



INTERACT

INSTITUTE FOR TRANSLATIONAL AND EMERGING
RESEARCH IN ADVANCED COMPARATIVE THERAPY



4TH ANNUAL
ONE HEALTH AND
ONE MEDICINE SYMPOSIUM

October 22-24, 2025



At the ConocoPhillips OSU Alumni Center

Contents



Map & Parking 3

Welcome Letters 4

Dr. Jim Hess, President, Oklahoma State University
Dr. Jerry Ritchey, Interim Dean, College of Veterinary Medicine
Dr. Jerry Malayer, Interim Director, INTERACT

Guest Speakers 7

Dr. Kelli Almes, Associate Coordinator, USDA-APHIS-NAHLN
Dr. Bassirou Bonfoh, Director, Afrique One-Cote d'Ivoire
Dr. James DeCaprio, Professor of Medicine, Dana-Farber Cancer Institute
Dr. Renukaradhya Gourapura, Professor and Director, Ohio State University
Dr. Vikram Gujar, Assistant Professor, OSU Center for Health Sciences
Dr. Matlock Jeffries, Oklahoma Medical Research Foundation
Dr. Sudhakar Jha, Associate Professor, OSU College of Veterinary Medicine
Dr. Madhavi Kadakia, Professor, Wright State University
Dr. Pankaj Kumar, Associate Professor, University of Virginia
Dr. Chison Mumba, University of Zambia
Dr. Saidu Oseni, Professor, Obafemi Awolowo University, Nigeria
Dr. Joseph Petrosino, Chair/Chief Scientific Innovation Officer, Baylor College of Medicine
Dr. Ashish Ranjan, Professor, Director & Vice Chair, UT Southwestern Medical Center
Dr. Karl Rich, Director, MIAP, OSU Agriculture
Dr. Magda Rich, International and Domestic Outreach Activities, OSU College of Veterinary Medicine
Dr. Amandeep Singh, Ph.D. Student, University of British Columbia
Dr. Patrick Stephens, Associate Professor, OSU College of Veterinary Medicine
Dr. David Ussery, Professor, OSU College of Veterinary Medicine
Dr. Michael Wimberly, Professor, University of Oklahoma
Dr. Aaron Wright, Professor, Baylor University

Agenda 27

Wednesday, October 22
Thursday, October 23
Friday, October 24

Platform Challenge Speakers 30

Kainat Ahmed
Bashar Albik
Niladri Bhowmik
Amal Cheemadan
Tanim Islam
Paige Johnson
Lydia Martins
Saidu Oseni
Anastasilia Oskolkova
Rakshya Shrestha
Amandeep Singh
Jyoti Singh
Cody Whitley

Contents Continued



Poster Challenge Abstracts 41

Sabiha Alam
Debarati Chanda
Mia Dickson
Prince Jhandai
Stephen Kotey
Xueying Liang
Samantha Loving
Anna Claire McMullen
Jeeviya Murugesan
Diani Nanayakkara
Emma Quintana
Bhavanesh Kumar Raju
Madeline Reichert
Ainslie Reynolds
Keerthana Santhosh
Shoroq Shatnawi
Yashaswi Raj Subedi
Akshaya Surendran
Jester Tresner
Vishnu Thayil Valappil
Binta Varghese
Sherry Wang
Emmanuel Yangué
Qianru Zhang
Xiaoming Zhang
Ziyang Zhang

Symposium Committee Members 67

Notes Page 68

Sponsors 69

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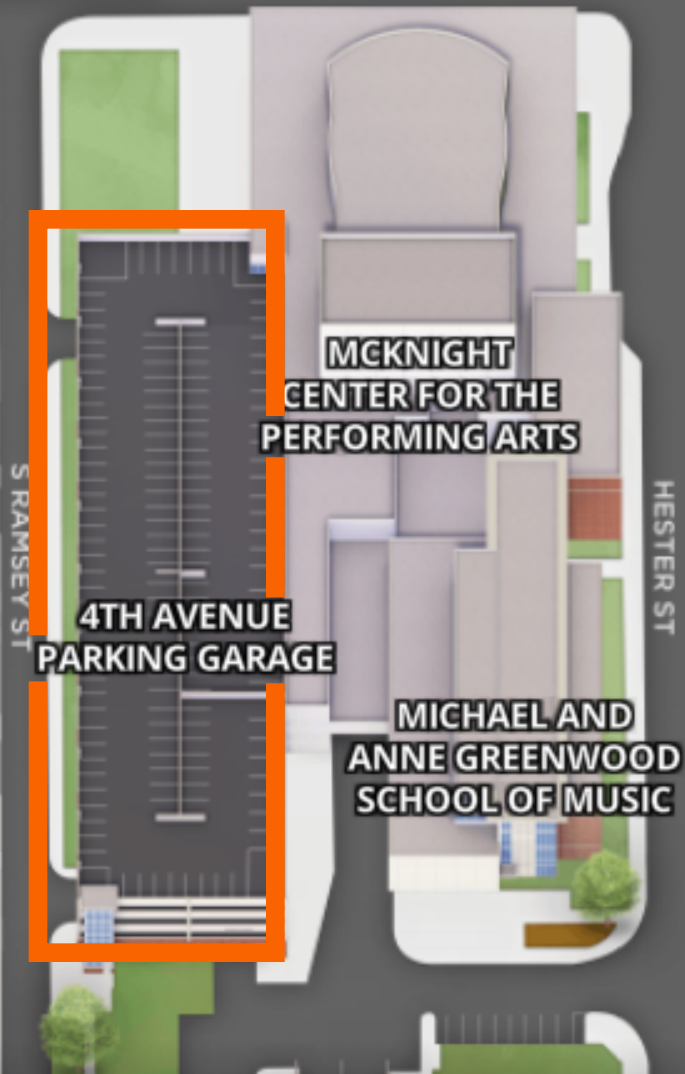
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Parking Available

McKnight Center –
4th Avenue
Parking Garage



A portrait of Dr. Jim Hess, a middle-aged man with grey hair and glasses, smiling. He is wearing a dark sweater over a checkered shirt. The background is a blurred indoor setting with warm lighting. A decorative geometric pattern of dots and lines is overlaid on the image.

DR. JIM HESS

Colleagues and Guests,

At Oklahoma State University, our land-grant mission calls us to serve the people of this state and to make a meaningful impact far beyond it. Right now, research conducted at OSU is making significant advances in human and veterinary medicine. These critical efforts are what it takes to tackle society's most pressing challenges.

OSU takes immense pride in hosting delegates and speakers from several countries on our Stillwater campus who are part of those efforts. We are privileged to learn from their scientific discoveries that have fundamentally reshaped our approach to health and medicine.

INTERACT is a living example of what happens when talented researchers from diverse fields collaborate with a shared purpose. This symposium reflects the best of OSU: curiosity that drives progress, teamwork that sparks innovation and a deep commitment to improving lives through science.

Thank you for being part of this effort and for the work you do every day to advance One Health and One Medicine forward. Congratulations also to our faculty and students for showcasing such meaningful and impactful research in this symposium.

Thank you,

Dr. Jim Hess

President, Oklahoma State University

A portrait of Dr. Jerry Ritchey, a middle-aged man with short brown hair, wearing a dark blue suit, a light blue shirt, and a patterned tie. He is smiling slightly and looking directly at the camera. The background is dark with a faint, light-colored geometric pattern of dots and lines.

DR. JERRY RITCHEY

Welcome to Stillwater and the beautiful campus of Oklahoma State University!

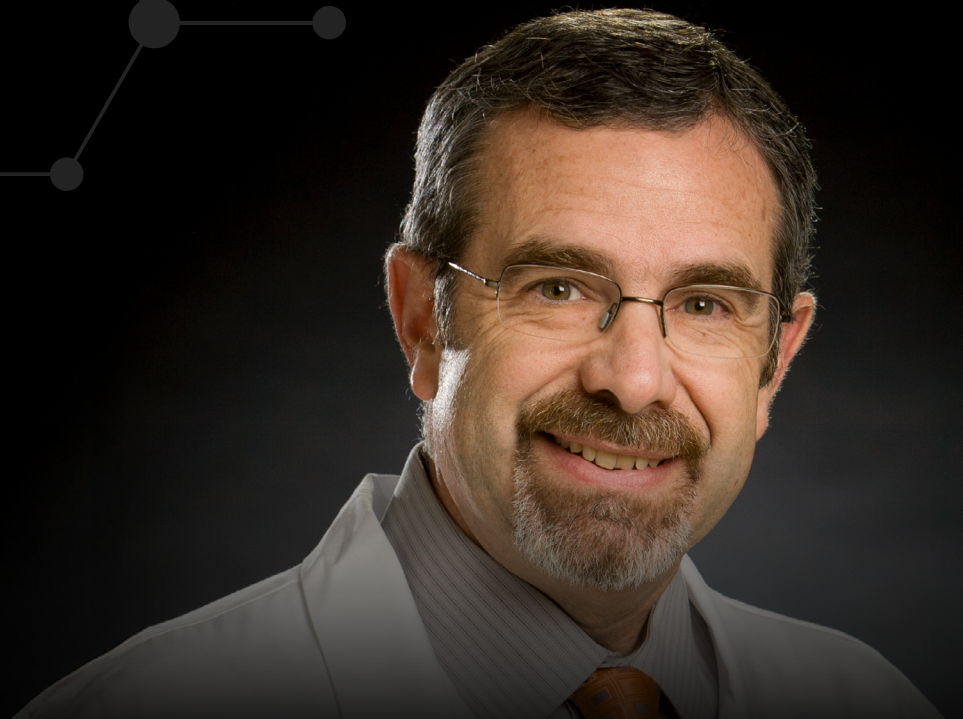
The College of Veterinary Medicine is excited to host the fourth annual INTERACT Research Symposium. This year's theme, "Integrative Approaches to Zoonotic and Environmental Disease Control: Focus on Rural Communities," is timely and fits perfectly into the CVM's, as well as OSU's Center for Health Sciences, mission to support the health and well-being of animals and people in rural communities, particularly those in underserved communities. One Health and One Medicine in action.

The INTERACT organizers have an exciting program to highlight innovative research conducted by rising stars as well as internationally recognized experts. These opportunities are not just important to communicate scientific data, but also provide a platform for networking and collaboration, in social and professional environments, and this is where the real magic happens. We hope you take advantage of every opportunity the INTERACT symposium has to offer.

Thank you to all the faculty, staff and students for your dedication in improving the well-being and health of both people and animals throughout the world.

Sincerely,

Jerry W. Ritchey DVM, Ph.D., DACVP
Professor of Pathology & Interim Dean
College of Veterinary Medicine



DR. JERRY MALAYER

Greetings,

The Institute for Translational and Emerging Research in Advanced Comparative Therapy is a designated university center at OSU. Numerous diseases and health conditions impact both human and veterinary medicine and there is a significant overlap in therapeutic and diagnostic approaches to address these conditions. INTERACT embraces a One Health and One Medicine approach, aiming to expedite the translation of diagnostic and therapeutic modalities for patient use. Since its inception, INTERACT has remained committed to recruiting professionals from diverse disciplines to its platform. We regularly organize networking events that foster collaboration and strengthen OSU's mission of translating research from the bench to the clinic.

This year's symposium highlights the innovative research conducted by our university researchers and features top investigators from around the world. The newly established Oklahoma Center for Microbiome Research is a feature this year, as is the Human Performance and Nutrition Research Institute. The research capabilities at OSU continue to play a crucial role in advancing fundamental and translational medicine in the fields of One Health and One Medicine within Oklahoma and nationwide. Sustainable development is another key feature of our efforts at OSU and will be featured also. This two-day symposium provides an opportunity to learn about the work of our university and hopefully serves as a source of inspiration and motivation for our researchers to initiate collaborative scientific relationships with other researchers worldwide.

I would like to express my thanks to our sponsors whose support has made this symposium a reality. Thank you also to all the speakers who traveled from various parts of the world to participate in this year's symposium. I commend the organizing committee members and our staff for their diligent efforts in organizing this symposium. I look forward to the ongoing interactions among INTERACT members and the development of interdisciplinary programs that align with our mission.

Best Wishes,

Jerry R. Malayer, Ph.D.

Interim Director of INTERACT

Senior Associate Dean for Research & Graduate Education

Professor and McCasland Chair

College of Veterinary Medicine

DR. KELLI ALMES

National Animal Health Laboratory Network H5N1 Outbreak Response

Bio/Research

Dr. Kelli Almes is an associate coordinator for the National Animal Health Laboratory Network, where she co-leads the Exercises and Drills Working Group and helps coordinate national disease outbreak response activities. She plays a key role in strengthening laboratory preparedness by guiding national exercises, webinars, emergency management programs and training of foreign animal disease diagnosticians, while also representing NAHLN in interagency networks such as the Integrated Consortium of Laboratory Networks.

Before joining the U.S. Department of Agriculture, Almes served as an anatomic pathologist and the Foreign Animal Disease and Outbreak section head at Kansas State University's Veterinary Diagnostic Laboratory, focusing on diagnostic service, student education, and research collaboration with African Swine Fever Virus and Highly Pathogenic Porcine Reproductive and Respiratory Syndrome. She is a graduate of KSU, where she earned her Bachelor of Science in Agriculture, Doctorate of Veterinary Medicine, and completed her residency in Anatomic Pathology.



DR. BASSIROU BONFOH

Enhancing One Health Research Capacities to address endemic and emerging health threats

Bio/Research

Dr. Bassirou Bonfoh holds a DVM and a Ph.D. in epidemiology. He worked between 1995-2000 in West Africa for Vétérinaires Sans Frontières. He went in 2000-04 for epidemiology postdoctoral fellowship at the Swiss Federal Institute of Technology Zurich and Swiss TPH. He led between 2004 and 2008, a research group in Africa and Asia to promote the One Health approach in health system with a focus on zoonosis and animal source food safety. Between 2009 and 2018, he was the managing director of CSRS in the area of transdisciplinary research, research capacity building and science partnerships and diplomacy (www.csrs.ch). After his sabbatical in Rakuno Gakuen University, Japan (2018), he has contributed to expand the One Health approach research to non-communicable diseases with a focus on human lifestyle and behavior change. Since 2009 to date, he is the Director of Afrique One (www.afriqueone.org) a leading African research capacity building program in One Health, funded under the African Institutions Initiative (Wellcome) and DELTAS Africa I&II (SFA, WT, UKAID). He is coordinating the African Research Consortium on Health, comprising 14 consortia under the Africa Sciences Foundation initiatives. Bassirou is a guest lecturer on One Health approach at University of Liège (Belgium), University of Oxford, University of Kara and Lomé (Togo), UAC (Benin) and at the veterinary school (Senegal) where he chairs the scientific advisory board. He is also guest scientist and lecturer at Swiss TPH (Switzerland), Rakuno Gakuen University. He is a fellow of the African Academy of Sciences. He advises ECOWAS in the One Health coordination mechanism.

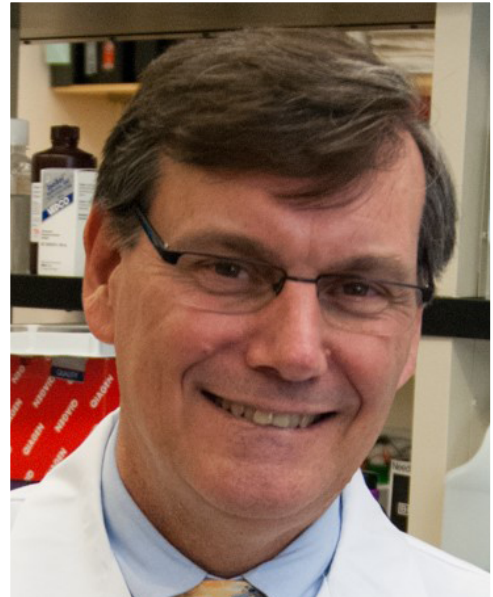


DR. JAMES DECAPRIO

Contribution of Merkel Polyomavirus to Merkel cell Carcinoma

Bio/Research

Dr. James De Caprio is a professor of medicine at Harvard Medical School and Dana-Farber Cancer Institute. He studied medicine at the State University of New York at Buffalo, internal medicine at the University of Chicago, and medical oncology at DFCI. He was a research fellow in the laboratory of David Livingston and began his own laboratory at DFCI in 1992. Currently, he leads a lab with five postdoctoral fellows, five graduate students and four undergraduate students. He serves as the chief, Division of Molecular and Cellular Oncology at DFCI, where he facilitates the research efforts of 30 independent faculty including several members of the National Academies of Medicine and Science.



DeCaprio's research interests include polyomaviruses, cell cycle, RB, DREAM and Merkel cell carcinoma. The DeCaprio laboratory studies the family of polyomaviruses, including SV40 and Merkel cell polyomavirus. The overall strategy has been to identify cellular factors that bind specifically to the viral oncoproteins, determine their normal function, and gain insight into the biologic consequences that occur when they become perturbed by viruses or mutated in cancer. As part of this effort, the lab identified the DREAM complex and determined that it serves as the master coordinator of cell cycle dependent gene expression. More recently, the lab determined that the Merkel cell polyomavirus Small T antigen recruits the MYC paralog MYCL to the Tip60 complex to transactivate genes that contribute to Merkel cell carcinoma. These studies have led to Phase I and 2 clinical trials testing the efficacy of MDM2 inhibitors and degraders in Merkel cell carcinoma. DeCaprio's research is funded in part by an NIH/NCI Outstanding Investigator Award and a newly awarded Program Project grant "Convergent Epigenetic Control of Neuroendocrine Cancers."

DR. RENUKARADHYA GOURAPURA

Nanovaccines for food animals to augment mucosal immunity

Bio/Research

Professor and Director
Center for Food Animal Health, Department of Animal Sciences
Scientific Director, Plant and Animal Agrosecurity Research Facility (Ag-BSL3)
College of Food, Agricultural, and Environmental Sciences
Ohio State University
<https://cfah.osu.edu/people/renukaradhya-gourapura-dvm-phd>



Education:

- DVM and MS — Veterinary College Bangalore, India
- Ph.D. — Indian Institute of Science Bangalore, India
- Postdoctoral training — Indiana University School of Medicine, Indianapolis
- Research funding: Current — 9; Completed — 47

Major Awards and Honors

- 2019 — National Academy of Inventors Fellow
- 2021 — “2021 Innovator of the Year Award” College of Food, Agricultural and Environmental Sciences
- 2021 — “2021 Innovator of the Year, one of the three finalists” for Ohio State University

Invention and Academic Records

- US patents — 8 (5 approved); Licensure of patents: 2
- Advisor for — Ph.D. students: 9 (2 current); MS students: 12 (2 current); Postdoctoral fellows: 4; Visiting Scholars: 10; Undergraduate students: 31
- Journal Articles — Research: 111; Journal Articles — Reviews: 13; Book chapters: 5
- Invited presentations — 69; Conference proceedings/abstracts/presentations: 157

Professional activities

- 2020 President Elect — American Association of Veterinary Immunologists (AAVI)
- Grant reviewer — NIH; USDA; Eight international funding agencies
- Associate Editor — Frontiers in Immunology; Vaccines; Indian Journal of Inflammation Research

Dr. Renukaradhya Gourapura’s laboratory research focus is on mucosal immunology in food animals infected with infectious and zoonotic diseases. Broad areas of research include understanding the host pathogen interactions, microbial pathogenesis, and role of human and animal microbiome in mucosal immune system development and vaccine responses. Currently, mucosal delivery of vaccines is gaining increased attention due to its ability to improve cross-protective immunity against RNA viruses and bacteria whose primary site of infection is at mucosal sites. We are developing innovative biodegradable and biocompatible polymer-based nanoparticle vaccine delivery platforms to target inactivated whole pathogen or subunit antigens to immune cells at mucosal sites, aimed at mitigating the bacterial and viral diseases of food animal species (pig, poultry and cattle). In animals vaccinated through intranasal and oral route, we evaluate the immune correlates of protection and efficacy analysis in vaccine-challenge studies. Our vaccine studies include target pathogens such as porcine reproductive and respiratory syndrome virus, swine influenza virus, highly pathogenic avian influenza virus, SARS-CoV-2, Salmonella, and African swine fever virus. Furthermore, we are involved in developing and evaluating the biomedical large animal models for conducting research on infectious and zoonotic diseases.

DR. VIKRAM GUJAR

Extracellular Vesicles as Mediators of Airway Inflammation: Molecular Insights from Human, Epithelial, and Bacterial Sources



Bio/Research

Dr. Vikram Gujar is an assistant professor of anatomy and cell biology at Oklahoma State University Center for Health Sciences. He earned his Ph.D. in Biomedical Sciences, with a focus on peripheral neuroinflammation and pain, and examined how neuroimmune signaling contributes to inflammatory pain conditions. He completed his postdoctoral training in the Department of Allergy and Immunology at Northwestern University, where he investigated type 2 inflammation and its impact on airway epithelial dysfunction.

At OSU-CHS, Gujar teaches neuroanatomy to medical and graduate students, incorporating human brain dissections, clinical correlations and systems-based instruction. He is also actively involved in course design, curriculum development and coordinating lab-based learning experiences.

In addition to his teaching responsibilities, Gujar mentors high school, undergraduate and graduate trainees, fostering research skills, critical thinking and scientific communication. He serves on multiple institutional committees and contributes to interdisciplinary academic initiatives.

His current research interests include inflammation biology, airway-sensory interactions and the discovery of biomarkers using extracellular vesicles.

Gujar's research focuses on understanding the molecular and cellular mechanisms that link chronic inflammation to sensory dysfunction, with a particular emphasis on the nasal epithelium and olfactory system. His work builds on a foundation in peripheral neuroinflammation and pain, extending into translational models of airway disease.

His current projects focus on the role of extracellular vesicles (EVs) as mediators and biomarkers of epithelial and neuroimmune responses in chronic rhinosinusitis (CRS) and olfactory loss. Using air-liquid interface (ALI) culture systems, patient-derived nasal samples, and transcriptomic analyses, Gujar investigates EV-associated cargo — including mRNAs, proteins and non-coding RNAs — that reflect disease state and therapeutic response.

Additionally, his lab explores host-microbe interactions by characterizing EVs derived from airway bacteria such as *Staphylococcus aureus* and *Cutibacterium acnes*, assessing their immunomodulatory effects on nasal epithelial cells. These studies aim to delineate mechanisms of epithelial barrier disruption and inflammation along the nose-brain axis.

By integrating cell biology, molecular profiling and clinical collaboration, Gujar seeks to develop EV-based, non-invasive diagnostic tools and identify novel targets for intervention in airway inflammatory diseases. His long-term goal is to translate mechanistic findings into precision medicine strategies for inflammatory and sensory disorders.

DR. MATLOCK JEFFRIES

Microbiome Contributions to Osteoarthritis: A Disease that Disproportionately Affects Rural Communities

Bio/Research

Dr. Matlock Jeffries is the director of the Arthritis Research Unit and associate professor in the Arthritis & Clinical Immunology Program at the Oklahoma Medical Research Foundation. He is also a Veterans Affairs clinician and researcher and holds an adjunct appointment at the University of Oklahoma Health Sciences Center, where he teaches medical students, residents and rheumatology fellows, both in the classroom and in the clinic.



A physician scientist, Jeffries' laboratory focuses on the effects of non-genetic factors in knee osteoarthritis, including epigenetics and the microbiome. His basic science work includes the first large-scale description of bacterial DNA within human and mouse joints, the identification of the gut microbiome as a key mediator of differences in mouse OA according to sex and strain. His clinical science work includes the development of the highly accurate peripheral blood biomarker models that can predict future knee OA onset and progression as well as work in clinical trials of novel OA therapies.

He has been granted research funding from the National Institutes of Health, the Department of Defense, the VA and ARPA-H.

Identifying epigenetic changes associated with OA in a variety of tissues: We have previously described differential DNA methylation within OA chondrocytes and subchondral bone in specimens from end-stage OA patients. My laboratory is now focused on determining, using mouse models of OA, which genes and/or gene pathways are epigenetically modified by individual OA risk factors including trauma, advanced age, and obesity induced by high-fat diet. We hope to better define which epigenetic aberrations are potentially "reversible," i.e. associated with diet and obesity, and which are "non-reversible," i.e. associated with aging and prior trauma.

Using epigenetics as clinical biomarkers for OA prediction: We have previously generated large-scale (about 600 patients) whole-genome peripheral blood epigenetic data to derive machine learning models to predict future radiographic, pain, and 'dual' radiographic+pain progression from a single baseline blood draw in knee OA patients within two large U.S.-based OA cohorts. We are now working to determine whether baseline peripheral blood epigenetic changes can also differentiate healthy individuals who will go on to develop radiographic signs of OA from those who will not. We are also working to access the large peripheral blood epigenetic datasets developed by the U.S. Department of Veterans Affairs (the Million Veterans' Program, MVP) to validate and enrich our models.

The microbiome and OA: A distinguishing feature of the two risk factors most strongly associated with OA, aging and obesity, is that they both are associated with changes in the gut microbiome. We are investigating the ways in which these microbiome changes affect OA progression via modulation of the immune system. We also recently published data demonstrating that cartilage healing is partially dependent on the gut microbiome; specifically, that microbiome transplantation from 'superhealer' MRL mice to non-healer B6 mice can transfer a cartilage healing phenotype. We are now investigating the mechanisms underlying this microbiome-mediated protection, as well as determining how the host immune system's response to the microbiome may be modified in OA and how this may be associated with disease phenotypes and progression.

Cartilage microbial DNA and OA: In 2019, we made a pivotal discovery in finding traces of microbial DNA within human and mouse cartilage. Moreover, we found that microbial DNA patterns from human OA patients differed from disease-free human samples, and these differences were mirrored in cartilage from OA-susceptible compared to OA-resistant mice. We are now working to determine the source of this microbial DNA signature as well as investigating its role, if any, in OA pathogenesis.

DR. SUDHAKAR JHA

PONDRing: To Be or Not to Be in Biomolecular Condensates

Bio/Research

Dr. Sudhakar Jha is an associate professor of physiological sciences at Oklahoma State University's College of Veterinary Medicine. With over two decades of experience in cancer biology, Jha's research focuses on chromatin remodeling, epigenetic regulation and the role of nuclear biomolecular condensates in cancer stemness and metastasis. He has made significant contributions to understanding the signaling pathways involved in colorectal cancers and the role of epigenetic modifiers in viral oncogenesis. Jha has led multiple research programs supported by competitive national and international grants. His funding portfolio includes projects from the Ministry of Education Singapore, National Medical Research Council and collaborative translational programs at OSU. Before joining OSU, he served as a principal investigator at the National University of Singapore's Cancer Science Institute. He has authored over 40 peer-reviewed publications and holds several patents on cancer diagnostics and therapeutics. In addition to his research, Jha is an editorial board member of *Frontiers in Cell and Developmental Biology*, *SLAS Technology*, and *Cancers*, and serves as an ad-hoc reviewer for high-impact journals such as *Nature Communications*, *Nucleic Acids Research*, *Cell Reports Medicine*, *Science Advances*, and others.



DR. MADHAVI KADAKIA

TIP60 and DNp63a: A therapeutic axis in targeting cisplatin resistance in Squamous cell carcinoma

Bio/Research

Dr. Madhavi Kadakia is a professor in the Department of Biochemistry and Molecular Biology at Wright State University. She earned her Ph.D. in Infectious Disease and Microbiology from the University of Pittsburgh and completed postdoctoral cancer research at the Pittsburgh Cancer Institute and the Cancer Institute of New Jersey. Joining WSU's BMB department in 2002, she was promoted to associate professor in 2008 and professor in 2013. She was department chair (2015–21), where she launched the undergraduate biochemistry and molecular biology program. She also served as associate dean of research affairs at the Boonshoft School of Medicine (2019–21), leading strategic initiatives to promote cross-disciplinary research, restructure the Clinical Trials Research Alliance, and optimize the Laboratory Animal Center.

As vice provost for research and innovation from July 2021 to June 2025, she made significant contributions to the university's research ecosystem. She strengthened partnerships with Wright-Patterson Air Force Base, the Air Force Research Laboratory, and the Naval Medical Research Unit Dayton, advancing shared infrastructure, collaborative projects and workforce development. She helped establish the a state-of-the-art MRI facility supporting research in cognitive neuroscience, human performance and brain-computer interfaces. She also introduced faculty recognition awards, standardized research policies, a grant development program and the annual Celebration of Research. Under her leadership, research awards increased by 22% in FY24.

Nationally, Kadakia completed the prestigious Hedwig van Ameringen Executive Leadership in Academic Medicine (ELAM) Fellowship in 2019 and serves in multiple leadership roles, including with the AAMC Group on Research Advancement and Development Steering Committee, Dayton Veterans Affairs Research Foundation Board, and the Wright Brothers Institute Board. An internationally recognized cancer scientist with a strong record of federal funding, she has published over 43 peer-reviewed articles, trained numerous trainees, organized international scientific meetings, and served as a reviewer for the National Institutes of Health, Department of Defense and leading journals. She also led WSU's Research Task Force during COVID-19, facilitating the safe return of researchers to campus. She is currently funded through a RO1 grant from NCI/NIH through 2028.

Kadakia's research team investigates molecular mechanisms driving tumor progression and therapeutic resistance in epithelial cancers, with a focus on Squamous cell carcinoma (SCC). Her current work centers on two interconnected themes which includes, TIP60-mediated chemoresistance and Δ Np63 α regulation of Rac1-driven invasion. Her laboratory has shown that TIP60 enhances cisplatin resistance by acetylating Δ Np63 α , altering its transcriptional activity to promote DNA repair and cell survival, particularly through nucleotide excision repair pathways. Ongoing studies aim to define TIP60- Δ Np63 α acetylation sites, identify downstream targets, and evaluate TIP60 inhibition as a therapeutic strategy to restore chemosensitivity. Her team has also demonstrated that Δ Np63 α suppresses tumor cell invasion by inhibiting Rac1, a small GTPase essential for actin cytoskeleton remodeling, via both direct transcriptional repression and suppression of PKC γ /Rac1 signaling, including regulation through miR-320a. Current efforts focus on understanding how loss of Δ Np63 α -mediated Rac1 suppression contributes to epithelial-mesenchymal transition and metastasis, and on assessing Rac1 pathway inhibitors as potential anti-metastatic agents. Collectively, Kadakia's research integrates transcriptional control, signaling networks and non-coding RNA biology to identify actionable molecular targets, with the goal of developing precision oncology strategies to overcome drug resistance and limit cancer invasion and metastasis in SCC.



DR. PANKAJ KUMAR

Extrachromosomal Circular DNA as a Driver of Oncogenic Amplification

Bio/Research



Dr. Pankaj Kumar is an associate professor in the Department of Biochemistry and Molecular Genetics at the University of Virginia and serves as the director of the Data Science/Bioinformatics Core at the UVA School of Medicine. He has a dual background in biology and computer science, with over two decades of experience in computational biology, pan-cancer genomics and microbiome research. Since joining UVA, Kumar has led numerous collaborative projects involving large-scale omics data integration, supporting both basic science and translational research.

His research focuses on understanding disease mechanisms through metanalysis and multi-omics data integration. He has played a key role in building a strong bioinformatics group and in benchmarking computational pipelines for standard bioinformatics analyses. As director of the Data Science Core, Kumar provides strategic leadership for data-driven initiatives and plays a key role in developing scalable, reproducible workflows for high-throughput datasets.

Kumar has authored over 50 peer-reviewed publications, including more than 20 as lead author, in high-impact journals, and contributes extensively to interdisciplinary research collaborations. His work bridges experimental and computational domains to advance precision medicine. He is also committed to mentoring researchers and building data science capacity within the cancer research community.

Kumar applies computational methods to make novel biological discoveries in gene regulation, focusing on the analysis of large-scale genomic and transcriptomic datasets from major consortia such as The Cancer Genome Atlas (TCGA), ENCODE, GTEx and AllofUs. His research integrates diverse high-throughput datasets to uncover previously unrecognized regulatory mechanisms underlying gene expression. His current research interests are in; 1) Studying Extrachromosomal circular DNA and 2) A novel non-coding RNA (transfer RNA fragments).

Using an unbiased computational approach, Kumar led the discovery of transfer RNA fragments (tRFs), a novel class of small regulatory RNAs. He applies a range of techniques — including PAR-CLIP, CLASH, Sylamer analysis, and CDF plotting — to investigate the role of tRFs in post-transcriptional gene regulation and built the first ever relational database of tRFs, tRFdb, available at <http://genome.bioch.virginia.edu/trfdb/>.

He has also made significant contributions to the study of extrachromosomal circular DNA (eccDNA or microDNA), pioneering the use of paired-end sequencing to identify these elements in normal mammalian tissues. Kumar developed Circle_finder algorithm for eccDNA detection, which has been adopted by research labs globally. With over 20 years of experience in genomics and more than 50 publications, Kumar currently leads the Bioinformatics Core at UVA.

DR. CHISON MUMBA

A One Health Perspective on the Control of Anthrax in Game Management Areas of Zambia

Bio/Research



Dr. Chisoni Mumba is an associate professor of animal health economics at the University of Zambia, School of Veterinary Medicine. He holds a Ph.D. in Veterinary Sciences – Animal Health Economics from the Norwegian University of Life Sciences, along with MSc and BVM degrees from UNZA. His academic and administrative career spans over a decade, with extensive experience in teaching, postgraduate supervision and curriculum development. As assistant dean – postgraduate studies (2020–24), he led the transformation of doctoral training at UNZA, including the implementation of a structured, competency-based Ph.D. framework.

Mumba is also an active contributor to academic publishing, serving as chief editor of the Journal of Agriculture and Biomedical Sciences, and as an editorial board member for PLOS ONE and Frontiers in Veterinary Science. He has published more than 60 peer-reviewed papers and mobilized over \$6 million in research funding for the School of Veterinary Medicine.

Beyond academia, he has been instrumental in strengthening Zambia's livestock sector through applied research, policy advocacy, and training of frontline staff. His work focuses on enhancing food safety, animal health systems and household livelihoods. Mumba is also a distance runner and enjoys challenging himself outside the academic sphere.

<https://scholar.google.com/citations?user=dOchJjQAAAAJ&hl=en>

Mumba's research sits at the intersection of animal health, economics and systems thinking, with a strong emphasis on improving livestock-based livelihoods in low and middle-income settings. His work integrates health economics with participatory epidemiology, risk assessment and foresight modeling to address complex challenges such as zoonoses, food safety and antimicrobial resistance (AMR).

He is particularly interested in measuring the societal burden of animal diseases using metrics like Productivity-Adjusted Life Years (PALYs) and the Animal Health Loss Envelope (AHLE) approach. His recent work includes developing models for estimating economic losses from anthrax and AMR in Zambia's livestock sector, aimed at informing national policy and investment planning.

Mumba is also committed to strengthening One Health approaches through interdisciplinary research, stakeholder engagement and capacity-building at the human-animal-environment interface.

He contributes to innovations in veterinary education and digital tools for e-learning. He leads the digitization and indexing of scientific journals through open-access initiatives and supports the development of risk-based meat inspection protocols across Zambia's border posts.

His interdisciplinary approach bridges academia, public health and policy, fostering evidence-based solutions for sustainable animal health systems and resilient food systems.

DR. SAIDU OSENI

Multivariate Analysis of Health and Welfare Indicators of Broiler Chickens in Southwest Nigeria

Bio/Research

Dr. Saidu Oseni is a professor of animal breeding at the Department of Animal Sciences, Obafemi Awolowo University, Nigeria. He received his Ph.D. from the University of Georgia. Oseni has authored numerous publications on animal genetics and health. He is the recipient of numerous international and national grants, and leads efforts in innovation for livestock production and adaptation



DR. JOSEPH PETROSINO

Metagenomics in the Age of Biosurveillance and Precision Medicine

Bio/Research

Dr. Joseph Petrosino is the chief scientific innovation officer at Baylor College of Medicine, where he is also professor and chairman of the Department of Molecular Virology and Microbiology, founding director of the Alkek Center for Metagenomics and Microbiome Research (CMMR) and a member of the Dan L. Duncan Comprehensive Cancer Center.

He received his Ph.D. from Baylor College of Medicine in 1998 and remained at Baylor for postdoctoral training in the Department of Molecular and Human Genetics until 2004, when he joined the faculty of the Department of Molecular Virology and Microbiology.

In 2013, Petrosino founded Diversigen, a start-up where the robust analytic pipelines built in the CMMR have been commercialized to support biotech and pharmaceutical industry needs. He sits on the scientific advisory boards of several biotechnology, pharmaceutical and nutrition companies and has contributed to more than 290 peer-reviewed microbiome studies.

As the BCM CSIO, Petrosino leads the commercialization mission for the institution, directing the strategy for how therapeutic, diagnostic, educational, digital health, device and other BCM intellectual property may be translated from the academic environment to the commercial realm.

Petrosino has over 25 years of experience in microbial genomics and metagenomic sequencing. He was a principal investigator for the NIH Common Fund Human Microbiome Project and established the CMMR in 2011. The CMMR aims to develop advanced technologies to understand the microbiome's impact on health and disease, translating this knowledge into better therapeutics and diagnostics. Over the past 14 years, the CMMR has initiated over 600 microbiome-related projects with more than 300 international collaborators, enhancing the understanding of the microbiome's role in various diseases and developmental processes, including autoimmune diseases, the brain-gut axis and cancer.

Recently, the Petrosino laboratory collaborated with Texas Medical Center laboratories to support a statewide wastewater surveillance program for the Texas Epidemic Public Health Institute. They use viral metagenomic sequencing to assess the viral content of wastewater in over a dozen treatment plants across Texas, predicting disease cases before they appear clinically and supporting public health agencies. Petrosino's contributions to the microbiome field earned him recognition as an American Society for Microbiology Distinguished Lecturer (2012-14) and a BCM Distinguished Alumni in 2024.



DR. ASHISH RANJAN

Bridging Nanomedicine and Focused Ultrasound to Advance One-Medicine Cancer Care

Bio/Research



Dr. Ashish Ranjan is an Endowed Professor and Vice Chair of Comparative Oncology & Research Innovations, and director of the Veterinary Research & Oncology Center within the Department of Radiation Oncology at UT Southwestern Medical Center. Prior to joining UTSW, he held the Kerr Foundation Endowed Chair at the College of Veterinary Medicine, Oklahoma State University, where he also directed the INTERACT program.

Ranjan's laboratory is internationally recognized for advancing device-drug combination strategies in comparative oncology, with translational studies spanning murine models and companion animals with naturally occurring cancers. At UTSW, he is leading the development of a cross-species clinical trial platform that embodies the One Medicine/One Health paradigm.

His research portfolio focuses on integrating focused ultrasound with nanomedicine to reprogram the tumor microenvironment in treatment-resistant cancers such as melanoma, soft tissue sarcoma and metastatic disease. His lab is supported by federal, state and private foundation funding and maintains active collaborations with both academic and industry partners.

A veterinarian by training with deep expertise in translational oncology, Ranjan is committed to bridging animal and human health through innovative cancer therapies. He is the recipient of numerous honors, including the NIH MERIT Award, and serves on advisory boards for several research foundations.

Ranjan's research program focuses on developing therapeutic strategies that integrate physical energy-based modalities with targeted nanoparticles for the treatment of solid tumors. A central theme is the use of focused ultrasound (FUS), both thermal and non-thermal approaches to synergize with immune-modulating and drug-delivering nano- and micro-particles to reprogram the tumor microenvironment.

His laboratory has pioneered the use of calreticulin-loaded nanoparticles and antibody-encapsulated microparticles in combination with FUS to induce immunogenic cell death and stimulate robust adaptive immune responses. These strategies are being evaluated in murine and canine models of melanoma, soft tissue sarcoma, and metastatic disease. A major goal is to overcome immunosuppressive barriers posed by myeloid-derived suppressor cells, aging-associated immune dysfunction and resistance to checkpoint blockade therapies.

By incorporating comparative oncology into the translational pipeline, Ranjan's group leverages naturally occurring cancers in companion animals to validate novel drug-device combinations prior to human clinical trials. His research is supported by the NIH, the Focused Ultrasound Foundation, and other funding agencies. The overarching aim is to accelerate the clinical translation of effective, cross-species combination therapies for patients with advanced disease.

DR. KARL RICH

Participatory Systems Dynamics in One Health

Bio/Research

Dr. Karl Rich is director of International Agricultural Programs for the Ferguson College of Agriculture, director of the Master of International Agriculture Program, and a professor of Agricultural Economics at Oklahoma State University. Rich joined OSU in July 2021 after 16 years of working overseas. He was previously Team Leader at the International Livestock Research Institute's West Africa Regional Office in Dakar, Senegal. At ILRI, Rich led a multidisciplinary team conducting applied research that informed livestock sector strategy and investment options in the developing world. Previous to his work at ILRI, Rich worked at Lincoln University in New Zealand; the Norwegian Institute of International Affairs in Oslo; the South Asia Office of the International Food Policy Research Institute in New Delhi, India; and the U.S. International Trade Commission in Washington, D.C. He also has served as an advisor to the CGIAR COVID-19 Hub; a World Health Organization panel on neglected tropical diseases; and the Scottish Government's Centre for Excellence on Animal Health (EPIC). Rich holds graduate degrees (M.S. and Ph.D.) in agricultural economics from the University of Illinois at Urbana-Champaign and a bachelors (A.B.) degree in economics from the University of California, Berkeley.

While trained as an agricultural economist, Rich's research cuts across disciplinary boundaries, integrating agricultural and livestock policy analysis, global agribusiness issues, animal and aquatic health, and research methods development. He has successfully worked with veterinarians, epidemiologists, animal scientists and crop experts on a variety of applied research topics around the world, providing contextual guidance on socio-economic drivers of behavior and uptake and empirical evidence of benefits and tradeoffs across economic, social and environmental dimensions. Most recently, he has pioneered the use of specialized participatory tools to develop decision-support models with stakeholders to help them co-conceptualize problems and co-create solutions. These tools have been applied in the context of urban agriculture planning in New Zealand; socio-economic drivers of animal disease in Zambia, Timor-Leste, Thailand, Vietnam, Senegal and Tanzania; One Health applications in Côte d'Ivoire; nutritionally sensitive marketing channels for horticulture in South Asia; livestock value chain development in Nicaragua, Myanmar and Ghana; and climate-smart agriculture in Senegal.



DR. MAGDA RICH

Participatory Systems Dynamics in One Health

Bio/Research

Dr. Magda Rich is coordinator of International and Domestic Outreach Activities at the College of Veterinary Medicine at OSU and an adjunct instructor in the Master of International Agriculture Program. She received her Ph.D. in Urban Planning from the University of Brighton. Her research interests focus on care farming and the therapeutic use of natural elements in urban areas, and the use of participatory research methods for community engagement in research projects. She gained professional experience at the Norwegian Institute of International Affairs, Lincoln University in New Zealand, and as director of a small consultancy company based in Europe. In these positions, she worked on a number of multidisciplinary projects in diverse international settings, e.g., India, New Zealand, Zambia and Tanzania, and gained further practical experience through living in multiple countries including New Zealand, Vietnam, and Senegal.

Understanding the value of international experiences prompted her to assume an administrative role to help enable students and faculty with international mobility and gaining international professional experience. Concurrently, she teaches courses on urban agriculture and participatory decision making and continues to investigate and develop new techniques and tools to support participatory research approaches.

Originally an urban planner, Rich has always been interested in the interactions of humans and the environment, both built and natural, and the impact that these interactions have on all aspects of a given system. During her Ph.D. project, in which she investigated the potential of providing care farming opportunities to older residents in dense urban areas, she grew deeply interested in issues related to human health and the environment, while also understanding the importance of community engagement and participation in research.

This experience inspired her future research activities, in which she has been using participatory research methods, particularly group model building discussions, to investigate issues related to urban agriculture, nature conservation and One Health. Her work in participatory research also includes the development of Layerstack, a hands-on, portable participatory research tool that uses layers to capture diverse, geographically grounded data.

Most recently, her research has been focused primarily on One Health, as she has been working on a project that aims to develop interventions to eliminate neglected zoonoses in Tanzania, focusing on brucellosis and bovine TB.



DR. AMANDEEP SINGH

Modeling the Impact of Lumpy Skin Disease in India: Sociobehavioural Analysis, Epidemiology and Economics

Bio/Research

Dr. Amandeep Singh is serving as assistant professor of veterinary extension at one of the state-funded veterinary universities in India. Being a veterinarian with a master's in Veterinary Extension Education, he has also served as a public veterinarian for the government. He has more than six years of work experience as an expert in veterinary sciences and animal husbandry. Moreover, he is a prolific writer with more than 100 publications in the form of research papers, review papers, books and book chapters. He has also been instrumental in the development of resources for the farming community in the form of extension/newspaper articles, leaflets, booklets, manuals, online courses, mobile apps, information systems, websites, and more. He is proficient in multiple languages like English, Hindi, Punjabi, Dogri, and many native dialects, which have enabled him to build a good rapport with the stakeholders involved in the livestock sector. Furthermore, he has completed six projects as principal investigator and four projects as co-principal investigator. Currently, he is on a long-term academic assignment at the University of British Columbia, Canada.



While working on his master's degree, Singh chose organic waste (agricultural waste, dairy waste and household waste) management as a subject of interest because he felt that effective management of organic waste can enhance environmental sustainability and farmers' income. His interest ultimately led him to work on carbon meta-analysis and delve deeply into sustainability. Singh is actively involved in human resource development through need-based training, transfer of livestock-based technologies, and other extramural projects. A significant area of his research is Information & Communication Technologies (ICTs), which include the development of mobile apps, Massive Open Online Courses (MOOCs), websites, information systems, and expert systems, including artificial intelligence, GIS and remote sensing in the livestock sector. He has developed an AI-based smart dairy app to ensure automation and prediction on dairy farms. Other research includes livestock health economics, impact assessment, development of research methods, documentation of Indigenous technical knowledge and biostatistics. He is a strong believer in applied research and aims to translate his research into useful tools for stakeholders and high-impact publications to reach the global scientific community.

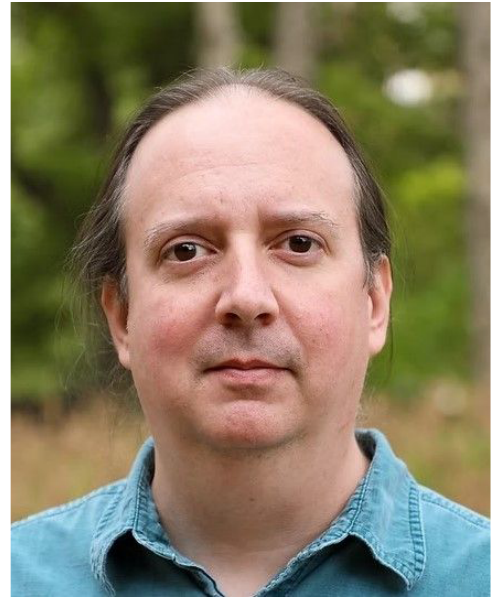
DR. PATRICK STEPHENS

Using machine learning to investigate spillover of ebolaviruses into human populations

Bio/Research

Dr. Patrick Stephens began his Ph.D. training in the Department of Biology at the University of Pittsburgh and the Carnegie Museum of Natural History in Pittsburgh. He completed Ecology and Evolution at Stony Brook University in Stony Brook, New York. He did his first post doc at the National Center for Ecological Analysis and Synthesis in Santa Barbara, California. Prior to coming to Oklahoma State University, he was an associate research scientist in the Odum School of Ecology at the University of Georgia. Three years ago, he came to OSU as part of a cluster hire focused on better understanding the factors that cause infectious disease pandemics. He started in the Department of Biology at the beginning of 2022. Stephens began his career focusing on the evolutionary ecology of emydid turtles, broadened his focus to large-scale patterns of biodiversity and community structure in general, and moved into the area of disease macroecology roughly 10 years ago. Before coming to OSU, he led the Macroecology of Infectious Disease Research coordination network, which produced more than 50 publications exploring various aspects of large-scale disease dynamics during the six years it was funded by the National Science Foundation.

He uses computational methods to explore large-scale disease dynamics and global patterns of biodiversity. His specialties include statistical modelling and machine learning, “big data” and phylogenetic comparative methods. His training is in vertebrate evolutionary ecology and macroecology. He has more than 40 peer-reviewed publications, and his scholarly works have been cited more than 3,000 times since 2020 alone. His two current largest projects are focused on (1) exploring the factors that drive spillover of the pathogens that cause ebolavirus disease from wild animals into human populations in sub-Saharan Africa, and (2) better understanding the factors that drive variation in outcomes for COVID-19 and other diseases here in Oklahoma.



DR. DAVID USSERY

A Genomic Epidemiology Approach to One Health in Oklahoma

Bio/Research



Dr. David Ussery has been working with bioinformatic analysis of bacterial genomes since the first sequence was published in 1995 and in 2024 was recognized as a 'highly ranked scholar' in genomics, for being in the top 0.05% of scholars worldwide.

Prior to joining the Oklahoma State University veterinary medicine physiology department in 2025, Ussery was a professor at the University of Arkansas for Medical Sciences in Little Rock, from 2016 through 2025, where he was the director for the Arkansas Center for Genomic Epidemiology and Medicine (ArC-GEM). Previously, he was at Oak Ridge National Labs, in Oak Ridge, Tennessee (2013-16). He led the Comparative Microbial Genomics group at The Technical University of Denmark from 1997-2014, where he has successfully supervised more than two dozen Ph.D. students in bioinformatics. Ussery received a doctorate in molecular biology in 1993 from the University of Cincinnati College of Medicine and did a postdoctoral fellowship at Oxford University (1992-96). He earned his master's degree in biophysical chemistry at the University of New Mexico in Albuquerque in 1986. He earned a bachelor's degree in chemistry from William Jewell College (Liberty, Missouri) in 1982, and graduated from Springdale High School (Springdale, Arkansas) in 1978.

His research involves using genomics to solve problems. For example, from an environmental sample, a simple question is: Who is there? That is, what viruses, bacteria, archaea, and microbial eukaryotes are present? We are developing methods to answer these questions quickly (in about an hour), and cheaply (a few dollars per sample). A related (and important!) question is whether there's something in the sample that might kill me? (!). It's possible to test for any known toxin family, in less than 30 minutes, for less than a dollar. It is possible to quickly probe blood samples for pathogens as well as for cancer markers.

Genomics has become a 'big data' field of research, with zettabytes of genomic sequences being produced yearly now. This generates a real need for understanding how to use high throughput computing to be able to quickly summarize and tease out important aspects from vast amounts of way too much data! Bioinformatics should be able to obtain simple molecular explanations from massive amounts of information — that is, reduce (not expand!) large data sets. Comparative genomics involves working with experimentalists generating the data, as well as biologists and domain experts who can help organize the information.

DR. MICHAEL WIMBERLY

A One Health Approach for Integrated Disease Surveillance and Forecasting

Bio/Research

Dr. Michael Wimberly is a professor of geography and environmental sustainability and interim director of the Data Institute for Societal Challenges at the University of Oklahoma. He holds a bachelor's degree from the University of Virginia, a master's degree from the University of Washington, and a Ph.D. from Oregon State University. Wimberly has a background in ecology and geospatial analytics and is an expert in using satellite Earth observations to monitor global change and explore environmental impacts on ecosystems and human health. He teaches courses on geographic data science and human-environment interactions and conducts research on a variety of topics including deforestation, wildfires, urbanization, and the ecology of vector-borne and zoonotic diseases. In 2022, he was awarded the American Geophysical Union's GeoHealth Section Award for his research on understanding the influences of weather, climate and land use on vector-borne disease transmission and his work on developing innovative tools for disease forecasting.

Wimberly's research combines satellite Earth observations with ecological and epidemiological models to address scientific questions and develop applications in the fields of public health and natural resource management. Areas of study include wildfire and vegetation dynamics in temperate and tropical forest ecosystems, drivers and consequences of urbanization and agricultural expansion, and the effects of land use and climate on vector-borne disease transmission. Dr. Wimberly has built upon this research to create software tools that apply geospatial data and analytics for decision support. He has developed disease early warning systems to predict West Nile virus outbreaks in the United States and malaria in Ethiopia, generated high-resolution risk maps of urban malaria and dengue in India, used Earth observations to monitor forest degradation and urban growth in West Africa, and designed spatial simulation models to project future landscapes under scenarios of global change.



DR. AARON WRIGHT

Activity-based profiling of health and disease in the gut microbiome

Bio/Research

Dr. Aaron Wright is the James R. Schofield Professor of Biomedical Sciences in the Department of Biology at Baylor University, with a secondary appointment in the Department of Chemistry & Biochemistry. His chemical biology research focuses on providing mechanistic understanding of host-microbiome-environment interactions with high resolution at the level of protein structure and activity. His group works at the interface of chemistry, microbiology and multi-omics to develop and apply new function-driven capabilities to enable discoveries in complex microbial systems. Prior to joining Baylor in 2022, he was the group leader for chemical biology and biological systems sciences at Pacific Northwest National Laboratory and held a joint faculty position at Washington State University from 2008-22. He received his B.S. in Chemistry from George Fox University in 2001, his Ph.D. in Organic Chemistry from the University of Texas at Austin in 2006, and was a postdoctoral fellow in Chemical Biology at the Scripps Research Institute from 2006-2008. He has had numerous grants from NIH, DOE, DOD, NSF and others. In 2024, he was awarded the NIH Director's Transformative Research Award.

Wright's research focuses on investigating health-relevant host-microbiome-environment interactions with high resolution at the level of protein structure and activity. Toward mechanistic understanding of microbiomes, his group has two primary goals: (1) identifying and validating the mechanisms driving microbial functions, interactions and spatial dynamics, and response to perturbation and disease in microbiomes; (2) developing novel phenotype-dependent chemical biology and multi-omics methodologies to enable discoveries in complex microbial systems. With these overarching goals in mind, his group's current research is primarily focused on profiling microbial mechanisms of carbohydrate, mucin, and drug metabolism in the gut related to irritable bowel diseases, with a long-term goal of developing personalized therapies including fecal microbiota transplantation, pathogen killing by targeted bacteriophage, and bioproduct synthesis.



Wednesday, October 22

8:30 a.m. **Bioinformatics workshop** — organized by Dr. Pratul Agarwal

11:30 a.m. **Lunch on your own**

1 p.m. **Opening** — Dr. Jared Taylor
Introduction and Welcome Message from President Jim Hess

AFTERNOON SESSION **Microbiome — Moderated by Dr. Joy Scaria**

1:15 p.m. **Dr. Joseph Petrosino** — Baylor College of Medicine
Metagenomics in the Age of Biosurveillance and Precision Medicine

1:50 p.m. **Tanim Islam** — Oklahoma State University
An Antibiotic-Microbial Metabolite Strategy to Mitigate Resistance in Multidrug-Resistant Gram-Negative Pathogens

2:05 p.m. **Dr. Vikram Gujar** — Oklahoma State University Center for Health Sciences
Extracellular Vesicles as Mediators of Airway Inflammation: Molecular Insights from Human, Epithelial, and Bacterial Sources

2:40 p.m. **General Track**
Niladri Bhowmik — Oklahoma State University
*Secondary Metabolite-Mediated Interactions Between *Achromobacter dolens* and *Pseudomonas aeruginosa* in Cystic Fibrosis Lung Environment*

2:55 p.m. **Dr. Aaron Wright** — Baylor University
Activity-Based Profiling of Health and Disease in the Gut Microbiome

3:30 p.m. **Coffee/Networking Break: 30 minutes**

4:10 p.m. **Dr. Matlock Jeffries** — Oklahoma Medical Research Foundation
Microbiome Contributions to Osteoarthritis: A Disease that Disproportionately Affects Rural Communities

4:45 p.m. **Dr. Amal Cheemadan** — Oklahoma State University
*Proof-of-Concept: Engineered *E. coli* Nissle 1917 to stably express CD27L endolysin enables targeted and sustained treatment of *Clostridioides difficile* infection*

5:30 p.m. **Reception/Networking**
Poster Session 1 — Students present in Legacy Hall during reception

Thursday, October 23

8:15 a.m. **Opening** — Dr. Jared Taylor

MORNING SESSION **Epidemiology/Computational — Moderated by Dr. Samuel Jeyasingh**

8:30 a.m. **Dr. Dave Ussery** — Oklahoma State University
A Genomic Epidemiology Approach to One Health in Oklahoma

9:05 a.m. **Paige Johnson** — Oklahoma State University
TLR4 and Myd88/TRIF Signaling Serve as Novel Immune Sensor and Adapters of SARS-CoV-2-membrane (M) Protein Induced Immune Response

9:20 a.m. **Dr. Patrick Stephens** — Oklahoma State University
Using Machine Learning to Investigate Spillover of Ebolaviruses into Human Populations

9:55 a.m.	Anastasiia Oskolkova — Oklahoma State University <i>Resampling-Empowered Incremental Learning For Cost-Effective Data-Driven Diabetic Retinopathy Prediction</i>
10:10 a.m.	Coffee/Networking Break: 25 minutes Poster Session 2 — Students present in Legacy Hall during break
10:35 a.m.	Dr. Michael Wimberly — University of Oklahoma <i>A One Health Approach for Integrated Disease Surveillance and Forecasting</i>
11:10 a.m.	Dr. Jyoti Singh Saket — DUVASU <i>Nanoparticle Tracking Analysis of Goat mEVs (pre-recorded to be shown during break)</i>
11:25 a.m.	Cody Whitley — Oklahoma State University <i>The anti-viral role of Blocking NOX Signaling During Coronavirus Infections</i>
11:45 a.m.	Lunch/Networking Dr. Dave Usery — Oklahoma State University <i>INTERACT: Past, Present, and Future Directions</i> Poster Session 2 — Students present in Legacy Hall during lunch
1:10 p.m.	Opening — Jared Taylor

AFTERNOON SESSION Cancer — Moderated by Dr. Rudra Channappanavar

1:15 p.m.	Dr. James DeCaprio — Harvard, Dana Farber Cancer Institute <i>Contribution of Merkel Polyomavirus to Merkel Cell Carcinoma</i>
1:50 p.m.	Kainat Ahmed — Oklahoma State University <i>Decoding the circRNA-miRNA-mRNA Regulatory Network in Hepatitis B Virus-driven Hepatocellular Carcinoma</i>
2:05 p.m.	Dr. Madhavi Kadakia — Wright State University <i>TIP60 and DNP63a: A therapeutic axis in targeting cisplatin resistance in Squamous cell carcinoma</i>
2:40 p.m.	Dr. Pankaj Kumar — Virginia Tech University <i>Extrachromosomal Circular DNA as a Driver of Oncogenic Amplification</i>
3:15 p.m.	Coffee/Networking Break: 30 minutes Poster Session 2 — Students present in Legacy Hall during break
3:45 p.m.	Dr. Ashish Ranjan — UT Southwestern Medical Center <i>Bridging Nanomedicine and Focused Ultrasound to Advance One-Medicine Cancer Care</i>
4:20 p.m.	Bashar Albik — Oklahoma State University <i>Targeting the Unfolded Protein Response: A Novel Therapeutic Strategy for Ulcerative Colitis in Preclinical Rat Models</i>
4:35 p.m.	Dr. Sudhakar Jha — Oklahoma State University <i>PONDRing: To Be or Not to Be in Biomolecular Condensates</i>
5:10 p.m.	Rakshaya Shrestha — Oklahoma State University <i>Differential TLR-ERK1/2 activity promotes viral ssRNA and dsRNA mimic-induced dysregulated immunity in macrophages.</i>
5:25 p.m.	Reception/Networking Poster Session 3 — Students present in Legacy Hall during reception

6 p.m. **Dinner**
Poster Session 3 — *Students present in Legacy Hall after dinner*
Dr. Karl Rich — Oklahoma State University
An Economist's Journey Through the World of One Health

Friday, October 24

8:15 a.m. **Opening** — Dr. Jared Taylor

SESSION **Zoonotic Disease — Moderated by Dr. Xufang Deng**

8:30 a.m. **Dr. Bassirou Bonfoh** — Director of Afrique One | Remote/Zoom
Enhancing One Health Research Capacities to address endemic and emerging health threats

9:05 a.m. **Dr. Amandeep Singh** — GADVASU
Modelling the Impact of Lumpy Skin Disease in India: Sociobehavioural Analysis, Epidemiology and Economics

9:20 a.m. **Dr. Kelli Almes** — US Department of Agriculture-APHIS
National Animal Health Laboratory Network H5N1 Outbreak Response

9:55 a.m. **Drs. Karl and Magda Rich** — Oklahoma State University
Use of specialized participatory tools to develop systems models and impact assessments with stakeholders to help them co-conceptualize problems and co-create solutions

10:30 a.m. **Dr. Saidu Oseni** — OAU Nigeria
Multivariate Analysis of Health and Welfare Indicators of Broiler Chickens in Southwest Nigeria (pre-recorded to be shown during break)

10:45 a.m. **Lydia Martins** — Oklahoma State University
Entrepreneurship And Innovation In Veterinary Telemedicine: Tackling Industry Challenges And Enhancing Producer-Veterinarian Connectivity

11:15 a.m. **Dr. Renukaradhya Gourapura** — Ohio State University
Nanovaccines for Food Animals to Augment Mucosal Immunity

11:50 a.m. **Dr. Chison Mumba** — University of Zambia
A One Health Perspective on the Control of Anthrax in Game Management Areas of Zambia

Noon **Recognition of Speakers-Poster and Platform Challenge Award**

KAINAT AHMED

Decoding the circRNA-miRNA-mRNA Regulatory Network in Hepatitis B Virus-driven Hepatocellular Carcinoma

Day 2 - Oral Presentation



Background

Hepatocellular carcinoma (HCC) accounts for 90% of primary liver cancer, with approximately 70% of HCC induced by chronic hepatitis involving the pathogenic factor Hepatitis B Virus (HBV). Integration of the HBV genome in the host chromosome poses a significant problem in HBV-HCC patients because of its asymptomatic behavior and difficulty in early diagnosis. CircRNAs are highly stable non-coding RNAs that form a closed loop by a unique back-splicing process. It is known for its oncogenic and biomarker potential in various cancers, including HBV-HCC, by primarily trapping tumor suppressive regulatory factors such as miRNAs, which, when free, can silence expression of oncogenic mRNAs. However, there is currently no knowledge about HBV integration associated with upregulated circRNA functioning in the circRNA-miRNA-mRNA axis in HBV-HCC. Therefore, the objective of this study is to provide new insight into the molecular mechanism underlying HBV-HCC development and identify precise diagnostic markers and therapeutic targets.

Methods

Firstly, we isolated RNA from HBV-negative HEPG2 and HBV-integrated HEP3B cells using the Trizol method and subjected the RNA to high-throughput RNA-seq with DESeq bioinformatic analysis to profile dysregulated host circRNAs and mRNAs in HBV-positive cells. We performed Sanger sequencing and RT-qPCR on 10 circRNAs with high fold-change and padj values and predicted the miRNA partners using mirDB and Targetscan bioinformatic analysis. We further validated the expression of miRNA from the GEO database (GSM1780817: HEP3B and GSE176602: HEPG2) and generated ECDF plots to check whether the sequestration of circRNA by miRNA results in a fold-change of the target mRNA. The mRNA targets of the miRNAs were then subjected to Gene Set Enrichment Analysis (GSEA) using the Molecular Signature Database (MSigDB) to identify dysregulated oncogenic pathways.

Results

We identified 218 upregulated circRNAs in HEP3B cells. The RT-qPCR results showed the highest expression of circADGRL2 (~25-fold) and circPALS2 (~11-fold) in HEP3B cells. Furthermore, miRNA-429 was significantly downregulated in HEP3B, as indicated by an ECDF plot showing a high log2 fold-change for its target mRNAs, specifically for circADGRL2 and circPALS2. Lastly, genes associated with KRAS, the liver development pathway, and hepatoblastoma were significantly upregulated in HEP3B cells.

Conclusions

Our data suggest that circADGRL2 and circPALS2 may bind to miRNA-429, resulting in the upregulation of oncogenic pathways. miRNA-429 has been shown to repress cell proliferation in HBV-HCC; however, the mechanism behind its downregulation remains unknown. Additionally, the high expression of these circRNAs makes them ideal for use as diagnostic biomarkers by further evaluating their presence in bodily fluids.

BASHAR ALBIK

Targeting the Unfolded Protein Response: A Novel Therapeutic Strategy for Ulcerative Colitis in Preclinical Rat Models

Day 2 - Oral Presentation



Background

An estimated 3.1 million adults (1.3%) in the United States have been diagnosed with inflammatory bowel disease (IBD), including Crohn's disease and ulcerative colitis, indicating chronic inflammation of the gastrointestinal tract. Its manifestation in adolescents is particularly concerning, leading to severe gastrointestinal distress and diminished quality of life. Current IBD management interventions are not efficient, necessitating the identification of novel targets for early detection and intervention. Emerging evidence highlights the role of the Unfolded Protein Response (UPR) in IBD pathogenesis, with particular emphasis on the regulation of key UPR components such as Activating Transcription Factor 6 (ATF-6) and X-box Binding Protein 1 (XBP1) among others. Our research aims to understand the molecular mechanisms of ATF-6 and XBP1 expression in the UPR and explore novel therapeutic strategies targeting these pathways. By addressing the epigenetic modulation of UPR, we aim to develop targeted interventions to mitigate ER stress and inflammation in the early stages of IBD, preventing the onset of chronic disease.

Methods

Colon inflammation was induced with 2, 4, 6 trinitrobenzenesulfonic acid (TNBS) in Sprague-Dawley WT and GlS+/- rats. Animals were pretreated with UPR inhibitors, Tauroursodeoxycholic acid (TUDCA) and 4-phenylbutyric acid (PBA). Following 24 hours post-inflammation, rats were sacrificed, and colon tissues were collected for molecular studies using DNA, RNA, and proteins.

Results

TNBS-induced colon inflammation increased proinflammatory mediators like IL-6 and TNF α more in WT compared to Het animals. TNBS-induced colon inflammation resulted in UPR activation, indicated by increased splicing and expression of XBP1 and ATF6 in WT animals compared to Het animals. Colon inflammation decreased DDIT3 expression, resulting in reduced apoptosis.

Conclusions

TNBS-induced colon inflammation resulted in UPR activation, especially in pathways essential to maintaining homeostasis but not apoptosis. This study highlights the potential therapeutic ability of UPR inhibitors in alleviating ER stress and inflammation, offering new avenues for IBD management, preventing chronic disease, and improving patient quality of life.

NILADRI BHOWMIK

Secondary Metabolite-Mediated Interactions Between *Achromobacter dolens* and *Pseudomonas aeruginosa* in Cystic Fibrosis Lung Environment

Day 1 - Oral Presentation



Background

Cystic fibrosis (CF) is a life-shortening genetic disorder caused by mutations in the cystic fibrosis transmembrane conductance regulator gene. As a result, the airways of people with CF accumulate a thick and sticky mucus layer. This mucus acts as a substrate for the growth of a polymicrobial community comprising opportunistic pathogens like *Pseudomonas aeruginosa*, and other commensal oral and respiratory bacteria. *Achromobacter* spp. are Gram-negative bacteria commonly found in soil but are increasingly isolated from older people with CF. Co-colonization by *Achromobacter* spp. and *P. aeruginosa* has been associated with detrimental effects on lung function in people with CF, yet the mechanism underlying these interactions are underexplored. Emerging evidence suggests that such interactions may be mediated by microbial secondary metabolites (SMs). Notably, *Achromobacter* spp. harbor multiple biosynthetic gene clusters (BGCs) that encode SMs, the majority of which remain uncharacterized.

Methods

In this project, we are investigating interactions between clinically isolated *Achromobacter* spp. and *P. aeruginosa*. We hypothesize that these two bacteria use SMs to engage in competition for resources. We isolated *Achromobacter dolens* by screening about 100 CF clinical samples on selective media and then performed co-culture assays with *P. aeruginosa*. These assays were carried out in synthetic CF medium (SCFM) to closely mimic the CF lung environment.

Results

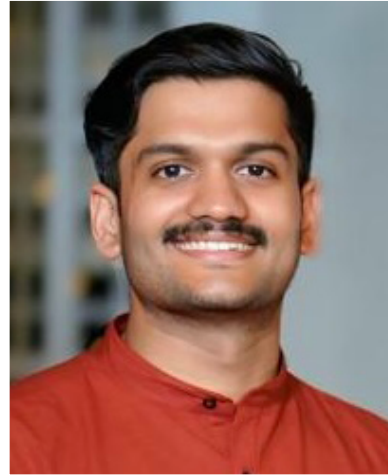
We observed that the presence of *A. dolens* induced pyoverdine production in *P. aeruginosa*. Pyoverdine is an iron-chelating siderophore released by pseudomonads under conditions of iron limitation. To further investigate, we sequenced the genome of *A. dolens* and identified multiple BGCs, including a siderophore BGC with high sequence similarity to alc cluster from *Bordetella* spp that encodes the production of alcaligin. Alcaligin is a siderophore that plays a central role in iron acquisition in *Bordetella bronchiseptica* and *Bordetella pertussis*.

Conclusions

Our results suggest that *A. dolens* may release their own siderophores that interfere with iron acquisition by *P. aeruginosa*, thereby enhancing pyoverdine production. Since pyoverdine is a well characterized virulence factor, this interaction may underlie the detrimental impact on lung health. While it is commonly believed that competition between bacteria during co-infection reduces virulence, our findings challenge this notion. Our future studies will involve the characterization of these bioactive SMs produced by *A. dolens* that induce pyoverdine production in *P. aeruginosa* using High-Performance Liquid Chromatography-Mass Spectrometry (HPLC-MS) based approaches. Furthermore, we will employ a *Galleria mellonella* infection model to determine co-infection virulence by *A. dolens* and *P. aeruginosa* in vivo.

AMAL CHEEMADAN

Proof-of-Concept: Engineered E. coli Nissle 1917 to stably express CD27L endolysin enables targeted and sustained treatment of Clostridioides difficile infection.



Day 1 - Oral Presentation

Background

Bacterial infections are becoming harder to treat due to rising antimicrobial resistance (AMR), which reduces the effectiveness of conventional antibiotics. Bacteriophages offers a potential alternative but delivering the complete phage as a therapeutic is challenging. Bioengineered probiotics expressing phage endolysin genes present a promising solution. As a proof of concept, we engineered E. coli Nissle 1917 to stably express CD27L endolysin with activity against broad range of C. difficile serotypes, enabling targeted C. difficile infection (CDI) control without antibiotics.

Methods

We selected Escherichia coli Nissle 1917 (EcN) as a probiotic chassis and the delivery system due to a unique combination of safety, colonization potential, and therapeutic versatility. Native plasmids were cured using CRISPR-Cas9 to allow stable integration of a synthetic pMUT2 plasmid carrying the optimized cd27l gene. After overexpression in BL21(DE3) using pET28a, the gene was cloned into pMUT2 under FNRS and J23101 promoters via Gibson assembly. Engineered EcN strains were validated with GFP and tested against Clostridioides difficile using 1:1 and 4:1 co-culture assays and spent media. Antimicrobial efficacy was evaluated by quantifying C. difficile colony-forming units (CFU).

Results

CD27L, a phage-derived endolysin, showed strong lytic activity against 30 Clostridioides difficile strains, including ribotype 027, without affecting commensal bacteria. After CRISPR-Cas9-mediated curing of native plasmids, a synthetic pMUT2 plasmid encoding CD27L was introduced under either constitutive (J23101) or anaerobic (FNRS) promoters. GFP confirmed expression and plasmid stability without antibiotics. In 1:1 co-culture assays, J23_CD27L_EcN significantly reduced C. difficile R20291 viability ($p < 0.01$), whereas FNRS_CD27L_EcN showed no significant effect. No significant reduction was observed at a 4:1 ratio. Spent media from J23_CD27L_EcN also significantly inhibited C. difficile growth ($p < 0.05$), suggesting extracellular CD27L activity.

Conclusions

This proof-of-concept study establishes engineered Escherichia coli Nissle 1917 (EcN) as a versatile and platform for delivering targeted antimicrobial proteins. Stable expression of the CD27L endolysin enabled effective inhibition of C. difficile without antibiotic selection, demonstrating therapeutic potential. These findings support the broader use of engineered EcN as a modular chassis for expressing diverse genes to develop precision bacteriotherapies against multidrug-resistant pathogens

TANIM ISLAM

An Antibiotic-Microbial Metabolite Strategy to Mitigate Resistance in Multidrug-Resistant Gram-Negative Pathogens

Day 1 - Oral Presentation



Background

The global rise of antibiotic resistance poses a critical threat to public health, necessitating the exploration of alternative antimicrobial strategies. Phenolic acids (PAs), naturally occurring compounds with known health benefits, have demonstrated promising antibacterial properties. This study investigates the broad-spectrum antibacterial activity of PAs as a potential alternative to last resort antibiotics.

Methods

We tested antimicrobial activity of 3,4-dihydrobenzoic acid (DHBA), 2,4,6-trihydroxybenzoic acid (THBA) and 3,4-Dihydroxyphenylacetic acid (DOPAC) against a panel of 38 pathogens with different resistant profiles. The MiniBioReactor model was run for 14 days to evaluate the impact of DHBA and colistin on gut microbial dysbiosis. To reveal the mode of action, we performed outer membrane permeability assay and membrane integrity assay. Specific genes or pathways targeted by DHBA were elucidated using a high-throughput antimicrobial susceptibility test of single-gene deletion *Escherichia coli* library. Checkerboard assay determined the efficacy of these compounds in combination with several classes of antibiotics. The potentiation effect of DHBA on antibiotics was further validated using in vivo *Galleria mellonella* infection model and an ex vivo wound infection model.

Results

Minimum inhibitory concentrations (MICs) for the tested PAs ranged from 3000–4000 $\mu\text{g/mL}$, with DHBA exhibiting the most potent activity. A 14-day study with a MiniBioReactor model revealed that DHBA induced significantly less gut microbiota disruption compared to colistin. Mechanistic studies showed that DHBA compromises the bacterial outer membrane, targeting the cell envelope in *Escherichia coli*. High-throughput screening of an *E. coli* single-gene deletion library identified 14 primary and 14 secondary gene targets associated with envelope integrity. In contrast, colistin resistance was linked to genes involved in DNA replication and repair. Sub-inhibitory concentrations of DHBA impaired swimming motility of *E. coli* and enhanced the susceptibility of *E. coli* and *Pseudomonas aeruginosa* to colistin and cefepime, respectively. These effects were validated in both an in vivo *Galleria mellonella* infection model and an ex vivo wound infection model, where DHBA-antibiotic combinations significantly reduced bacterial burden and improved survival outcomes.

Conclusions

Phenolic acids, particularly DHBA, exhibit broad-spectrum antibacterial activity with minimal disruption to microbial communities. By targeting the bacterial cell envelope and potentiating conventional antibiotics, DHBA represents a promising adjunct therapy to combat multidrug-resistant infections and mitigate the spread of resistance.

PAIGE JOHNSON

TLR4 and Myd88/TRIF signaling serve as novel immune sensor and adapters of SARS-CoV-2-membrane (M) protein induced immune response.

Day 2 - Oral Presentation



Background

Severe COVID-19 is associated with excessive lung inflammation and impaired antiviral response. This process is often driven by a “cytokine storm,” in which viral pathogen-associated molecular patterns (PAMPs) trigger excessive production of pro-inflammatory cytokines and chemokines. SARS-CoV-2 structural spike (S), nucleocapsid (N), and envelope (E) proteins are key mediators of excessive inflammation. The immune sensors and signaling pathways for S-, N-, and E-proteins are well described. However, the role of M-protein in SARS-CoV-2-induced immunity, and the immune sensor and downstream pathways promoting M protein-induced immunity are not well characterized.

Methods

Here, we stimulated murine bone marrow derived macrophages (BMDMs) with M-protein and examined the host response, identified the immune sensor, and defined key adapter molecules that facilitated excessive inflammation and impaired antiviral response. Western blot and Coomassie staining confirmed the identity and purity of the recombinant M protein. Cytokines and chemokines were measured with sandwich ELISA, multiplex assay, and flow cytometry with intracellular staining. Activation of downstream pathways was assessed with Western blot. We also treated mice intranasally (i.n.) with SARS-CoV-2 M-protein to investigate whether M-protein alone had direct pro-inflammatory effects in-vivo and determine whether lung inflammation post-treatment was rapidly induced or delayed using RT-qPCR and histopathology scoring.

Results

We show that M-protein elicits a robust inflammation and suppresses antiviral immunity in-vitro. Using wild-type and knock-out BMDMs, we identified TLR4 as the primary immune sensor, while Myd88 and TRIF facilitated M-protein-induced inflammation via both NFkB and MAPK pathway activation. We also show that while TLR4 is the primary immune sensor, the importance of MyD88, TRIF, and, to a lesser extent, TLR2 in the M protein-induced pro-inflammatory response are cytokine and chemokine-dependent. Interestingly, M-protein suppressed antiviral interferon (IFN) response via ERK1/2 activation, while blocking M-protein-induced ERK1/2 activity reduced inflammation while improving IFN-I response. Mice treated i.n. with M-protein and collected at 12hpt showed inflammatory gene expression. This gene expression was not seen in mice treated a second time at 48hr and collected at 96hpt, indicating M-induced inflammation occurred rapidly (<12hpt) after administration.

Conclusions

Collectively, we defined the role of M-protein in SARS-CoV-2-induced inflammation, both in-vitro and in-vivo, and identified TLR4 and Myd88/TRIF as key molecules orchestrating dysregulated immunity largely via the ERK1/2 pathway; specifically targeting these pathways may mitigate SARS-CoV-2-mediated dysregulated immunity and severe disease.

LYDIA MARTINS

Entrepreneurship and Innovation in Veterinary Telemedicine: Tackling Industry Challenges and Enhancing Producer-Veterinarian Connectivity

Day 3 - Oral Presentation



Background

Access to timely and affordable veterinary care is a persistent challenge in rural areas, where shortages of large-animal veterinarians, long travel distances, and high costs limit service delivery. These gaps impact animal health, producer livelihoods, and the broader food system. As veterinary shortages grow, especially in production animal practice, digital innovation has emerged as a potential solution.

Methods

This study evaluates the feasibility and strategic potential of AgriCare, a proposed mobile telemedicine platform designed to improve communication between livestock producers and veterinarians. A qualitative, stakeholder-driven approach was used, including 25 semi-structured interviews with U.S. and international livestock producers, veterinarians, and animal health professionals. Interview data were analyzed thematically and interpreted using business frameworks including Lean Canvas and Blue Ocean Strategy to assess product-market fit and innovation viability.

Results

Five key themes emerged: (1) logistical access barriers; (2) limited engagement due to high thresholds for veterinary care; (3) erosion of continuity and trust; (4) veterinary workforce constraints; and (5) rising openness to digital tools, particularly among younger, tech-savvy producers. While regulatory structures in the U.S. limit telemedicine's clinical use without an established veterinarian-client-patient relationship (VCPR), participants expressed interest in asynchronous communication, decision-support tools, and digital record keeping. Internationally, more flexible tele-health regulations and unmet veterinary demand create clearer pathways for implementation.

Conclusions

AgriCare presents a promising innovation for addressing structural challenges in rural veterinary care, though its viability depends on regulatory navigation and user-centered design. In the short term, U.S. applications may focus on continuing professional development (CPD) and decision-support features, while international markets offer greater flexibility for clinical deployment. This study demonstrates how stakeholder insight and strategic business modeling can guide veterinary entrepreneurship and create new access points for animal health services in underserved areas.

ANASTASIIA OSKOLKOVA

Resampling-Empowered Incremental Learning For Cost-Effective Data-Driven Diabetic Retinopathy Prediction

Day 2 - Oral Presentation



Background

Diabetic retinopathy (DR) is a serious complication of diabetes that can lead to vision impairment or even blindness if not detected and treated in the early stage. Early detection is critical to prevent irreversible vision loss, yet individuals in rural and resource-limited settings face significant barriers to routine eye screenings and follow-up care. Leveraging electronic health records (EHR) data, machine learning-based DR prediction has emerged as a promising direction for timely detection. However, the periodic growth of EHR databases necessitates an efficient approach to update prediction models without incurring high retraining costs.

Methods

This study proposes establishing an effective incremental learning (IL) framework that enables the machine learning-based DR prediction model to continuously learn from new data while retaining knowledge from previous observations. To address the challenges of imbalanced data in EHR and mitigate catastrophic forgetting — a phenomenon where models lose previously learned knowledge when updated with new data — a novel sampling method called Batch Balanced Weighted Sampling (BBWS) is proposed. BBWS ensures that the training data is balanced while preserving knowledge from prior data, enabling the model to adapt effectively to expanding datasets. The proposed approach was tested on different classification models using real-world EHR data.

Results

The results demonstrated that the proposed IL-BBWS enables higher efficiency and even more accurate prediction of DR, while mitigating the challenges associated with periodically updated EHR database. The selected random forest model achieved an F1 score of 0.34, indicating the effectiveness of the approach compared to benchmark methods.

Conclusions

By leveraging IL with BBWS, healthcare providers can achieve significant cost savings while maintaining DR prediction accuracy. This work is especially relevant for rural communities, where limited access to healthcare and low income exacerbates the burden of DR. Reducing costs is critical in these settings, as individuals may not seek timely help due to financial constraints, and hospitals may lack the necessary equipment due to high costs.

RAKSHYA SHRESTHA

Differential TLR-ERK1/2 activity promotes viral ssRNA and dsRNA mimic-induced dysregulated immunity in macrophages.

Day 2 - Oral Presentation



Background

Emerging and re-emerging RNA viruses pose a significant threat to global public health. RNA virus-induced excessive inflammation and impaired antiviral interferon (IFN-I) responses are associated with severe disease. This innate immune response, also referred to as “dysregulated immunity” is caused by viral single-stranded RNA (ssRNA)- and double-stranded-RNA (dsRNA)-mediated exuberant inflammation and viral protein-induced IFN antagonism. However, key host factors and the underlying mechanism driving viral RNA-mediated dysregulated immunity are poorly defined.

Methods

Murine bone marrow-derived macrophages (BMDMs) were stimulated with TLR3 agonist (Poly I:C) and TLR7/8 agonist (R848); each at 5-10 ug/ml for 8 or 24 h. After 8 and 24 h of stimulation, cells in trizol or cell supernatants were collected to assess the mRNA and protein levels of inflammatory cytokines and chemokines and type-I interferons (IFN-I)s through qPCR and ELISA. BMDMs were treated with pathway-specific inhibitors of MAPK (mitogen-activated protein kinase); ERK1/2 (Trametinib), p38 (Losmapimod), JNK inhibitor and NF-kB (BAY11-7082), each at 5μM concentration, for inflammatory pathway inhibition studies. Surface and intracellular expression of TLR3 and TLR7 on BMDMs were determined by FACS analysis whereas phosphorylated levels of MAPKs were assessed by western blot assay.

Results

Our results showed that BMDMs when stimulated with TLR3 and TLR7 agonists, induced differential inflammatory and antiviral cytokine response. TLR7 activation triggered a robust inflammatory cytokine/chemokine induction compared to TLR3 activation, whereas TLR3 stimulation induced significantly increased IFN/IFN stimulated gene (ISG) response relative to TLR7 activation. We identified significantly higher cell-surface and endosomal TLR7 levels compared to TLR3, which were associated with early and robust MAPK (p-ERK1/2, p-P38, and p-JNK) and NF-kB activation in TLR7-stimulated macrophages. Furthermore, blocking ERK1/2 and NF-kB activity reduced TLR3/7-induced inflammatory cytokine/chemokine levels, whereas only ERK1/2 inhibition enhanced viral RNA mimic-induced IFN/ISG response.

Conclusions

Collectively, our results illustrate that high cell-surface and endosomal TLR7 expression and robust ERK1/2 activation drive viral ssRNA mimic-induced excessive inflammatory and reduced IFN/ISG response and blocking ERK1/2 activity would likely mitigate viral-RNA/TLR-induced dysregulated immunity. Further, our findings also highlight an under-appreciated role of viral ssRNA- and dsRNA-mediated differential ERK1/2 activation in virus-induced dysregulated immunity and severe disease.

JYOTI SINGH SAKET

Nanoparticle Tracking Analysis of Goat mEVs

Day 1 - Oral Presentation



Background

Milk-derived extracellular vesicles (EVs) are nanoscale, lipid bilayered particles mediating the intercellular communication by acting as cargos which transports proteins, lipids, and nucleic acids. Goat milk has emerged as an excellent source of EVs due to its advantages over bovine milk, including smaller and more uniform casein micelles, lower allergenicity, and greater similarity to human milk, all of which facilitate higher yield and purity during isolation. The Jamunapari breed — an Indian dairy goat breed mainly found in the state of Uttar Pradesh — is of great economic value for its size, rich milk composition, and high milk yield, making it highly desirable for scalable EV production and other breeding programs which aim at improving dairy traits in other breeds.

Methods

The methodology of this study mainly focuses on differential centrifugation of the goat milk to obtain a clear whey. After defatting the milk, the casein protein present in the milk was precipitated by lowering the pH with the administration of 17.5% acetic acid. Once the clear whey was obtained, it was treated with Polyethylene glycol (24%PEG) and incubated at 4°C for overnight and further centrifuged at 10,000g for 1.5 hours to obtain a pellet containing mEVs.

Results

The results of NTA have shown a heterogeneous population of extracellular vesicles with the particle size in the range of 50-200 nm and particle diameter a slightly smaller than bovine mEVs. Goat milk consistently produced higher concentrations of extracellular vesicles (EVs) per millilitre, highlighting its value for both research and industrial purposes.

Conclusions

NTA is a reliable method for characterizing goat milk extracellular vesicles, aiding quality control and protocol optimization. High-yield breeds like Jamunapari goats further enhance EV production, making goat milk a superior, scalable source for biomedical and nutritional applications.

CODY WHITLEY

The anti-viral role of blocking NOX signaling during coronavirus infections

Day 2 - Oral Presentation



Background

Emerging coronaviruses pose a major threat to public health, as seen during the COVID-19 pandemic, when casualties surged before virus-specific vaccines and therapeutics were available. Corticosteroids are often used early in outbreaks to reduce inflammation; however, they also suppress antiviral responses and offer limited protection. More effective early interventions may come from modulating, rather than suppressing the immune response. One promising target is reactive oxygen species (ROS), which become dysregulated during infection. ROS produced by NADPH oxidase enzymes (NOX), particularly NOX-2, can amplify inflammation and enhance disease severity. In this study, we examined whether inhibition of NOX signaling, an understudied antiviral target, could reduce coronavirus replication and disease severity.

Methods

To evaluate this, we used diphenyleneiodonium (DPI), a broad NOX inhibitor, and assessed the anti-viral and immunological response during coronavirus infections. We infected multiple cell lines with either Mouse Hepatitis Virus (MHV) or SARS-CoV-2, then treated them with low micromolar concentrations of DPI. Virus replication was measured using cytopathic effect (CPE) imaging, plaque assays, and RT-qPCR for sub-genomic viral RNA. Time- of-addition assays were conducted to assess DPI's efficacy when administered post-infection to determine clinical relevancy.

Results

DPI treatment resulted in significant reductions in viral titers, ranging from 1 to 4 log₁₀ across both virus models and cell types, with decreased CPE and viral RNA levels. Notably, these effects persisted even when DPI was added after infection, indicating its therapeutic potential. Preliminary mechanistic analysis suggests that this antiviral activity occurs independently of type I interferon signaling. Preliminary in-vivo findings in NOX-2-deficient mice further support DPI's therapeutic potential, and ongoing studies aim to validate these effects mechanistically.

Conclusions

Our results show that NOX inhibition suppresses coronavirus replication and virus-induced cytopathology, potentially preventing severe disease outcomes. While the exact mechanism remains under investigation, these findings point to a novel, interferon-independent antiviral mechanism. Ongoing studies will further define the molecular underpinnings of this response and evaluate DPI's therapeutic potential in vivo. Taken together, this work highlights host-directed NOX inhibition as a promising approach to mitigate coronavirus infections during future outbreaks, especially when pathogen-specific therapies are not yet available.

SABIHA ALAM

Altered Gut Microbiome and Gut Barrier Interplay: Peripheral Consequences of Fmr1 Gene Mutation in Fragile X Syndrome Mouse Model

Session 1 - Poster Presentation



Background

Fragile X Syndrome (FXS) is the leading monogenic cause of autism spectrum disorders (ASD), resulting from a mutation in the Fmr1 gene on the X chromosome. Besides, well-known cognitive and behavioral challenges, FXS patients often experience altered gut microbiome-induced gastrointestinal (GI) problems, but the mechanisms are not clearly understood. Evidence exists that changes in gut microbial composition and diversity in ASD can exacerbate intestinal barrier integrity and barrier function in the digestive tract. This observation raises a key question whether gut dysbiosis mediates GI dysfunctions in mammals with FXS. Therefore, our research aims to test the hypothesis that altered gut microbiome exacerbates impaired intestinal barrier integrity, inflammation and dysfunctional gut barrier in FXS.

Methods

To test our hypothesis, we used a Fmr1 Knockout (KO) mouse model, that recapitulates FXS disease phenotypes and wildtype (WT) littermates as controls. We utilized 12-16 weeks old male and female mice and stratified them into 5 groups for genotype and sex specific analyses- male Fmr1 KO, Female Fmr1 KO, Male WT, Female WT and Heterozygous (Het) females (n = 6-8/group). Firstly, we explored 16s ribosomal RNA sequencing to analyze microbial composition and diversity in the gut samples of both KO and WT mice. Subsequently, we used RT-qPCR technique to examine gene expression analysis involved in gut barrier integrity and intestinal inflammatory responses in FXS. Finally, we used the Ussing chamber assay to evaluate gut barrier physiology by testing epithelial permeability, and barrier function in the FXS mouse model.

Results

16s ribosomal RNA sequencing data showed that male and female KO mice had a distinct gut microbiome composition, while female Het mice had significantly increased gut microbial diversity compared to control female mice. To reveal molecular mechanisms, RT-qPCR analysis elucidated increased expression of inflammatory gene (TNF- α) and significantly decreased gut barrier integrity regulatory gene expression (Occludin-1, Claudin-2, TJP1, PLVAP) in the gut of female KO mice compared to WT female mice, suggesting GI disturbances in FXS. In parallel, the physiological data from the Ussing chamber suggested that both male and female KO mice showed compromised gut barrier function, indicating GI pathology in FXS.

Conclusions

Overall, these findings indicate that onset of FXS alters gut microbiome, promoting impaired intestinal barrier integrity, inflammatory response and disrupted gut barrier function in mice. Thus, this research highlights the key microbial and genetic biomarkers to establish translational therapies for restoring balanced gut microbiome and gut barrier physiology in FXS.

DEBARATI CHANDA

Assessing the T and B cell responses generated by macrodomain1 deleted SARS-CoV-2 at different phases of infection

Session 3 - Poster Presentation



Background

SARS-CoV-2, which caused the global outbreak of COVID-19 is made of 4 structural proteins- spike (S), membrane (M), envelope (E) and nucleocapsid (N) and 16 non-structural proteins. Mutations in the S protein lead to the emergence of variants of concern (VOCs) that are highly transmissible and infectious. Current FDA approved vaccines encoding the S protein offer protection against severe infection. However, S mutagenesis reduces efficacy of these vaccines in preventing re-infection by VOCs. Therefore, a vaccine incorporating conserved structural proteins (E, N and M) and inducing a robust and durable T cell response is urgently needed to offer long-term protection against VOCs. Δ mac1 is a recombinant SARS-CoV-2 developed by deleting the conserved macrodomain1 which renders the virus non-pathogenic upon infection. Our hypothesis is Δ mac1 infection induces a strong memory T and B cell response protecting against re-infection.

Methods

K18-hACE2 female mice (8-10 W.O) were infected intranasally with 1000 PFUs/lung dose of (Δ mac1) and wild type (WT) SARS-CoV-2 respectively. Lung and spleen tissues were harvested at 9 days post infection (DPI) for acute, 42 DPI for memory and 54 DPI for rechallenge phases. Cells were stained with SARS-CoV-2 spike specific MHC class I-tetramer to determine magnitude and stimulated with CD4 and CD8 T cell peptides to study the polyfunctionality of T cells. Serum IgG levels, lung histopathology, memory T and B cell responses were also compared between Δ mac1 and WT groups.

Results

SARS-CoV-2 spike specific CD8 T cells and cytokine induction were lower in Δ mac1 infected mice compared to WT infected at acute phase. However, increased spike specific CD8 T cells and cytokines were observed in Δ mac1 infected mice at memory and rechallenge phases. Serum IgG levels in Δ mac1 group showed an increasing albeit non-significant trend. Lung histopathology showed that Δ mac1 imparted protection against severe infection with WT. Memory T and B cell responses were lower in Δ mac1 infected group compared to WT.

Conclusions

Our results indicate that non-pathogenic effect of Δ mac1 SARS-CoV-2 infection causes lower T cell response during the acute phase of infection and reduced antibody response following rechallenge with WT infection. However, an enhanced T cell response observed during rechallenge phase of infection indicated a strong T cell response generated could be conferring protection against WT infection. Therefore, Δ mac1 SARS-CoV-2 can serve as a potential vaccine candidate through induction of a robust T cell response following re-infection.

MIA DICKSON

Investigating the role of Nsp1 in Porcine Epidemic Diarrhea Virus replication

Session 1 - Poster Presentation



Background

Porcine Epidemic Diarrhea Virus (PEDV) is an alpha-coronavirus that has a high mortality rate in neonatal piglets and has devastating economic effects. No effective vaccine for PEDV exists. Research has shown that PEDV nonstructural protein 1 (Nsp1) plays an important role in modulating host antiviral response. We hypothesize that Nsp1 is a promising target for designing live-attenuated virus vaccines. The goal of the project is to investigate the cellular localization and interactome of Nsp1 during PEDV infection.

Methods

To this end, we generated a recombinant PEDV expressing a Nsp1 fused with a V5 epitope tag (rPEDV-Nsp1WT-V5), allowing Nsp1 detection in the case of lacking Nsp1 specific antibodies. By comparing the growth kinetics of the wild-type PEDV (rPEDV-WT) and rPEDV-Nsp1WT-V5, we demonstrated that the V5 tag does not impact PEDV replication. To determine the cellular localization of Nsp1, immunofluorescence assays were conducted to detect Nsp1-V5 inside porcine kidney PK1 cells at various time points.

Results

The results reveal that Nsp1 forms unique cytoplasmic puncta that co-localize with the viral nucleocapsid protein, a key protein involved in viral replication. This result suggests that Nsp1 may interact with N protein and participate in modulating viral infection. The experiment revealed that at 6hpi WT Nsp1 is arranged in distinct puncta in the cell, but at 8hpi Nsp1 forms aggregates in the cytoplasm. I further compared the Nsp1 production/location between the rPEDV-Nsp1WT-V5 and two Nsp1 mutants that have been shown to induce higher antiviral response: rPEDV-Nsp1F44A-V5 and rPEDV-Nsp1N93A/N95A-V5.

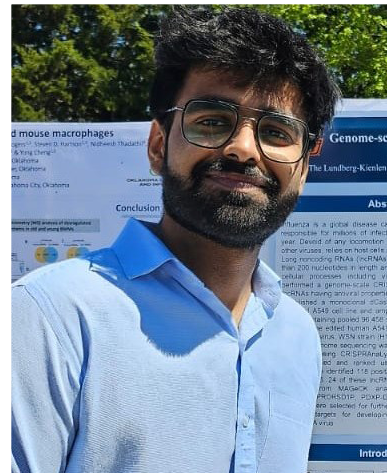
Conclusions

Research and collection of data is ongoing. Future direction of research is to investigate the interactions between Nsp1 and N protein, and uncover the molecular mechanism of Nsp1-mediated immune antagonism.

PRINCE JHANDAI

RHPN1-AS1: A novel antiviral lncRNA against influenza A virus

Session 3 - Poster Presentation



Background

Influenza A virus (IAV) continues to pose a global public health burden due to frequent antigenic changes and resistance to existing antivirals. Host-directed therapeutic strategies are promising alternatives to directly target the virus. Long non-coding RNAs (lncRNAs) have recently gained attention as modulators of host responses to infection, though their specific roles during IAV infection remain largely unexplored. This study aimed to identify antiviral lncRNAs and functionally characterize RHPN1-AS1 as a potential antiviral of influenza virus replication.

Methods

A genome-wide CRISPR activation (CRISPRa) screen was performed in A549 lung epithelial cells infected with the H1N1 WSN strain of IAV. Surviving cells were subjected to next-generation sequencing, revealing 118 candidate antiviral lncRNAs. Expression levels of selected lncRNAs were analyzed by qRT-PCR following IAV infection. RHPN1-AS1 was selected for functional validation based on expression profile and novelty. A549 cells were transfected with RHPN1-AS1-targeting siRNA or stably overexpressed RHPN1-AS1 via lentiviral vectors. Cells were then infected with multiple influenza strains, and viral replication was quantified through western blotting for nucleoprotein (NP), viral titers via TCID₅₀, and luciferase reporter assays, which measure viral polymerase activity and genome replication using reporter viruses. Host response markers including RIG-I were examined by western blot. Time-course experiments were also performed to assess dynamic changes in viral suppression.

Results

Next-generation sequencing analysis identified 118 potential antiviral lncRNAs, among these, RHPN1-AS1, CTAGE7P, ABCC13, PRORS1P, PDXP-DT, LINC01257, LINC01501, and CRNDE were selected for further investigation. Among these, RHPN1-AS1 and PRORS1P showed significant upregulation during IAV infection. Functional studies revealed that knockdown of RHPN1-AS1 led to increased NP levels and viral titers at multiple time points. In contrast, overexpression of RHPN1-AS1 consistently suppressed NP expression and reduced viral titers in cells infected with H1N1 WSN, PR8, and California strains, as well as H3N2 Oklahoma. Luciferase assays showed significant reduction in viral polymerase activity, supporting the suppressive effect of RHPN1-AS1 on viral replication. Additionally, Influenza B virus (Malaysia) infection showed reduced NP expression upon RHPN1-AS1 overexpression, indicating cross-strain antiviral potential. A modest increase in RIG-I protein levels was observed, suggesting a possible link to innate immune activation.

Conclusions

RHPN1-AS1 is a novel antiviral lncRNA that restricts replication of multiple IAV subtypes and Influenza B virus. These results highlight RHPN1-AS1 as a promising candidate for host-targeted antiviral therapies and lay the groundwork for future mechanistic studies to explore its role in innate immunity and virus-host interactions.

STEPHEN KOTEY

The Role of Long Non-Coding RNA Mir155HG During Host – Mycobacteria Tuberculosis Interactions in Macrophages

Session 2 - Poster Presentation



Background

Long non-coding RNAs (lncRNAs) are a group of RNAs with more than 200 nucleotides that do not code for proteins. It remains unclear how lncRNAs regulate the host-pathogen interactions in host cells. However, lncRNA differential expression in host cells has been implicated in immune regulation during bacterial infection, playing beneficial or detrimental roles in host defence against bacterial infections. With the causative agent of tuberculosis, *Mycobacterium* (M.tb), being a major source of human suffering since antiquity, there is an urgent need to understand how this global threat interacts with the host.

Methods

Next-generation RNA sequencing, Mycobacterial survival assay, Confocal Microscopy, Mass spectrometry analysis, RNA and protein pulldown assays, Western blot, Histopathology studies.

Results

Through a next-generation RNA sequencing analysis, we identified a list of host lncRNAs that were differentially regulated in the lung of mice that were infected with mycobacteria. Among them, the expression of one host lncRNA, Mir155HG, was highly upregulated in the infected mice. Through various cell and molecular biology assays we discovered that this lncRNA is important for M.tb survival and proliferation within macrophages as well as the lungs of mice. Moreover, it played a crucial role in the inflammatory state of mice histopathology after M. tb infection. Functional analysis showed that lncRNA Mir155HG aids in the phagolysosomal processing of M. tb within macrophages through the regulation of actin cytoskeleton polymerization. Furthermore, proteomics and interaction studies showed that lncRNA Mir155HG associates with the beta subunit of the well-known heat shock protein Hsp90. We showed that Hsp90 β has an effect opposite to lncRNA Mir155HG on the proliferation and survival of M. tb, as well as the effect on actin cytoskeleton polymerization.

Conclusions

Our findings point to an interesting relationship between these two biomolecules that determines the pathogen clearance ability of host macrophage cells and the progression of M. tb infection within the immune cells. This work highlights the immune regulatory role of the non-coding regions of the DNA in the development of host-directed therapy for tuberculosis patients.

XUEYING LIANG

SARS-CoV-2 nsp15 endoribonuclease subverts host defenses to enhance viral fitness in lung cells

Session 3 - Poster Presentation



Background

SARS-CoV-2, like other coronaviruses, encodes the conserved endoribonuclease Nsp15/EndoU to evade host innate immunity. While its enzymatic activity has been biochemically characterized, its precise role in respiratory epithelial cells remains unclear. This study aimed to define the role of Nsp15 in immune evasion and pathogenesis during SARS-CoV-2 infection of lung epithelial cells and in vivo.

Methods

The recombinant SARS-CoV-2 viruses used in this study were generated using a reverse genetics system. Viral replication and host immune responses were assessed in human lung-derived epithelial cell lines, air-liquid interface (ALI) cultures, and iPSC-derived alveolar type 2 (AT2) organoids. Stress granules were visualized via immunofluorescence. In vivo pathogenicity was evaluated using K18-hACE2 transgenic mice.

Results

SARS-CoV-2 Nsp15 endoribonuclease activity is dispensable for viral replication in Vero cells but is critical for immune suppression and efficient infection in human lung epithelial cells and organoids. Compared to rWT, infection with rH234A elicited stronger type I and III interferon (IFN) responses and enhanced RNase L activation, suggesting that Nsp15 suppresses these antiviral pathways during SARS-CoV-2 infection. Although rH234A infection increased PKR phosphorylation, downstream eIF2 α phosphorylation and stress granule formation remained limited, implying incomplete PKR/eIF2 α pathway activation. Furthermore, Nsp15 mutants lacking key residues required for hexamerization or double-stranded RNA (dsRNA) binding also showed impaired replication and heightened IFN responses, highlighting the importance of these structural features for EndoU function. In vivo, rH234A was attenuated in K18-hACE2 mice, leading to improved survival, lower viral loads, and milder lung pathology. These findings highlight the critical role of Nsp15 structural integrity and enzymatic function in immune evasion and pathogenesis.

Conclusions

These findings establish SARS-CoV-2 Nsp15/EndoU as a key immune antagonist that facilitates lung infection, underscoring its potential as a target for antiviral development.

SAMANTHA LOVING

An equation to determine the percent of blood contamination in a stallion's ejaculate: based on PCV

Session 2 - Poster Presentation



Background

Hemospermia, the presence of blood in stallion ejaculate, is a clinically significant condition associated with reduced fertility. The exact mechanism by which blood contamination impairs fertility is unknown, existing studies suggest blood contamination $\geq 20\%$ consistently causes infertility. Currently, no established method to quantify the extent of blood contamination in semen. We used mathematical modeling to predict the PCV of a hemospermic ejaculate based on PCV from whole blood resulting in the following equation: Percent of blood in ejaculate = $[\text{PCV semen} + (\text{PCV blood}/100) \div (\text{PCV blood}/100)]$. This experiment validated the equation's accuracy in estimating blood contamination.

Methods

The protocol compared the actual percent of blood in the ejaculate to predicted percent of blood in the ejaculate based on PCV of blood and artificially induced hemospermic samples created with mare, self, and another stallion. Blood from jugular venipunctures was added to semen at 0, 20, 40, 60, 80, and 100% dilutions; blood and semen-only controls were included. PCV of all dilutions and controls were measured and applied to the equation.

Results

Data from all blood sources were combined as they were not statistically different. The equation, on average, slightly underpredicted, a mean difference of -2.73 ± 0.87 ($p = 0.002$). The test for zero bias was not significant and there was no significant correlation in the independence of bias.

Conclusions

These results suggest the amount of blood contamination in semen can be estimated based on the PCV of the stallion's blood and PCV of the ejaculate using this equation. This study provides a practical diagnostic tool for clinical assessment of hemospermia.

ANNA CLAIRE MCMULLEN

Pharmacokinetics of high dose oral gabapentin in horses

Session 2 - Poster Presentation

Background

Gabapentin use is ubiquitous in the veterinary field. It is usually administered for sedation and analgesia, including for the therapy for chronic pain in equine patients. However, little is known about the pharmacokinetics of multiple-dose administration of gabapentin in horses.

Methods

In this study, gabapentin was administered orally to 8 horses at a dose rate of 100 mg/kg twice daily for 56 doses. Plasma samples were collected at preset times after the first dose, immediately before the 14th, 28th, and 42nd dose, and at regular intervals after the 56th dose.

Results

Analytical method development using LC-MS/MS is ongoing. For the mobile phase, a gradient consisting of 10 mM ammonium acetate at pH 5.7 and 50:50 methanol :acetonitrile was chosen. After trialing five reverse-phase columns for liquid chromatography, the retention time of gabapentin was consistently less than two minutes. Due to gabapentin's high polarity, a normal-phase column (Atlantis HILIC Silica 3 μ m) was instead chosen to extend its retention time to 6.5 minutes. Deuterated gabapentin was originally tested as an internal standard (IS). However, cross-interference occurred between the gabapentin and IS, likely due to competition for electrospray ionization. Thus, pregabalin was chosen as an alternate IS. Oasis MCX columns were selected to perform solid phase extraction of plasma samples. Recovery was determined by adding 10 μ g/mL gabapentin to triplicate blank plasma samples pre-extraction, post-extraction, and after reconstitution. Mean $\pm$ SD recovery from pre-extraction fortified samples was 86% $\pm$ 9%, while recovery from post-extraction fortified samples was 100% $\pm$ 5%.

Conclusions

Data collection and analysis is ongoing.



JEEVIYA MURUGESAN

A single-cycle live-attenuated respiratory syncytial virus vaccine focused on prefusion F and the central conserved region of attachment protein G



Session 3 - Poster Presentation

Background

Respiratory Syncytial Virus (RSV) is a leading etiological agent of bronchiolitis and pneumonia in infants and young children, causing over 100,000 fatalities annually worldwide. Despite its substantial clinical burden, no vaccine is available for the pediatric population. Previously, we developed RSV-preF, a live prefusion-F (preF) expressing single-cycle RSV vaccine that demonstrated protective efficacy in a murine model. Because preF is no longer fusion-competent, RSV-preF is propagated in a trans-complementing cell line that provides baculovirus fusion protein GP64 to mediate viral entry. Following viral entry, RSV-preF cannot spread to neighboring cells and has a single-cycle phenotype. To further enhance efficacy and cross-protection against diverse RSV strains, we focused the anti-attachment protein (G) response on the G central region (GCR) which is the most conserved area and contains a receptor-binding domain.

Methods

An experimental vaccine was generated by molecular cloning techniques expressing only the GCR (RSV-preF-GCR) and compared to a vaccine expressing full-length G (RSV-preF-G). Mice were vaccinated intranasally using a prime-boost regimen. Blood samples were collected three weeks post-boost and levels of anti-preF and anti-G antibodies examined by ELISA. In another study, vaccinated mice were challenged with wildtype RSV and lungs were harvested at five days post-challenge.

Results

Antibody assay showed that in spite of expressing only a small portion of the G protein, RSV-preF-GCR induced robust anti-G antibody levels demonstrating that the GCR was immunogenic. RSV-preF-GCR also elicited high levels of antibodies against the preF protein. Histopathological analysis of lung sections indicated that RSV-preF-GCR vaccination conferred protection similar to that of the full-length RSV-G vaccine.

Conclusions

Thus, a vaccine expressing only the GCR elicited a broad and protective response, including a high level of GCR-specific antibodies. Based on the conserved nature of the GCR, the next phase of this study will assess the efficacy of RSV-preF-GCR vaccination against a range of RSV strains, to determine its potential to improve cross-protection.

DIANI NANAYAKKARA

Palmitic Acid-Induced Structural Changes in Human and Mice Brainstem Astrocytes: Implications for Sympathoexcitation in Obesity

Session 2 - Poster Presentation



Background

Obesity is increasingly recognized as a condition involving chronic low-grade inflammation that extends to the central nervous system. Saturated fatty acids such as palmitic acid (PA), common in high-fat diets, are implicated in astrocyte dysfunction, particularly within the brainstem, a key autonomic center. However, the temporal dynamics of PA-induced astrocytic structural changes remain unclear. We have previously shown that high-fat diet (HFD)-induced obesity in mice activates brainstem astrocytes and could contribute to sympathoexcitation. This study investigated the time-dependent structural changes and lipid accumulation in human and mice brainstem astrocytes following PA exposure.

Methods

Primary human brainstem astrocytes (HBA) were cultured at 50,000 cells per well in a 24-well plate, with four independent experiments including triplicates of control, vehicle, and PA-treated groups. HBA were treated with 60 μ M of PA for 24 hours, followed by immunocytochemistry (ICC) for astrocyte marker glial fibrillary acidic protein (GFAP) and confocal imaging. A time-course experiment (8, 10, 12, and 14 hours post-treatment) was conducted to determine the initiation of morphological changes in astrocytes. BODIPY staining was used to assess lipid accumulation. In addition, astrocytes from HFD and low-fat diet (LFD) mice were analyzed using immunohistochemistry (IHC) for GFAP.

Results

PA-treated astrocytes exhibited structural remodeling, transitioning from their normal star-shaped morphology to a rounded phenotype with process retraction, first emerging at 10 hours post-treatment. BODIPY staining confirmed increased lipid accumulation, indicating PA-induced lipotoxicity. Astrocytes in HFD mice displayed a similar reactive phenotype, reinforcing the link between dietary lipids and astrocyte dysfunction.

Conclusions

PA treatment induces significant morphological alterations and lipid accumulation in brainstem astrocytes, leading to a reactive phenotype. These findings emphasize the role of dietary lipids in astrocyte dysfunction, contributing to obesity-related neuroinflammation. This study provides insights into obesity-induced astrocytic dysfunction, highlighting astrocytes as potential therapeutic targets for mitigating lipotoxicity and neuroinflammation in metabolic disorders.

EMMA QUINTANA

HSV-2 infection is directly promoted by progesterone and progesterone receptor in vaginal keratinocytes

Session 2 - Poster Presentation



Background

Herpes Simplex Virus-2 (HSV-2) is a globally prevalent pathogen that causes lifelong infection and significant burden of disease. After primary infection, HSV-2 establishes latency in sensory neurons of the spinal ganglia, with recurrent outbreaks resulting from viral reactivation. No vaccines currently exist to prevent transmission or latency, and antiviral therapies offer only modest symptomatic relief by shortening the duration of outbreaks. Transmission to neonate during delivery is devastating with a 65% mortality rate and 80% rate of long-term disability. Women consistently show disproportionately higher rates of HSV-2 acquisition and experience more frequent and severe recurrences than men. However, research into sex-specific mechanisms of HSV-2 pathogenesis remain sparse and primarily focus on the immunomodulatory effects of female hormones estrogen and progesterone. Our lab's previous work suggests that hormone receptors themselves directly regulate herpesvirus infections. Therefore, we hypothesize that the progesterone receptor (PR) promotes HSV-2 infection and facilitates reactivation from latency.

Methods

Vaginal keratinocytes were grown in serum free media and split into multiwell plates. At desired time points, hormones were added in desired concentrations and combinations. Besides testing for cytotoxicity, hormones were added generally at 10 μ M since it is close to plasma concentration achievable in humans. Our control well contained no hormones. An HSV-2 strain was added to the wells at the desired time point for desired infection time. Analysis was completed via cytotoxicity, plaque assay, and RT-qPCR.

Results

Cytotoxicity of our vaginal keratinocyte model did not occur up to 50 μ M of hormones tested. We found that progesterone (P4) significantly increased HSV-2 replication, while treatment with PR antagonists reduced both HSV-2 lytic gene expression and viral titers. Strikingly, HSV-2 induced nuclear localization of PR even in the presence of antagonists.

Conclusions

These findings suggest a direct role for PR in HSV-2 productive infection. Ongoing studies will validate these results using organoid and in vivo infection models, determine PR binding sites on the HSV-2 genome driving acute infection, and investigate PR's role in HSV-2 reactivation from latency. Studies like ours examining progesterone and PR as a mechanism for HSV reactivation from latency are critical as nearly 151 million women of reproductive age worldwide use hormonal contraceptives formulated with progesterone, progesterone is a first-line treatment during pregnancy to prevent preterm birth, and over 50 million women use progesterone therapy to manage menopausal symptoms.

Exploring Nasal *Staphylococcus aureus* Extracellular Vesicles: Isolation, Characterization, and Their Impact on Host Interaction in an Air-liquid interface culture

Session 1 - Poster Presentation



Background

Staphylococcus aureus is a major opportunistic pathogen that resides in the skin and nose of humans. Infections caused by *S. aureus* vary from mild to severe and potentially fatal, especially when they enter the bloodstream and cause sepsis, pneumonia in the lungs, endocarditis in the heart valves, and osteomyelitis in the bone. In the community, people with chronic conditions such as diabetes, cancer, and vascular disease are more prone. In contrast, in the healthcare setting, the *S. aureus* infection is highly prevalent in the intensive care units, in people who have undergone surgeries with medical device implantations, etc. Even though antibiotics are available to suppress *S. aureus* infections, the bacteria develop resistance to these drugs, making it difficult to treat them. Recent studies indicate that *S. aureus* produces extracellular vesicles (EVs), also known as membrane vesicles (MVs), which play a role in cellular communication, the transport of virulence components, and in the regulation of the host immune response. Nonetheless, the influence of culture conditions on the physicochemical and functional characteristics of these EVs remains largely unexplored.

Methods

Nasal-derived *S. aureus* was cultured with brain-heart infusion broth overnight in a shaking incubator at 37 degrees Celsius at 250 rpm. The extracellular vesicles were extracted from the media by concentrating using Amicon filters (100 kDa) and further processed through IZON columns, with the resulting fractions being collected. These EVs were then characterized using Spectradyme's nanoparticle analyzers, transmission electron microscopy (TEM), and Western blot. After the characterization, the extracted EVs were used to treat air-liquid interface (ALI)-grown respiratory epithelial cells (10 µg of EV protein concentration). The cells were harvested, and RNA was extracted and sequenced to understand the mechanistic role of the EVs.

Results

Our preliminary analysis showed that the isolated EVs' size falls in the 70-100 nm range, which is confirmed through particle size analyzer and TEM, and the western blot validated that the extracted EVs are LTA positive. The single-cell RNA differential gene expression analysis revealed that there is a significant upregulation of specific immunological transcripts.

Conclusions

This study provides the first general picture of isolation and characterization of nasal-derived *S. aureus* EVs. To understand how these EVs play a role in host-bacterial communication, RNA and protein sequencing need to be done in our future studies to explore and understand cell-cell communication.

MADELINE REICHERT

Isolation of Novel Antimicrobial Compounds from the Nasal Mutualist *Dolosigranulum pigrum*

Session 1 - Poster Presentation



Background

Acute respiratory tract infections (ARIs) are responsible for over two million deaths each year, and with the rise of antibiotic resistance, there is an increasing need for novel therapeutics. *Dolosigranulum pigrum* is a Gram-positive lactic acid bacterium found primarily in human upper respiratory tract (URT). Microbiome sequencing studies have shown an inverse relationship between common nasal pathobionts (e.g., opportunistic pathogens) associated with ARIs, like *Staphylococcus aureus*, and *D. pigrum*, suggesting that *D. pigrum* may act as a mutualist in the URT by limiting pathobiont colonization. How *D. pigrum* inhibits these pathobionts is not well-understood. One common way bacteria compete is through the production of bioactive secondary metabolites, such as ribosomally synthesized and post-translationally modified peptides (RiPPs), many of which have antimicrobial properties (e.g. nisin). Our hypothesis is that *D. pigrum* competes with nasal pathobionts through production of antimicrobial secondary metabolites.

Methods

We isolated and sequenced the genomes from 40 *D. pigrum* strains in order to examine the presence of Biosynthetic Gene Clusters (BGCs) predicted to encode for secondary metabolites. We also screened our *D. pigrum* collection for in-vitro inhibitory activity by co-culturing them alongside a panel of common nasal pathobionts: *Haemophilus influenzae*, *Micrococcus luteus*, *Moraxella catarrhalis*, *Staphylococcus aureus*, and *Streptococcus pneumoniae*.

Results

When we analyzed the genomes, we found BGCs predicted to encode for RiPPs, and the majority of these RiPPs are not similar to any characterized compound. Additionally, the inhibitory activity of *D. pigrum* against the panel of nasal pathobionts was highly variable between strains.

Conclusions

D. pigrum has strain-dependent inhibitory activity against common URT pathobionts and encode for a variety of uncharacterized RiPPs. In the future we plan to isolate the antimicrobial peptides responsible for the inhibitory activity we observed in my co-cultures. We also plan to establish a genetic system for *D. pigrum* in order to test how manipulating BGCs impact RiPP production and bioactivity.

AINSLIE REYNOLDS

Evaluating protective efficacy of inactivated bovine H5N1 vaccine with STING adjuvant in mouse model

Session 3 - Poster Presentation



Background

The emergence of highly pathogenic influenza viruses poses a significant threat to both human and animal health. The recent detection of highly pathogenic avian influenza (HPAI) H5N1 in bovine species presents a serious hazard to the food industry and increases the risk of zoonotic transmission. Developing an effective vaccine that elicits robust and durable immunity is critical, not only to prevent animal mortality but also to reduce the potential for spillover into the human population.

Methods

In this study, we used a mouse model to evaluate the immunogenicity and magnitude of antibody and T-cell responses to inactivated H5N1 bovine influenza adjuvanted and non-adjuvanted with a novel STING (stimulator of interferon genes) agonist as a vaccine.

Results

Our results demonstrated that, compared to PBS-treated and adjuvant-only control groups, mice immunized with inactivated H5N1 exhibited a significant increase in hemagglutinin (HA)-specific, but not neuraminidase (NA)-specific, antibody responses at both two- and four-weeks post-immunization. Furthermore, the addition of the novel STING agonist significantly enhanced immune responses relative to the non-adjuvanted vaccine group at both time points.

Conclusions

These findings suggest that the STING-adjuvanted inactivated H5N1 vaccine elicits a more robust immune response in animal models. Effective antibody and T-cell responses are essential for durable protection against HPAI in livestock. By extension, this strategy holds potential for inducing strong, protective immunity in bovine species.

KEERTHANA SANTHOSH

PARP16 Promotes Intracellular Survival of Mycobacterium abscessus: A Host-Directed Therapeutic Target

Session 2 - Poster Presentation



Background

Mycobacterium abscessus (M. ab) is a rapidly growing non-tuberculous mycobacterium (NTM) increasingly associated with chronic pulmonary infections and poor treatment outcomes. Its intrinsic drug resistance and ability to persist within macrophages present significant therapeutic challenges. Identifying host factors that support M. ab survival is essential for developing alternative, host-directed treatment strategies. Poly ADP-ribose polymerases (PARPs) are host enzymes that regulate DNA repair, immune signaling, and endoplasmic reticulum (ER) stress responses. Among them, PARP16 resides in the ER and governs the unfolded protein response. We hypothesized that M. ab exploits PARP16 to enhance its intracellular persistence. This study evaluated PARP16 as a pro-bacterial host factor and potential therapeutic target in M. ab infection.

Methods

THP-1 monocytes were differentiated into macrophages and infected with M. ab at a multiplicity of infection (MOI) of 5 for 24 hours. Expression profiles of all 17 PARP family members were analyzed using qRT-PCR. A systematic siRNA knockdown (KD) screen targeting each PARP was conducted to assess their impact on bacterial survival using colony-forming unit (CFU) assays. CFU kinetics were subsequently monitored in both THP-1 macrophages and bone marrow-derived macrophages (BMDMs) isolated from wild-type (WT) and PARP16 knockout (KO) mice. In vivo infections were then performed in PARP16 KO and WT mice to quantify bacterial burden and evaluate disease progression.

Results

qRT-PCR revealed differential regulation of all 17 PARPs during M. ab infection. Notably, PARP16 knockdown significantly reduced intracellular bacterial load, identifying it as a host factor exploited by M. ab to sustain survival. In both THP-1 cells and BMDMs, PARP16-deficient conditions consistently exhibited lower bacterial loads across multiple time points. In vivo, PARP16 KO mice showed a marked reduction in bacterial burden compared to WT controls, confirming the in vitro findings.

Conclusions

Collectively, our data establishes PARP16 as a host factor that facilitates M. ab survival. While the downstream mechanisms remain to be clarified, PARP16's known role in ER stress presents a compelling avenue for future investigation. Understanding how M. ab leverages host pathways may uncover new aspects of pathogenesis. Targeting PARP16 could provide a novel, resistance-proof therapeutic strategy against drug-resistant NTM infections, addressing an urgent unmet clinical need.

SHOROQ SHATNAWI

Insights into Inter-Species Infectivity and Variable Pathogenicity between SARS-CoV-2 Variants of Concern

Session 3 - Poster Presentation



Background

Understanding how SARS-CoV-2 variants differentially interact with host tissues is essential for guiding variant-specific diagnostics and therapeutics. Among variants of concern, Delta and Omicron XBB.1.5 have demonstrated divergent clinical profiles, transmissibility, and immune evasion capacities, but their comparative pathogenic mechanisms remain incompletely defined. Objectives: This study aimed to dissect the virological and molecular differences between Delta and Omicron XBB.1.5 using physiologically relevant feline models and human nasal epithelial cell (HNEC) cultures. We investigated viral replication dynamics, tissue-specific pathogenesis, and host transcriptional responses to illuminate variant-specific adaptation strategies.

Methods

We quantified viral RNA and infectivity across time points in feline and HNEC systems, evaluated histopathological changes, localized SARS-CoV-2 antigen in ACE2⁺ cells, and profiled variant-specific viral gene expression patterns. Gene targets such as ORF1ab, S, M, ORF6, ORF7a, ERCC-00113, and ERCC-00046 were analyzed to characterize transcriptional remodeling.

Results

Delta-infected cats exhibited significantly higher viral loads in the upper respiratory tract (URT) and retained greater infectivity in HNECs compared to Omicron. Interestingly, Omicron induced more severe URT pathology despite lower viral burden, suggesting a stronger immune-mediated response. Delta infection caused extensive lower respiratory tract (LRT) damage and broader ACE2⁺ cell targeting. Transcriptional profiling revealed that Delta underwent marked downregulation of viral genes across feline tissues, while Omicron maintained a more static expression profile. Cross-model comparisons showed Delta's enhanced transcriptional adaptability and host-specific remodeling.

Conclusions

Delta and Omicron XBB.1.5 utilize fundamentally distinct replication and adaptation strategies. Delta is characterized by high transmissibility, aggressive replication, and tissue invasion, while Omicron favors restricted URT replication with a robust local immune response. These findings highlight the importance of evaluating variant-specific host interactions and support the use of feline and HNEC models in anticipating zoonotic potential and informing pandemic preparedness.

SARS-CoV-2 serological surveillance in zoo animals

Session 3 - Poster Presentation



Background

SARS-CoV-2 is capable of interspecies transmission, infecting both humans and animals. Zoo animals are particularly vulnerable due to their close and frequent contact with humans. While viral infection and vaccine responses have been extensively studied in humans, serological surveillance in zoo animals remains limited. In this study, we analyzed serum samples from vaccinated zoo animals to evaluate the antibody responses.

Methods

Serum samples were obtained from animals at two zoological institutions, Fort Wayne Children's Zoo, Indiana, and Brookfield Zoo, Illinois. The animals were vaccinated against SARS-CoV-2 in 2021 with a recombinant WH1 Spike-based protein vaccine. Three serological assays were used in the study: a nucleocapsid protein-based ELISA (N-ELISA) detected antibodies elicited by prior SARS-CoV-2 infection, a surrogate virus neutralization test (sVNT), and a neutralization assay with Spike-pseudotyped viral particles evaluated the Spike-specific neutralizing antibodies.

Results

37 out of 118 samples (31.4%) from Fort Wayne Children's zoo were sVNT-positive, 2 were N-ELISA-positive, and 1 Sumatran tiger tested positive on both assays. At Brookfield, 20 out of 122 samples (16.4%) were sVNT-positive, with 1 sloth bear positive on both assays. It marks the first documented SARS-CoV-2 exposure in the species. We tested some of the sVNT positive samples with the neutralization assay. These samples exhibited strong activity against the WH1 spike pseudotyped lentivirus but showed significantly reduced neutralization against the Omicron LP8.1 Spike pseudotyped lentivirus.

Conclusions

Our findings indicate that the vaccine induced high levels of antibodies in some animal species, but they may not effectively neutralize the emerging Omicron variants of the virus. Our results highlight the fact that there is a need for continued serological monitoring of the captive animals and a need for updated vaccines that can act against the new variants of SARS-CoV-2.

AKSHAYA SURENDRAN

Silencing of Circular RNA COL1A1 (circCOL1A1) Alleviates Bleomycin-Induced Pulmonary Fibrosis in Mice

Session 3 - Poster Presentation



Background

Idiopathic Pulmonary Fibrosis (IPF) is a life-threatening interstitial lung disease that primarily affects older individuals. This condition severely impacts on the patient's quality of life due to poor prognosis and the lack of effective treatment. A key mechanism contributing to the development of lung fibrosis is the abnormal activation of pulmonary fibroblasts, leading to an increase in myofibroblast population. This results in the overproduction of ECM and scar tissue. Circular RNAs (circRNAs) have gained significant attention over the past few decades because of their regulatory roles in neoplastic, inflammatory, and fibrotic diseases. They can exert their regulatory mechanisms by acting as a miRNA or protein sponge. However, the exact role of circRNAs in fibroblast activation has not been explored in full potential.

Methods

Several dysregulated host genes with conserved circRNAs were identified through the reanalysis of publicly available bulk and single-cell RNA sequencing datasets from IPF lungs and healthy controls. Among them, circCOL1A1 was chosen for further study. CircCOL1A1 expression was evaluated in lung tissues and isolated fibroblasts from a bleomycin-induced pulmonary fibrosis mouse model. In human pulmonary fibroblasts (HPFs), pro-fibrotic cytokine TGF- β 1 treatment was used to assess circCOL1A1 regulation. Functional studies involved lentiviral-mediated knockdown or overexpression of circCOL1A1 in HPFs, followed by analysis of α -smooth muscle actin (α -SMA) expression using qPCR and Western blot. To investigate the underlying mechanism, a dual-luciferase reporter assay was conducted to assess the miRNA sponging activity of circCOL1A1 toward miR-29b, miR-665, and miR-138, using circCOL1A1 overexpression vector, miRNA sensors, and miRNA mimics. For in vivo studies, adenoviral shRNA was used to knock down circCOL1A1 in bleomycin-treated mice. Fibrosis was evaluated through clinical scoring, body weight monitoring, fibrosis scores, and Sircol collagen assay, along with α -SMA immunostaining and Trichrome staining.

Results

CircCOL1A1 was significantly upregulated in fibrotic mouse lungs and in HPFs following TGF- β 1 stimulation. In HPFs, circCOL1A1 knockdown reduced α -SMA expression, while overexpression increased α -SMA expression, indicating circCOL1A1 promotes fibroblast activation. Mechanistic analysis showed that circCOL1A1 sponges antifibrotic miR-138. In Vivo, circCOL1A1 knockdown led to reduced fibrosis, improved clinical scores, and better weight recovery in mice. Histological and biochemical analyses confirmed decreased collagen deposition and fibroblast activation.

Conclusions

The results strongly suggest that circCOL1A1 is a profibrotic gene that activates pulmonary fibroblasts, highlighting its therapeutic potential for Idiopathic Pulmonary Fibrosis.

JESTER TRESNER

Healios: Dynamic Bacteriophage Based Photodynamic Therapy

Session 2 - Poster Presentation



Background

We are a team of undergraduate students from the OSU Synthetic Biology Club, aiming to compete in the annual iGEM (International Genetically Engineered Machine) competition, where students from around the world design and build biological systems to address real-world problems. Last year, we participated by developing a novel approach to treat bacterial skin infections.

Around 30% of the human population is colonized with *Staphylococcus aureus*, a bacterium that functions as both a commensal species and a major cause of medical infections. The rise of antibiotic-resistant strains has led to about 100,000 deaths in 2019, highlighting the urgent need for new therapies. Bacteriophage therapy offers one approach, using naturally lytic viruses that infect only bacterial cells. Their surface proteins enable them to bind specific targets, making them potential delivery vehicles. Photodynamic therapy (PDT) is another strategy shown to combat drug-resistant pathogens, including *S. aureus*. PDT uses photosensitizing compounds that, when activated by light, generate reactive oxygen species (ROS) to rupture bacterial cells. While effective and capable of enhancing immune response, PDT can harm healthy cells. To minimize damage, photosensitizers can be bound to bacteriophages, allowing targeted killing of pathogenic cells.

Methods

Our goal was to engineer M13 bacteriophages to display *S. aureus*-binding peptides (Gp3 or Gp8 fusions) using a modular system composed of a helper phage and a plasmid for fusion protein expression via GoldenGate cloning. This system will allow rapid substitution of targeting peptides. Engineered phages will be chemically conjugated to the photosensitizer Rose Bengal. After binding *S. aureus* cells, targeted photodynamic therapy will be applied using wavelength-specific light to activate Rose Bengal, generating reactive oxygen species (ROS) and inducing bacterial lysis.

Results

We designed our two-plasmid system using *in silico* cloning and had a commercial company synthesize the gene fragments. Due to time and budget constraints, we are still in the process of assembling the Helios system.

Conclusions

We anticipate continuing this work to complete the project and compete in the 2026 iGEM competition.

VISHNU THAYIL VALAPPIL

Mucin-Adhering Bacteria Alter Gut Mucosal Microbiome Assembly via Priority Effects

Session 1 - Poster Presentation



Background

Early microbial colonizers establish trajectories that shape gut microbiome composition, metabolic output, and lifelong host health. Yet the filters selecting these pioneers remain poorly defined. Many early colonizing taxa gain an early advantage by binding and degrading mucin glycans thereby securing nutrients, altering pH and excluding competitors. Such mucin mediated priority effects could dictate subsequent community assembly and pathogen resistance, but direct mechanistic evidence is scarce. We hypothesized that strong mucin-adhering taxa exploit spatial anchorage to monopolize early niches, thereby orchestrating community trajectories through priority effects.

Methods

Adhesion to mucin by the 102 human gut microbiome library was assessed using a crystal violet screening assay. Confocal microscopy was utilized to visualize the structure and viability of biofilms formed on mucin-coated surfaces. A packed column and drip flow bioreactor were utilized to mimic the mucosal gut environment for exploring colonization dynamics and microbial recruitment. We tested ecological relevance in germ free *Caenorhabditis elegans* after which we monitored colonization dynamics. Community composition was quantified by 16S rRNA and metagenomic shotgun sequencing. Community composition shifts were assessed by PERMANOVA and visualized with PCoA.

Results

We screened 102 species from our gut microbiome library for adherence capacity and identified species with strong, moderate, or no mucin-binding ability. Upon addition of mucin, only eight species bound effectively, with *Bifidobacterium longum*, *Bacteroides caccae*, *Bacteroides finegoldii*, and *Olsenella umbonata* showing strong adherence. In a mucin-coated mini bioreactor colonized with a fecal community, these strains occupied >50 % relative abundance 48 h post-inoculation retaining their mucin-binding ability, confirming adherence even within a complex microbiota. Stability assays in a drip-flow bioreactor proves these species remained detectable after fecal introduction. To examine their influence on downstream community assembly, each strain was used to pre-colonize the drip flow bioreactor before fecal community addition. *B. longum* favored limited recruitment, while *Bacteroides* showed similar patterns of bacterial recruitment. In contrast, *O. umbonata* led to a distinct community structure. Validation in *C. elegans* confirmed that early colonizers drive unique community recruitment, suggesting that initial mucin-binding species can shape microbiota composition as observed in-vitro.

Conclusions

Our findings indicate that species capable of robust mucin adhesion act as ecological gatekeepers by monopolizing the mucosal niche dictate downstream community structure. These findings highlight adhesion phenotype as a key determinant of priority effects in gut microbiome assembly and suggest that targeted administration of strong mucin adherers may represent a viable strategy for microbiome modulation in early life.

BINTA VARGHESE

Redefining the Global Core Human Gut Microbiome: Global Prevalence, Metabolic Activity, and Clinical Significance of Low-Abundance Bacteria

Session 1 - Poster Presentation



Background

Gut microbiome research has predominantly focused on high-abundance bacterial taxa, often dismissing low-abundance species in the metagenome analysis as technical noise with minimal biological relevance. Yet emerging evidence reveals that these taxa exert disproportionate influence on microbial community structure, host physiology, and ecosystem resilience. Deciphering the roles of these microbes is essential for advancing microbial ecology and deepening our understanding of host-microbe interactions in health and disease. This study presents the first comprehensive global analysis of core and low-abundance gut microbiota, illuminating their prevalence, functional contributions, and clinical significance across diverse populations and disease contexts.

Methods

We re-analyzed 40,000 fecal metagenome samples from 150+ studies encompassing five continents, integrating healthy individuals and patients with 21 diseases. Shotgun metagenomic analysis using custom-pipeline and taxonomy classification by Kraken-v2.1.6. Machine learning was used to identify global and clinical biomarkers. Complementary clinical validation included, PacBio full-length16S rRNA sequencing of 50 *Clostridioides difficile* infection (CDI) patients and controls, core low abundance bacterial co-culture assays with *C. difficile*, and genome-scale metabolic modeling(GEM) via flux balance analysis to distinguish ecological roles of low- and high-abundance taxa.

Results

Our global gut microbiome analysis identified more than 10,000 bacterial species with vast majority occurred at relative abundance below 0.01%. Within this diversity, we defined a novel global core of 354 species nearly half classified as low abundance that consistently detected in over 70% of individuals worldwide. Longitudinal tracking showed that these low abundant core members persisted for months to years, while culture-based assays recovered strains at abundances as low as 0.00005% confirming their biological authenticity. GEM revealed distinct nutrient use niches and short chain fatty acid producers which could suppress pathogen and stabilize community structure. We also identified taxa which serves as dominant nutrient donor within microbial network. Machine learning models built on clinical datasets validated low abundance taxa as robust biomarkers for CDI and other disease states. Thirteen of these species inhibited *C. difficile* growth in vitro, a result that global CDI datasets corroborated. Overall, we prove low abundance species remained metabolically active, temporally stable, exhibited disease specific enrichment patterns and serve as potential biomarkers.

Conclusions

This large-scale study redefines the concept of a core human gut microbiome, emphasizing the ecological and clinical relevance of low-abundance species. These findings advocate for their inclusion in future microbiome-based diagnostic and therapeutic strategies and mark a paradigm shift in understanding microbial contributions to human health and disease.

SHERRY WANG

Influence Flow Channel Design and Actuation Flow Rate on Medicinal Particle Transport and Deposition in Dry Powder Inhalers: A Computational Fluid Particle Dynamics Study



Session 1 - Poster Presentation

Background

Airway drug delivery via inhalation therapy is an essential treatment for chronic lung diseases. The effectiveness of current dry powder inhalers (DPIs), e.g., Spiriva HandiHaler, is limited by low drug delivery efficiency to peripheral lung (i.e., less than 25%). A large fraction of the medication is often lost in the DPI flow channels or upper airways. Current DPIs are also highly sensitive to DPI flow channel designs and patient-device coordination, which is significantly dependent on the inhalation flow rates. The potential issue of inefficient DPIs is especially relevant to the quality of pulmonary healthcare available to rural communities, where access to quality healthcare facilities is challenging and limited. Patients who live in rural areas rely much more on unsupervised and easy-to-use DPIs for effective treatments. By optimizing DPI designs and the resultant performance (i.e., increased emitted dose from the mouthpieces of DPIs), rural populations can receive more consistent and effective treatment at home, thereby reducing the need for excessive clinical visits. Computational fluid particle dynamics (CFPD) is a cost-effective method for virtually evaluating inhaler performance, ultimately identifying optimal design features that enhance drug delivery efficiency. Specifically, CFPD can provide first-principle-based predictions of airflow velocity distributions, particle transport dynamics, and particle deposition patterns inside DPIs' flow channels, which would be much more difficult to obtain experimentally.

Methods

Employing an experimentally validated CFPD model, this study investigates how chamber size and inhalation flow rate will influence the emitted dose of a series of HandiHaler-based DPIs. Specifically, three chamber sizes (i.e., 7.25, 7.5, and 7.75 mm), three aperture/strip sizes (i.e., 1.0, 1.6, and 3.0 mm), and three inhalation flow rates (20, 39, and 60 L/min) are examined. The different chamber sizes and strip sizes are simulated on the representative airway geometry. Airflow simulations are initially run under single-phase conditions to analyze spatiotemporal distributions of airflow velocity, pressure, and shear stresses. Then, a one-way coupled Euler-Lagrange discrete phase model (DPM) is used to predict particle transport and deposition. The particle diameters simulated in this study are 1, 2, 5, and 10 μm . A total of nine case combinations of chamber and aperture size geometries are run across three flow rates to generate the comparative data mentioned above.

Results

The anticipated outcomes of this study include demonstrating a strong correlation between emitted dose and DPI design parameters as well as the inhalation flow rate. It can be hypothesized that smaller chamber sizes may result in higher airflow velocity and shear stress surrounding the capsule may potentially reduce particle deposition inside DPIs. Meanwhile, larger aperture sizes may allow particles to enter the DPI flow channels in a more scattered manner, potentially increasing the emitted dose. Higher flow rates may increase the inertial impaction and turbulence dispersion-induced particle deposition in DPI flow channels. It is expected that this study will test the above-mentioned hypothesis and provide a comprehensive understanding of airflow-particle interactions in DPIs, thereby generating insights on DPI design optimization.

Conclusions

This study aims to quantify the influence of inhaler geometric designs and inhalation flow rates on air-particle flow dynamics inside DPIs. This is particularly helpful for rural communities, which would greatly benefit from having confidence and independence in managing respiratory diseases. This study aims to make a positive contribution to equitable healthcare, including providing accessible and effective treatment for underserved populations.

EMMANUEL YANGUE

Diffusion Generative Model-enhanced Integrative Approach for Adaptive Tumor Segmentation in Ultrasound Imaging

Session 1 - Poster Presentation



Background

Accurate identification of tumor regions of interest (ROIs) in ultrasound imaging is critical for advancing image-guided drug delivery (IGDD) through ROI segmentation. However, even though ultrasound imaging stands out due to its safety, accessibility, and low cost, it may also lead to other technical challenges, such as the high levels of noisy interference (speckle), limiting boundary clarity. Moreover, manual or edge-based segmentation approaches are often inaccurate and subjective, while deep learning methods typically require large labeled datasets, lengthy training, and significant computational resources. To overcome these challenges, this study introduces a novel segmentation framework, IC-DREAM, designed to improve tumor ROI identification without reliance on annotated datasets or extensive training.

Methods

The proposed IC-DREAM integrates three complementary steps: (1) an intensity-based centroid estimation for precise ROI center identification, including support for user-defined seed input. (2) A diffusion generative model enhanced edge detection method, leveraging denoising diffusion implicit modeling (DDIM) to refine tumor boundary estimation. Finally, (3) an improved region-growing algorithm that incorporates both detected boundaries and ROI center information for robust segmentation. IC-DREAM was evaluated using both simulated ultrasound images and real-world clinical ultrasound datasets to assess segmentation accuracy, reliability, and computational efficiency.

Results

The proposed IC-DREAM framework achieved superior tumor ROI segmentation compared to conventional edge detection, manual delineation, and prior algorithmic approaches, including the previously developed B-CLEAR method. Experimental validation demonstrated that IC-DREAM significantly improved boundary delineation accuracy and segmentation robustness across diverse ultrasound datasets. Notably, the approach required unlabeled training data and operated with relatively low computational cost, highlighting its practical potential for clinical translation.

Conclusions

IC-DREAM offers an automatic identification and segmentation of ROI, specifically the tumor region, in ultrasound imaging. It operates without requiring a training process but employs three key steps for ROI segmentation: DDIM-enhanced boundary detection, intensity-based centroid estimation for ROI center locating, and an edge-assisted region growing algorithm. The experimental results validate the effectiveness of the proposed method. IC-DREAM has shown the capability to accurately and efficiently segment tumor ROIs across a variety of ultrasound images used in IGDD.

QIANRU ZHANG

Inactivation of Dual IFN Antagonists in PEDV as a Strategy for Live-Attenuated Vaccine Design

Session 2 - Poster Presentation



Background

Porcine Epidemic Diarrhea Virus (PEDV) is a coronavirus that can cause high morbidity and mortality in neonatal piglets and has resulted in significant loss to pork industry worldwide. No effective vaccine is available. PEDV encodes multiple IFN antagonists, including nonstructural proteins nsp1, nsp15, and nsp16 to antagonize the host type I and III interferon (IFN) response, which plays a critical role in controlling infection. We hypothesize that these IFN antagonists can be targeted for live-attenuated vaccine development.

Methods

Using an infectious clone of PEDV-Colorado strain, we engineered double mutants: icPEDV-nsp1+15mut and icPEDV-nsp15+16mut. We evaluated the growth kinetics, IFN induction, and IFN sensitivity of these two mutants in cell cultures. We compared the pathogenesis and antibody responses in piglets of these two mutants with the wild-type control virus.

Results

We found that both mutants exhibited impaired replication in the IFN-competent PK-1 cell line, with icPEDV-nsp15+16mut showing the most pronounced replication defect. icPEDV-nsp15+16mut also induced stronger IFN activation at 24 hpi, whereas icPEDV-nsp1+15mut induced greater IFN signaling at 36 hpi in luciferase reporter cell lines. Quantitative RT-PCR revealed significantly higher expression of IFN- β , IFN- α , IFN- λ 1, and IFN- λ 3 at 12 hpi for both mutants compared to wild-type (WT), with icPEDV-nsp15+16mut being the strongest inducer. Both mutants were more sensitive to exogenous IFN- β treatment, with icPEDV-nsp15+16mut exhibiting the highest inhibition. In piglet challenge studies, both mutants were significantly attenuated with reduced virus shedding and clinical scores compared to WT infection, while elicited comparable levels of PEDV-specific IgG and neutralizing antibodies.

Conclusions

Our studies demonstrate that simultaneous mutation of multiple IFN antagonists in PEDV elicits a more host antiviral response, resulting in attenuated viral replication and pathogenesis while preserving a strong antibody response in animals. These findings highlight the potential of these mutants as promising live-attenuated vaccine candidates.

XIAOMING ZHANG

Identification of nsp6 as key player in SARS-CoV-2 Omicron BA.1 attenuation.

Session 3 - Poster Presentation



Background

The early SARS-CoV-2 Washington strain (WA1) is highly pathogenic, while the later Omicron variant BA.1 strain is attenuated. The BA.1 genome harbors over 50 mutations. Besides the 30 mutations located within the spike gene, our early research demonstrated that the rest 20 non-spike mutations also contribute to the viral attenuation. This study aims to investigate the key mutations responsible for Omicron BA.1 unique phenotype and elucidate the underlying mechanism.

Methods

Using a reverse genetic system, recombinant virus icBA1-SWA1 containing all the non-spike mutations and recombinant viruses carrying mutations in each mutated gene were generated. To identify the key mutations contributing to the BA.1 attenuation, viral growth and plaque size were measured. Immunofluorescence staining and protein interaction analysis were performed for further investigation of possible mechanism.

Results

By generating a panel of recombinant viruses and testing their replication, we discovered that the nsp6 mutant virus exhibited reduced replication and high genome instability, with compensatory mutations rapidly emerging. Notably, the nsp6 mutant virus demonstrated increased resistance to reversion in the presence of other non-spike mutations. Additionally, a recombinant virus carrying all non-spike mutations except nsp6 exhibited similar replication to icWA1, highlighting the critical role of nsp6 mutations in BA.1 attenuation. Further analysis revealed that harboring nsp6 mutations didn't enhance interferon production and didn't alter viral sensitivity to interferon, suggesting that nsp6 mutations didn't influence the viral phenotype through the interferon signaling pathway. Interestingly, while wildtype nsp6 significantly reduced lipid droplets (LDs) staining, BA.1 nsp6 mutant had weaker effect on that. This implicates altered LDs modulation as a possible mechanism underlying the phenotypic differences associated with nsp6 mutations.

Conclusions

This study identified nsp6 as a major factor in SARS-CoV-2 replication defect and it affects the lipid droplets turnover. These findings provide insights into SARS-CoV-2 natural attenuation, which may guide the development of new antiviral strategies.

ZIYANG ZHANG

Artificial Intelligence-Empowered User-Centered Smart Inhaler for Targeted and Evenly Distributed Small-Airway Drug Delivery

Session 1 - Poster Presentation



Background

Small airways are the primary sites of airflow obstruction in chronic obstructive pulmonary disease (COPD). Effective delivery of aerosolized medications to these regions is crucial to maximize treatment efficacy while minimizing side effects. However, conventional inhalers are typically designed for general use and do not adapt to the variability among patients in respiratory patterns, airway geometries, or inhalation dynamics. Consequently, drug deposition in small airways is often not optimal and unevenly distributed across the five lung lobes. This limitation highlights the need for a personalized and adaptive inhaler design. To address this, this study proposes a machine learning (ML)-enabled inverse design framework that integrates high-fidelity computational fluid-particle dynamics (CFPD) simulations and predictive modeling to guide patient-specific inhaler nozzle optimization for improved small airway drug deposition.

Methods

A subject-specific airway geometry was reconstructed from computerized tomography (CT) scans. Using this geometry, 105 CFPD simulations were performed under varied physiological and design parameters, including inhalation flow rates, particle diameters, release locations and release timing. Particle release maps generated via backtracking identified optimal nozzle diameters and locations for uniform multi-lobe deposition with minimal off-target delivery. A dataset was compiled containing input features (flow rates, particle size, release z-coordinates, release time) and corresponding optimized nozzle parameters (release x-coordinates, release y-coordinates, nozzle diameter). 16 ML models, including fully connected regressor, dropout-regularized MLPs, CNNs, and transformer-based regressors, are trained to learn the complex nonlinear mapping between patient-specific inputs and nozzle design targets. Performance was evaluated using MSE and MAE across the overall predictions and each individual target feature.

Results

The multivariate regression-based ML models effectively learned the inverse mapping from inhalation features to optimal design parameters. Among all candidates, four top-performing models were selected due to their low MSE and MAE for both overall and feature-specific predictions. An ensemble model combining their strengths was also established. Preliminary results using predicted nozzle settings in test cases showed high potential for improving deposition uniformity and reducing off-target delivery across the lung lobes. These findings support the feasibility of ML-guided inverse design for smart inhalers.

Conclusions

This study presents a novel ML-CFPD teaming framework for inverse design of inhaler nozzle parameters, enabling adaptive and patient-specific drug delivery to small airways. By leveraging high-fidelity simulations and advanced regression models, we demonstrated that optimal nozzle configurations can be predicted directly from individual inhalation features. This study builds a methodological foundation for smart inhaler design to enhance COPD therapeutic efficacy.

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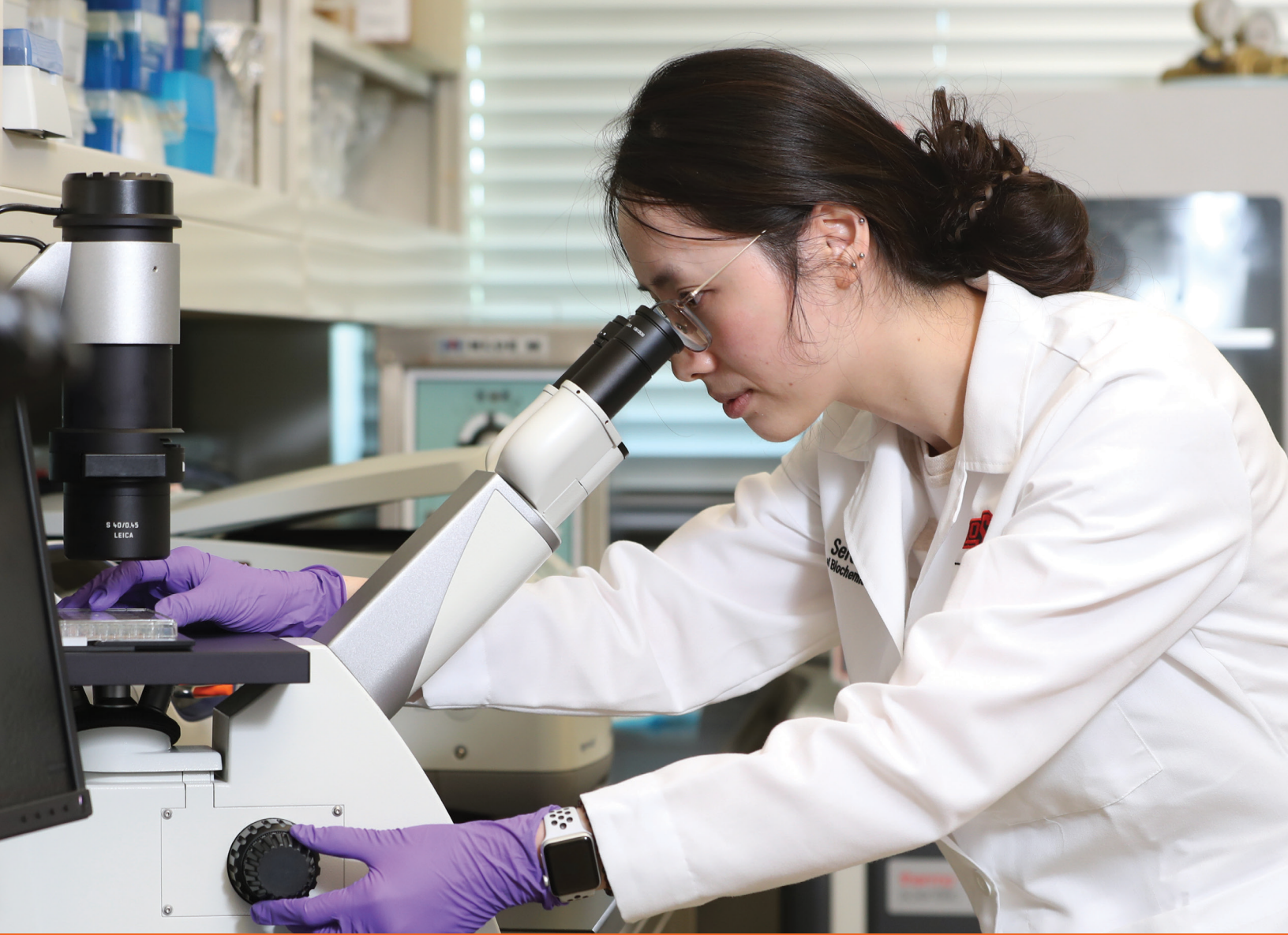
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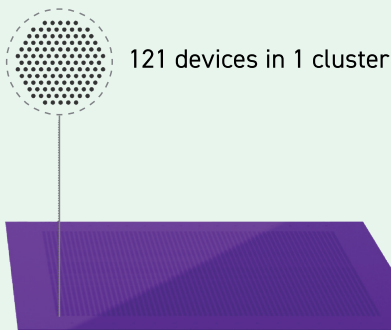


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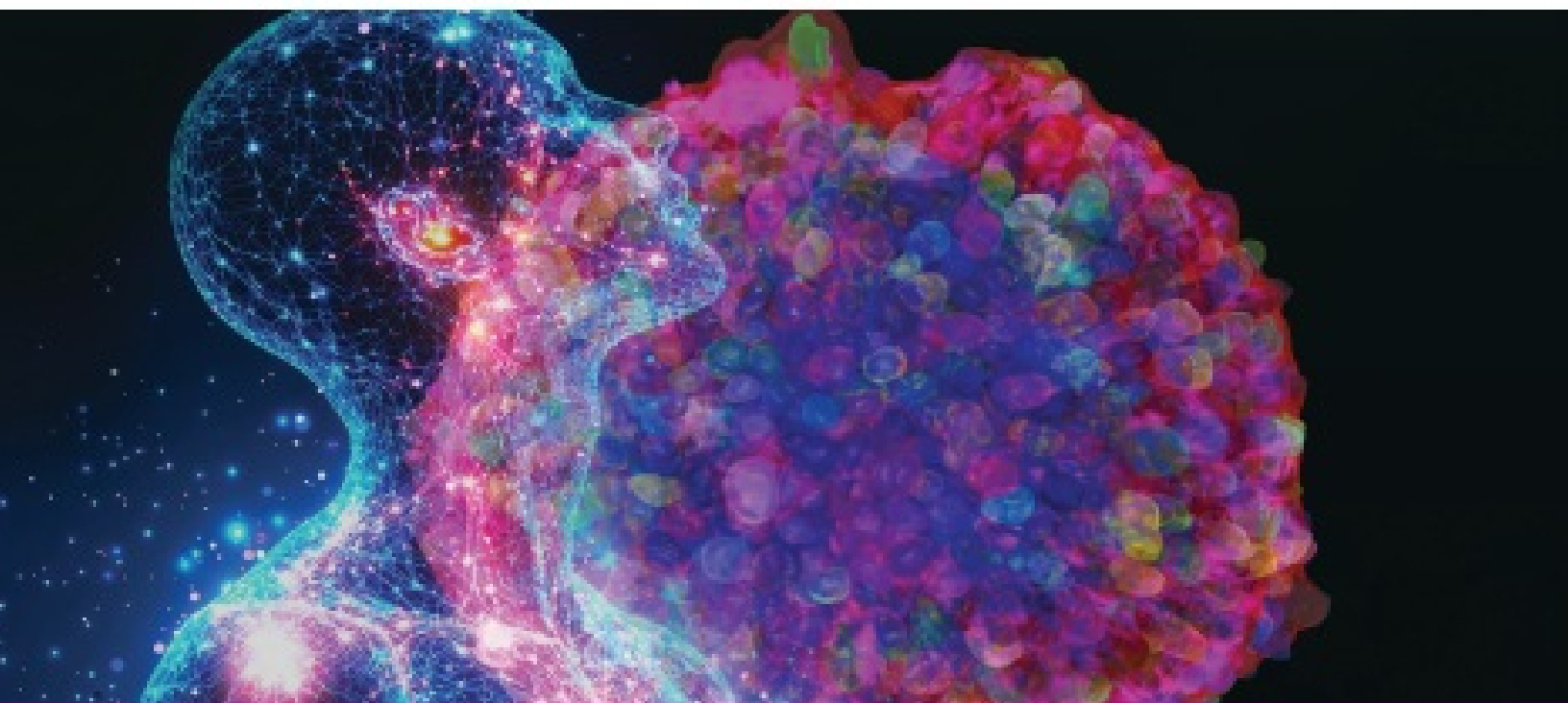
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