequencing of the entire coding genome to track genetic changes acquired over time.

Results: Our results show that leukemias of the same broad type, whether arising from immune cell type B or T, tend to originate from similar developmental stages, though the amount of internal genetic diversity varies between individual leukemias. In some fish, different leukemia types share early genetic changes, indicating they arose from a common ancestral cell, even when they went on to become different immune cell types. These related leukemias also acquired distinct, later genetic changes, which may help explain their different behaviors.

Translational Impact: Fully characterizing the genetic features of these zebrafish leukemia types will allow comparison to their human counterparts. This work will help identify new subtype-specific treatment targets and provide a valuable model for drug testing, ultimately supporting the development of more effective, personalized therapies for leukemia patients.

ABSTRACT TITLE

TARGETING CD40L ATTENUATES PATHOGENIC INFLAMMATORY RESPONSES IN HUMAN MACROPHAGES

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ABSTRACT TITLE

TARGETING CD40L ATTENUATES PATHOGENIC INFLAMMATORY RESPONSES IN HUMAN MACROPHAGES

Introduction: Progressive multiple sclerosis (MS) is characterized by persistent neuroinflammation and limited therapeutic options for preventing disability progression. Macrophages are prominent mediators of inflammation and tissue injury in MS lesions. Notably, cells of the monocytic lineage, including macrophages and microglia, constitute the predominant CD40-expressing population in active MS lesions, implicating CD40L-CD40 signaling in myeloid-driven inflammation. Therapeutic CD40L blockade has shown clinical promise in MS, yet its direct effects on macrophage inflammatory function remain poorly understood. We investigated whether CD40L promotes inflammatory cytokine production in human monocyte-derived macrophages and whether these responses can be suppressed by CD40L neutralization.

Methods: Human monocyte-derived macrophages were exposed to soluble CD40L (sCD40L) and treated with a CD40L-neutralizing antibody or IgG control. Cell culture supernatants were analyzed using a multiplex bead-based cytokine assay to characterize inflammatory macrophage responses. Cytokine concentrations were assessed for normality and analyzed using appropriate parametric or nonparametric statistical tests.

Results: sCD40L induced a proinflammatory cytokine profile characterized by IL-12p40, IL-23, IL-12p70, and IFN-gamma production. CD40L neutralization significantly reduced IL-12p40 and IL-23 compared with sCD40L stimulation and significantly decreased IL-12p70 and IFN-gamma. In contrast, IgG control treatment did not reproduce the overall suppressive effect of CD40L neutralization, supporting a CD40L-specific effect on macrophage inflammatory activity.

Translational Impact: These findings identify CD40L as a modifiable regulator of inflammatory macrophage function and provide mechanistic support for therapeutically targeting CD40L in MS. Suppression of macrophage-derived inflammatory mediators provides a potential mechanism through which CD40L-targeted therapies may reduce chronic inflammation and supports further investigation of macrophage responses as translational biomarkers of therapeutic activity.

Funding: Funding for this work was provided by NIH NIAID Autoimmune Center of Excellence grant 2UM1AI144292-06, NIH NCATS grant 9R44TR005293-02, and NIH/NIDA grant R01DA060226.

POSTER PRESENTATIONS

SESSION 2 | 38B

ABSTRACT TITLE

TRANSFORMING PATIENT CARE THROUGH PERSONALIZED AI SPEECH RECOGNITION FOR INDIVIDUALS WITH NEUROLOGIC SPEECH IMPAIRMENTS

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ABSTRACT TITLE

TRANSFORMING PATIENT CARE THROUGH PERSONALIZED AI SPEECH RECOGNITION FOR INDIVIDUALS WITH NEUROLOGIC SPEECH IMPAIRMENTS

Introduction: Neurologic speech and motor impairments can often limit communication and independence, while conventional solutions, especially speech recognition systems, often struggle to interpret atypical speech patterns and may lack accessibility for users with physical limitations. We developed ClearSay, a personalized and unique speech-to-text system for a University of Tulsa student with cerebral palsy, spastic quadriplegia, and vocal dysarthria. The project seeks to determine whether individualized speech-model training can improve transcription accuracy and provide a practical tool for independent communication.

Methods: An open-source general speech recognition model was fine-tuned using hours of individualized speech recordings from the participant. The model was trained to recognize his unique speech patterns and was optimized for transcription accuracy. Performance was compared with conventional speech recognition before personalization. The personalized model was subsequently incorporated into an offline interface designed for independent use.

Results: Baseline transcription accuracy of approximately 33% with conventional speech recognition improved to as high as 96% following model personalization. The resulting system provides reliable offline transcription and supports independent written communication. Development is ongoing to adapt the framework for additional users and create an accessible mobile interface incorporating live transcription, accessibility features, and word-processing integration.

Translational Impact: ClearSay demonstrates the potential of personalized artificial intelligence to translate advances in speech recognition into an assistive technology for individuals whose speech is poorly recognized by conventional systems and others. Expanding this approach to additional individuals with dysarthria will help determine its generalizability across neurologic speech impairments and may play a role in physician-to-patient interactions. Continued development may support applications in assistive communication, rehabilitation, education, and independent daily living.

Funding: None

POSTER PRESENTATIONS

SESSION 2 | 39B

ABSTRACT TITLE

MATERNAL PHENYLKETONURIA: INVESTIGATING THE ROLE OF CAPN2 AND ADH1C IN EMBRYONIC TERATOGENESIS

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ABSTRACT TITLE

MATERNAL PHENYLKETONURIA: INVESTIGATING THE ROLE OF CAPN2 AND ADH1C IN EMBRYONIC TERATOGENESIS

Introduction:

Phenylketonuria (PKU) is a genetic disorder in which the body cannot break down the amino acid phenylalanine (Phe), due to a deficiency of the enzyme phenylalanine hydroxylase. Maternal PKU (MPKU), a disease in the fetus caused by elevated levels of Phe in the bloodstream of a mother with PKU, is associated with severe congenital anomalies including cranial defects and cardiovascular malformations. The specific genes affected by high Phe exposure in the developing embryo are currently unknown.

Methods:

Fertilized chicken eggs were treated with Phe through yolk injection, incubated until HH10–12–14, and dissected. Embryonic thoracic/heart tissue was collected for RNA extraction and submitted for RNA sequencing to identify differentially expressed genes. RNA was isolated, reverse transcribed into cDNA, and gene-specific primers were designed for PCR amplification. A T7 RNA polymerase promoter was added to PCR products to generate DIG-labeled RNA probes. In-situ hybridization was performed using T7 RNA polymerase, and probes were detected with an anti-DIG antibody to visualize gene expression in embryonic tissue. Gene expression changes were confirmed by qPCR, and KEGG pathway analysis was used to identify enriched molecular pathways.

Results:

Two pathways, Retinoid Acid (RA) signaling and Focal Adhesion (FA) signaling, were found to be significantly enriched. Literature suggests that RA signaling plays a critical role in heart development, and fluctuating RA levels can cause significant developmental defects. Our study analyzed how phenylalanine exposure affects the gene expression patterns by using in-situ hybridization. We analyzed the differences in expression patterns ADH1C and CAPN2.

Translational Impact:

This data provides a foundation for utilizing an avian model to understand the gene expression effects of MPKU and the involvement of both the Retinoic Acid Pathway and the Focal Adhesion Pathway— establishing the potential for the development of novel MPKU treatments and prevention programs.

Funding: OCU Biology Endowment, OCU Faculty Scholarship Funds

POSTER PRESENTATIONS

SESSION 2 | 41B

ABSTRACT TITLE

**LONGITUDINAL INFANT EEG REVEALS ALTERED CO-ACTIVATION PATTERN DEVELOPMENT
TRAJECTORIES IN INFANTS AT RISK OF CEREBRAL PALSY**

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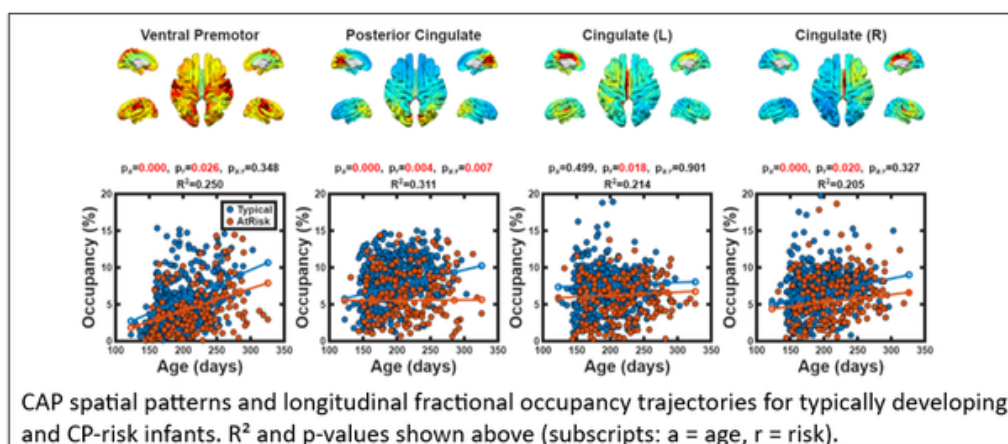
ABSTRACT TITLE

LONGITUDINAL INFANT EEG REVEALS ALTERED CO-ACTIVATION PATTERN DEVELOPMENT TRAJECTORIES IN INFANTS AT RISK OF CEREBRAL PALSY

Introduction. Cerebral palsy (CP) is a group of neurological disorders that affects movement and coordination. Although diagnosis occurs between 1 to 2 years of age, earlier intervention can improve outcomes. Resting brain activity matures rapidly during infancy, and differences in its trajectory have been linked to atypical development. Here, we demonstrate that longitudinal analysis of electroencephalography (EEG) can reveal differences in brain development trajectories during the first year of life between typically developing infants and infants at risk of CP.

Methods. Weekly resting-state EEG recordings from 61 infants were analyzed using co-activation pattern (CAP) analysis. CAP analysis identifies brief, recurring patterns of brain activity and describes the transitions between these patterns. CAP expression was quantified using occupancy, or the percentage of time spent in each pattern. Developmental trajectories were evaluated using linear mixed-effects models.

Results. Fifteen brain-activity patterns were identified, with four showing notable differences in developmental trajectories. These patterns corresponded to regions including the ventral premotor cortex, posterior cingulate cortex, and left and right cingulate cortices. Infants at risk of CP showed lower expression of these patterns compared with typically developing infants. Most patterns showed an increase in expression with age; however, for infants at risk of CP, the posterior cingulate pattern showed no age-related increase.



Translational Impact. Our findings suggest that differences in brain activity during the first year of life may provide measurable indicators of atypical neurodevelopment before CP is formally diagnosed. Longitudinal EEG is a relatively accessible method that could potentially be used to track brain development over time. With further validation, developmental trajectories of brain activity may support earlier identification of infants who could benefit from timely intervention.

Funding. This research was supported in part by NSF EPSCoR RII Track-2 1539068 and NSF NRI 1208639.

POSTER PRESENTATIONS

SESSION 2 | 42B

ABSTRACT TITLE

STOMACH (GASTRIC) CANCER INCIDENCE, MORTALITY, AND SURVIVAL DISPARITIES IN OKLAHOMA COMPARED TO THE UNITED STATES (1999 –2023)

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ABSTRACT TITLE

STOMACH (GASTRIC) CANCER INCIDENCE, MORTALITY, AND SURVIVAL DISPARITIES IN OKLAHOMA COMPARED TO THE UNITED STATES (1999–2023)

Introduction: Stomach cancer remains highly fatal, with an estimated 10,740 U.S. deaths, and *Helicobacter pylori* as the strongest risk factor. In 2023, Oklahoma ranked 38th in incidence and 16th in mortality, yet outcome disparities remain uncharacterized. This study compares Oklahoma's incidence, mortality, stage-at-diagnosis, and survival to national trends to inform prevention and early detection priorities.

Methods: Age-adjusted incidence (1999–2023) and mortality (1999–2021) rates for malignant gastric adenocarcinoma (ICD-O-3 C16.0–C16.9) were obtained from the Oklahoma Central Cancer Registry via OK2SHARE and CDC WONDER. Stage-at-diagnosis and 5-year relative survival data (2019–2023) came from the CDC's NPCR and U.S. Cancer Statistics Data Visualizations Tool. Joinpoint regression estimated the average annual percent change (AAPC), and ArcGIS mapped county-level incidence/mortality distribution. Rates per 100,000 were stratified by sex, age, race/ethnicity, and metropolitan/rural status.

Results: Oklahoma's age-adjusted incidence (5.6 per 100,000) and mortality (2.9 per 100,000) were below national rates (6.8 and 3.4), and incidence was lower than the nation within every racial/ethnic group. Incidence and mortality were highest among non-Hispanic Black, non-Hispanic Asian/Pacific Islander, and lowest among non-Hispanic White populations, and were approximately twice higher in males in both geographies. Both rates increased at ages 45–54 with continued incline through 85+. Incidence and mortality declined in both geographies, though more slowly in Oklahoma (mortality AAPC: –1.03 vs. –2.40 nationally). Localized-stage survival was nearly identical between Oklahoma and the U.S. (74.6% vs. 74.9%), but Oklahoma trailed substantially for regional-stage (29.3% vs. 37.9%) and distant-stage disease (5.3% vs. 9.3%). Rural Oklahoma counties showed slightly lower incidence but higher mortality than metropolitan/micropolitan counties.

Translational Impact: Findings suggest Oklahoma's disadvantage lies not in overall disease burden but in survival following later-stage diagnosis, particularly in rural areas, reflecting gaps in oncology and gastroenterology access. Expanding rural specialty care and continued *H. pylori* testing in higher-incidence populations could target this gap.

POSTER PRESENTATIONS

SESSION 2 | 45B

ABSTRACT TITLE

**STRUCTURAL INSIGHTS INTO A PHEROMONE BINDING PROTEIN TO ADVANCE SUSTAINABLE
PEST MANAGEMENT AND SUPPORT PUBLIC HEALTH**

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ABSTRACT TITLE

STRUCTURAL INSIGHTS INTO A PHEROMONE BINDING PROTEIN TO ADVANCE SUSTAINABLE PEST MANAGEMENT AND SUPPORT PUBLIC HEALTH

Introduction. The Asian corn borer (*Ostrinia furnacalis*) is a major agricultural pest responsible for significant losses in crop production worldwide. Current management strategies rely heavily on broad-spectrum chemical pesticides, contributing to environmental contamination, pesticide resistance, and potential risks to human health from pesticide exposure. Developing species-specific pest control strategies that disrupt insect mating offers a sustainable alternative to chemical pesticides while reducing their impact on human health and the environment. Pheromone Binding Protein 3 (OfurPBP3), which enables male moths to detect female mating pheromones, is a promising molecular target for species-specific pest control.

Methods. Recombinant OfurPBP3 was expressed in *Escherichia coli*, purified, and refolded to obtain functional protein. Ligand-binding activity was evaluated using fluorescence spectroscopy. Circular dichroism spectroscopy was used to assess structural integrity, while high-resolution solution NMR spectroscopy was used to determine the three-dimensional structure and characterize the conformational properties of OfurPBP3.

Results. Recombinant OfurPBP3 was successfully produced as a structurally stable and functional protein. Fluorescence assays confirmed pheromone-binding activity, while circular dichroism demonstrated a predominantly α -helical secondary structure. High-resolution solution NMR revealed a well-defined tertiary fold, providing the first detailed structural characterization of OfurPBP3 and identifying structural features underlying pheromone recognition and transport.

Translational Impact. These findings establish the molecular framework needed for the rational design of pheromone mimetics and other species-specific pest control technologies that disrupt insect mating while minimizing effects on beneficial organisms. By enabling safer alternatives to conventional chemical pesticides, this work has the potential to reduce environmental contamination and human exposure to pesticides, promoting sustainable food production and protecting public health.

Funding: National Science Foundation (NSF)

POSTER PRESENTATIONS

SESSION 2 | 46B

ABSTRACT TITLE

MITOCHONDRIAL TRANSFER FROM PROGENITOR CELLS TO RETINAL ENDOTHELIAL CELLS IN DIABETIC RETINOPATHY

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ABSTRACT TITLE

MITOCHONDRIAL TRANSFER FROM PROGENITOR CELLS TO RETINAL ENDOTHELIAL CELLS IN DIABETIC RETINOPATHY

Introduction: Diabetic retinopathy (DR) is the leading cause of blindness in working-age adults with diabetes and is characterized by retinal vascular injury associated with endothelial cell loss and inadequate vascular repair. Previous studies showed that progenitor cells, such as Myeloid Angiogenic Cells (MACs), also known as CD34+ cells, can support repair of damaged retinal endothelium, but the underlying mechanism remains unclear. We hypothesize that healthy CD34+ donate functional mitochondria to diabetic retinal endothelial cells (RECs), restoring cellular bioenergetics and promoting vascular repair.

Methods: We examined mitochondrial transfer into injured RECs in a proof-of-concept donor-recipient cell model and assessed the respiratory competence of the rare CD34+ donor population. Bovine RECs (BRECs) were treated with TNF α to induce DR related inflammatory stress and then co-cultured with mito-GFP-tagged mesenchymal stem cells (MSCs) as mitochondrial donors. Recipient BRECs were identified by IB4 labelling, and z-stack imaging was used to determine whether GFP-labelled donor mitochondria localized within recipient cells. Because the reparative potential of donor cells may depend on their bioenergetic competence, phase-shift fluorimetry-based nano-respirometry was used to quantify oxygen consumption rate (OCR) in low numbers of isolated peripheral blood-derived CD34+ cells.

Results: GFP-labelled donor mitochondria were detected within IB4-positive, TNF α -treated BRECs by 3D imaging. In parallel, nano-respirometry measured OCRs of 0.094 ± 0.046 and 0.141 ± 0.044 $\mu\text{M O}_2/\text{s}$ from 337 and 1,180 isolated CD34+ cells, respectively (mean $\pm$ SD, n=4). Confidence-band analysis of OCR versus cell number estimated a preliminary detection limit of approximately 520 CD34+ cells, demonstrating the feasibility of respiratory assessment in this rare cell population.

Translational Impact: This work targets mitochondrial dysfunction and impaired vascular repair as upstream mechanisms of DR rather than late-stage symptoms. Nano-respirometry demonstrates the feasibility of measuring cellular respiration in rare cell populations, supporting future regenerative cell-based therapies.

Funding institute: National Eye Institute

Funding number: R01EY034133

ABSTRACT TITLE

MULTI-OMICS MAPPING OF THE HUMAN LIPIDOME IDENTIFIES AN IMMUNOVASCULAR PATHWAY TO CARDIOMETABOLIC DISEASE

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ABSTRACT TITLE

MULTI-OMICS MAPPING OF THE HUMAN LIPIDOME IDENTIFIES AN IMMUNOVASCULAR PATHWAY TO CARDIOMETABOLIC DISEASE

Introduction: Cardiometabolic diseases disproportionately affect diverse populations, yet molecular mechanisms linking lipid metabolism to disease remain poorly defined. Although genome-wide association studies (GWAS) have identified numerous loci for cardiometabolic diseases, most identified variants exert modest effects and offer limited mechanistic insight. Integrating genetic variation with molecular phenotypes can reveal disease-relevant biological pathways. Here, we characterized the genetic architecture of the lipidome in Asian Indians and identified lipid-gene pathways with potential translational relevance to cardiometabolic disease.

Methods: We conducted metabolite GWAS of 516 lipid metabolites in 3,000 Punjabi Sikh individuals, followed by replication in 1.13 million Europeans and 15,000 individuals of Asian Indian ancestry across UK Biobank, GeneRISK, DIAMANT, PROMIS, and other cohorts. We used colocalization, polygenic risk scoring, and Mendelian randomization to prioritize causal lipid-gene relationships. We integrated single-cell transcriptomic and epigenomic datasets to define cell-specific mechanisms.

Results: We identified 609 genome-wide significant SNP-metabolite associations representing 236 SNP-metabolite pairs; 36 signals survived multiple-testing correction, including 33 novel and 3 ancestry-specific associations. LPC O-16:0 showed evidence for a causal association with T2D through PTPRC (CD45), a key regulator of immune-cell receptor signaling. CD45 was expressed in macrophages and lymphocytes in the heart single-cell dataset and in mast cells and leukocytes in the pancreas, linking lipid dysregulation to immune-mediated cardiometabolic injury. CD45 expression was also increased in vascular aging cells, with increased methylation (~30%), suggesting an epigenetically regulated component of vascular immune activation.

Translational Impact: These findings establish a multi-omics framework connecting ancestry-specific lipid variation with immune activation, vascular aging, and cardiometabolic disease. The LPC O-16:0-CD45 axis identified in this study could potentially be an actionable immune-metabolic pathway, highlighting opportunities to design targeted therapeutics for ancestry-informed precision prevention.

Funding

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POSTER PRESENTATIONS

SESSION 2 | 49B

ABSTRACT TITLE

A MULTIPLEX RNA VISUAL COLORIMETRIC SENSOR FOR CANCER BIOMARKER DETECTION

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ABSTRACT TITLE

A MULTIPLEX RNA VISUAL COLORIMETRIC SENSOR FOR CANCER BIOMARKER DETECTION

Cancer diagnosis at early stages is essential for preventing cancer progression and treatment decisions, thereby improving patient survival. Due to the risk of false-positive or false-negative results, relying on a single biomarker is not a smart practice. As a result, cancer diagnostics require a sensor capable of detecting multiple biomarkers to capture molecular malignancies. Colorimetric detection methods offer simple, rapid, and real-time analysis capability, making them suitable for resource-limited settings. However, multiplexing remains a major challenge for colorimetric sensors, such as ELISA-based sensors, which are limited to longer analysis times, lower sensitivity, and increased complexity when multiplexing, in addition to their cost, while the CRISPR-Cas systems face challenges in distinguishing multiple targets due to the non-specificity of collateral cleavage. Here, we introduce a magnetic-particle-based nucleotide multiplex sensor designed for detection of simultaneous cancer-associated RNA biomarkers within a single sample. This approach enables complete analysis within an hour, compared to traditional ELISA methods using collective signal amplification. To enhance specificity and sensitivity for early disease detection, nucleotides were used. The developed biosensor uses streptavidin-modified citrate-functionalized magnetite particles to efficiently capture biotin-functionalized capture RNA sequences and thus increase the analytical sensitivity of the target recognition. Complementary detection RNA sequences were hybridized upon introduction of the respective complementary target sequences. Based on the intensity of the color, biomarker concentrations were quantified. The developed biosensor was capable of simultaneously detecting up to three RNA sequences in undiluted serum over the range of 100 aM to 1 nM, and the observed limit of detection was 100 aM. Moreover, the hybridization mechanism of the multiplex sensor was determined by analyzing the associated binding energies of probes, and the obtained results were in accordance with the sensor's performance. Therefore, this sensor can benefit cancer diagnostics by detecting multiple biomarkers in clinical assay applications.

Funding: National Institute of Biomedical Imaging and Bioengineering

ABSTRACT TITLE

MATERNAL DIETARY EXPOSURES DURING PREGNANCY ARE ASSOCIATED WITH GESTATIONAL AGE ACCELERATION IN UMBILICAL CORD BLOOD AT BIRTH: THE HEALTHY START STUDY

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ABSTRACT TITLE

MATERNAL DIETARY EXPOSURES DURING PREGNANCY ARE ASSOCIATED WITH GESTATIONAL AGE ACCELERATION IN UMBILICAL CORD BLOOD AT BIRTH: THE HEALTHY START STUDY

Objectives

To determine whether maternal diet-related exposures during pregnancy are associated with gestational age acceleration (GAA) in offspring at birth.

Methods

We used data from a subset of 483 mother-infant dyads from the Healthy Start study (births 2010–2014). Umbilical cord blood DNA methylation measured from Illumina 450K array was used to calculate the Knight, Bohlin, and Horvath skin & blood epigenetic clocks. GAA was measured as the residual from regressing the clock-estimated epigenetic age on infant's chronological age. Multiple 24-hour Dietary Recalls (ASA24) and a Food Propensity Questionnaire were completed during pregnancy. Dietary exposures were total and individual components of the healthy eating index 2015 (HEI-2015), dietary inflammatory index, vitamin D intake (mcg), saturated fat (SF) intake (g), monounsaturated fat intake (g), and polyunsaturated fat intake (g). Multivariable linear regressions were used to estimate associations between each diet-related exposure and GAA. The models were adjusted for maternal age, maternal education, race and ethnicity, prepregnancy body mass index, parity, infant sex, smoking during pregnancy, and estimated proportions of 6 cell types in cord blood.

Results

Greens and beans score was negatively associated with Horvath GAA (β : -0.016, 95% CI: -0.029 to -0.003; p : 0.013). Using the skin & blood clock, mean SF intake was positively associated with GAA (β : 0.001, 95% CI: 0.000 to 0.002; p : 0.038), whereas HEI-2015 total vegetable score (β : -0.010, 95% CI: -0.019 to -0.001; p : 0.032) and fatty acid score (β : -0.004, 95% CI: -0.008 to 0.000; p : 0.046) were negatively associated with GAA.

Translational Impact

A healthier dietary pattern, lower saturated fat and higher vegetable intake, was associated with lower GAA at birth. Maternal diet during pregnancy may be a potentially modifiable lifestyle factor. However, it remains unclear whether lower GAA is beneficial for long-term child health.

Funding Sources

1. TSET Health Promotion Research Center (R01CA221819, U54GM104938)
2. Colorado Nutrition and Obesity Research Center (NORC; NIH, P30DK048520)
3. Healthy Start study: (NIH, R01DK076648, R01DK133235, Dabelea)
4. NIH/NCATS Colorado CTSA (UL1TR001082)

POSTER PRESENTATIONS

SESSION 2 | 53B

ABSTRACT TITLE

NOVEL METHOD FOR GENE BIOMARKER IDENTIFICATION FROM SINGLE CELL GENOMICS DATA

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ABSTRACT TITLE

NOVEL METHOD FOR GENE BIOMARKER IDENTIFICATION FROM SINGLE CELL GENOMICS DATA

Introduction: Differential expression (DE) analysis is central to interpreting single-cell RNA sequencing (scRNA-seq) data, as it enables the identification of biomarkers and genes that are over- or under-represented across cell types, states, or biological conditions. Existing methods use log2 fold change (L2FC), which, although effective at identifying genes enriched in a target cluster, can overemphasize genes with low overall expression, causing important highly expressed marker genes to be filtered out before downstream enrichment analysis.

Methods: We propose a novel algorithmic metric, Contrastive Scatter Score (CSS), as an alternative approach for evaluating changes in gene expression across cell populations. Our approach emphasizes genes that are both highly expressed and strongly contrastive between populations, reducing the influence of weakly expressed genes whose apparent specificity may be driven primarily by low expression levels. To improve robustness, we further apply bootstrapping techniques to filter unstable gene rankings. We examine CSS against a widely used method in the field, DESeq2, and evaluate both methods on PBMC and Covid19 datasets.

Results: Our proposed method reliably identifies established marker genes reported by DESeq2, while additionally detecting other biologically relevant genes that were not prioritized by DESeq2. Although CSS and the other examined metrics differed in their highest-ranked genes, high level biological processes-level trends were largely consistent, with standard L2FC showing the greatest divergence from the other approaches. However, each method showed specificity in detailed level molecular functions and pathways.

Translational Impact: CSS could help researchers identify significant genes in single cell genomics data by providing an additional ranking criterion alongside L2FC and statistical significance, especially in cases where genes exhibit both moderate-to-high expression and strong cluster specificity, leading to a more comprehensive identification of biologically meaningful biomarker genes.

Funding: National Science Foundation [NSF-2341725, NSF-2443386], National Institutes of Health [NIH-K25CA270079], University of Oklahoma Big Idea Challenge 2.0 award (BIC2.0), National Institute of General Medical Sciences of the National Institutes of Health [P20GM162339].

POSTER PRESENTATIONS

SESSION 2 | 54B

ABSTRACT TITLE

**PREDICTING GENE MUTATIONS IN COLON CANCER USING LONG-TERM TEMPORAL
DEPENDENCY LEARNING ON A DIRECTED CO-OCCURRENCE ASYMMETRY GRAPH**

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ABSTRACT TITLE

PREDICTING GENE MUTATIONS IN COLON CANCER USING LONG-TERM TEMPORAL DEPENDENCY LEARNING ON A DIRECTED CO-OCCURRENCE ASYMMETRY GRAPH

Introduction: Accurately predicting mutational dependencies in tumor evolution can improve our understanding of cancer progression and support earlier diagnosis and intervention. This is especially important in colorectal tumorigenesis, which often follows stepwise mutational progression patterns.

Methods: We propose a directed co-occurrence asymmetry graph to infer graph-derived mutational paths from 2,344 colon adenocarcinoma samples covering 23,858 mutated genes. We additionally introduce a gene-conditioned shared Long Short-Term Memory (LSTM) model with attention to predict mutation status along inferred paths. The attention mechanism learns to weight informative predecessor mutations directly, while gene embeddings adapt the shared model to each target gene. We compare this model with standard LSTM and dilated Convolutional Neural Network (CNN) architectures, where separate models are trained and used to predict mutations in each gene.

Results: Graph-derived paths substantially improved precision and recall over the reproduced frequency-ordering baseline, with weighted paths performing best overall. The attention-based shared LSTM achieved competitive area under the ROC curve (AUC) and high recall, indicating that attention over predecessor mutations provides a useful representation for target-gene prediction. The standard per-gene LSTM and dilated CNN models also outperformed the reproduced baseline with overall similar metrics, though with lower recall than the shared attention-based LSTM.

Translational Impact: This approach could help researchers and clinicians better understand which mutations are likely to occur next as colorectal cancer (CRC) progresses. It may be useful for identifying CRC progression patterns and pathways and guiding more personalized monitoring of tumor progression, leading to more timely and effective intervention.

Funding: National Science Foundation [NSF-2341725, NSF-2443386], National Institutes of Health [NIH-K25CA270079], University of Oklahoma Big Idea Challenge 2.0 award (BIC2.0), National Institute of General Medical Sciences of the National Institutes of Health [P20GM162339].

ABSTRACT TITLE

DEFINING A SHARED CILIARY PROTEOMIC LANDSCAPE LINKS DIVERSE JOUBERT SYNDROME GENES TO CONVERGENT SIGNALING DYSFUNCTION

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ABSTRACT TITLE

DEFINING A SHARED CILIARY PROTEOMIC LANDSCAPE LINKS DIVERSE JOUBERT SYNDROME GENES TO CONVERGENT SIGNALING DYSFUNCTION

Primary cilia, cellular signaling hubs, maintain a unique protein composition distinct from the cell body, which is essential for receiving and interpreting extracellular cues. Ciliary proteome disruptions likely drive disease as regulators are directly implicated in ciliopathies. Joubert syndrome (JS) is a neurodevelopmental ciliopathy caused by variants in >40 genes, yet no therapeutics are available. While individual JS-gene dysfunction affects specific ciliary protein localization like certain GPCRs, how genetically diverse causes of JS reshape the global ciliary proteome remains unknown.

To address this gap, we developed a protocol to isolate human primary cilia and performed a cross-genetic ciliary proteomics screen across 22 loss-of-function HEK293T cell lines representing 11 JS genes. Employing data-independent differential mass spectrometry analysis on isolated cilia, we generated a dataset of proteins exhibiting altered abundance in JS mutant cilia compared to controls.

Bioinformatics analysis identified statistically significant differentially abundant proteins involved in key biological processes, including cell cycle regulation, protein folding, post-translational modifications, and, most significantly, multiple signaling pathways. Convergence was strongest on Hedgehog and Wnt signaling and novel implicated Ephrin signaling. To determine whether these proteomic alterations reflect functional signaling defects, we generated a mutant library of top candidate proteins altered in ≥ 6 JS mutant cell lines and evaluated the effects of altered candidate abundance on key biological processes, beginning with Hedgehog and Wnt signaling. Functional assays validated our bioinformatic findings: Hedgehog and Wnt signaling were significantly impaired. Our unparalleled cross-genetic proteomic approach defined a shared pathogenic landscape in JS, identified previously unrecognized negative regulators of Hedgehog and Wnt signaling, and revealed Ephrin signaling as a new pathway potentially disrupted in JS.

These findings provide new insight into shared mechanisms underlying JS pathogenesis. Since GPCRs, kinases, and other signaling proteins are already FDA-targetable, uncovering these shared pathway defects may reveal new therapeutic directions for JS.

Funding: NIH NICHD R00HD100554 to JCV

POSTER PRESENTATIONS

SESSION 2 | 57B

ABSTRACT TITLE

**LABEL FREE RAMAN SPECTROSCOPY REVEALS BIOCHEMICAL REMODELING OF OVARIAN
CANCER CELLS FOLLOWING PBMC INTERACTION**

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ABSTRACT TITLE

LABEL FREE RAMAN SPECTROSCOPY REVEALS BIOCHEMICAL REMODELING OF OVARIAN CANCER CELLS FOLLOWING PBMC INTERACTION

Introduction: Tumor-immune interactions can alter cancer-cell behavior, but these changes are typically assessed with targeted assays. Here, it is investigated whether label-free Raman spectroscopy can detect biochemical changes in ovarian cancer cells following 72 h interaction with peripheral blood mononuclear cells (PBMCs).

Methods: Raman spectra were collected from uninteracted, and 72 h PBMC interacted ovarian cancer cells. After preprocessing, spectra were evaluated using principal component analysis (PCA), PCA loadings, and peak-intensity ratios for the tyrosine doublet (I850/I830), nucleic-acid-associated signal relative to phenylalanine (I785/I1003), and phosphate-associated signal relative to phenylalanine (I1095/I1003).

Results: PCA showed partial overlap between conditions, while interacted spectra formed a visibly tighter cluster, showing reduced spectral heterogeneity after PBMC interaction. PC1-PC3 explained 53.92% of normalized spectral variance. Loading profiles localized major contributions to variance near 850–854, 1004, 1300, 1428–1448, 1556, and 1658 cm^{-1} . The median I850/I830 ratio decreased slightly from 1.495 to 1.456, and the I785/I1003 ratio changed from 0.259 to 0.255. The clearest ratio-level change was an increase in I1095/I1003 from 0.349 to 0.389, with the same direction observed across all measured collections. These results suggest that PBMC interaction altered the ovarian cancer cell Raman phenotype, with the strongest consistent biochemical change involving phosphate-associated spectral features.

Translational Impact: This study investigates whether label-free Raman spectroscopy can detect a biochemical response of ovarian cancer cells to immune-cell interaction. Future integration of these Raman phenotypes with functional measurements of proliferation, stress, and treatment response could determine whether tumor-PBMC interactions promote or suppress malignant behavior and support development of label-free phenotyping tools for translational cancer research.

Funding: University of Oklahoma Startup Fund.

POSTER PRESENTATIONS

SESSION 2 | 64B

ABSTRACT TITLE

LLM-GUIDED AUTONOMOUS OPTIMIZATION FOR RADIATION TREATMENT PLANNING

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ABSTRACT TITLE

LLM-GUIDED AUTONOMOUS OPTIMIZATION FOR RADIATION TREATMENT PLANNING

Introduction: Radiation treatment planning requires iterative adjustment of optimization objective weights to balance target coverage and organ-at-risk sparing. This process is time-consuming, depends on planner experience, and may become a key bottleneck for time-sensitive workflows such as real-time adaptive radiation therapy. We investigated whether a large language model (LLM) can autonomously guide optimization-weight adjustment using planning goals and feedback from prior trials.

Methods: Three standardized test cases from AAPM TG Report 119 (C-shaped, head-and-neck, and prostate) were adapted for proton therapy planning and evaluated using an open-source optimization engine. Plans were optimized with a weighted-quadratic objective containing three to four adjustable penalty weights under a hard target-coverage constraint. At each trial, a locally hosted open-weight LLM proposed new weights based on planning goals, prior-trial history, and per-weight summary statistics. Performance was compared with Bayesian optimization (TPE) and random search over five seeds of 30 trials each. For the C-shaped case, the central-core dose limit was progressively tightened to increase optimization difficulty. Plans meeting all predefined goals were considered acceptable.

Results: For head-and-neck, the LLM generated more acceptable plans than TPE and random search (22.2 ± 2.2 vs. 10.2 ± 3.8 and 4.2 ± 2.2). For prostate, it reached the first acceptable plan in fewer trials (4.6 ± 3.0 vs. 15.6 ± 4.9 and 19.0 ± 11.1). Performance was comparable to TPE for the baseline C-shaped case. With tighter constraints, the LLM produced the most acceptable plans at each difficulty level, although all methods degraded.

Translational Impact: LLM-guided optimization may reduce manual trial-and-error while improving consistency and standardization of plan quality. Autonomous background iterations could provide ongoing feedback and support continual refinement from prior planning experience, creating a foundation for more efficient, adaptive, and autonomous radiation treatment-planning workflows.

Table 1. Acceptable plans and iterations to the first acceptable plan (mean $\pm$ SD over five seeds)

CASE	LANGUAGE MODEL		BAYESIAN OPTIMIZATION		RANDOM SEARCH	
	Acceptable plans †	Iterations to first acceptable plan ‡	Acceptable plans †	Iterations to first acceptable plan ‡	Acceptable plans †	Iterations to first acceptable plan ‡
<i>Phantoms</i>						
C-shaped	19.4 ± 11.5	4.8 ± 4.3	19.0 ± 1.7	2.2 ± 0.8	9.8 ± 2.6	3.6 ± 2.1
Head-and-neck	22.2 ± 2.2	2.8 ± 0.8	10.2 ± 3.8	6.2 ± 5.5	4.2 ± 2.2	6.6 ± 3.8
Prostate	15.8 ± 6.4	4.6 ± 3.0	5.2 ± 3.6	15.6 ± 4.9	1.0 ± 1.0	19.0 ± 11.1
<i>C-shaped — central-structure dose limit tightened</i>						
core ≤ 13 Gy	11.4 ± 5.6	4.8 ± 1.9	4.2 ± 2.2	12.6 ± 10.2	2.0 ± 1.7	11.8 ± 10.3
core ≤ 16 Gy	23.4 ± 2.1	2.2 ± 1.1	13.0 ± 2.5	2.8 ± 1.3	7.0 ± 2.4	4.8 ± 3.6
core ≤ 19 Gy	23.4 ± 4.5	3.2 ± 3.3	18.2 ± 2.3	2.2 ± 0.8	9.8 ± 2.6	3.6 ± 2.1
core ≤ 22 Gy	25.8 ± 4.9	1.8 ± 1.1	19.0 ± 1.7	2.2 ± 0.8	9.8 ± 2.6	3.6 ± 2.1

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