CARE Conference 2025

Collaborative Academic Research Excellence Conference 2025

Molecules to Medicine: Bioengineering for One Health

December 4–6, 2025

TTJ Auditorium,
IC&SR Building
Indian Institute of Technology
Madras, India

Abstract Booklet

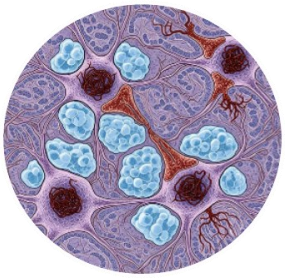
About the Conference

The Collaborative for Academic and Research Excellence conference (CARE) is an annual event started with the generous support of the Mehta Family Foundation to promote collaborations among IITs, IISERs and IISc to address the unmet challenges in frontier areas of biology. This 2.5-day program will convene distinguished scientists from around the world, industry leaders, and representatives from funding agencies, with the objective of shaping the trajectory of biomedical research in India.

Objectives

- A platform for researchers and industry experts to share the latest findings, innovations and methodologies.
- Foster partnerships across academia, industry, and funding agencies.
- Facilitate mentoring, networking, and career development for students and early-career faculty.
- Promote interdisciplinary research aligned with the One Health vision.

Themes



Disease Biology

Interventional Biology

Structural Biology

Computational Biology

Translational Biology

About the Department

The Department of Biotechnology at Indian Institute of Technology Madras, broadly encompasses three major domains: Biological Engineering, Biological Sciences, and Computational Biology, with 35 regular faculty members. Established in 2004, the Department hosts at present ~330 undergraduate students (Dual Degree Programs), ~60 Master's level students (M.Tech/MS), ~200 doctoral scholars (Ph.D.), ~30 postdoctoral scholars and project staff, and 13 technical and administrative staff. The Department offers two integrated (Dual Degree) programs, namely, BS/MS in Biological Sciences or BTech/MTech in Biological Engineering, with strong emphasis on both modern biology and engineering and on extensive practical laboratory training.

The Department also offers Master of Science (MS) by research, Doctor of Philosophy (Ph.D.), and an MTech program in Bioprocess Engineering. The Department has made significant strides in positioning itself as one of the best centres of excellence in the Biotechnology field in the past decade. The Department is generously supported by Mehta Family Foundation.

About the Mehta Family Foundation

Originally from Mumbai, India, Rahul Mehta is the Founder and Chairman of the Board of the Bhupat & Jyoti Mehta Family Foundation. He established the Foundation in 1996 after successfully exiting his first startup and has since dedicated himself to advancing higher education and building research ecosystems around the world.

The Bhupat & Jyoti Mehta Family Foundation (MFF), one of the largest philanthropic collaborators in higher education in India, has been a visionary and catalyst in introducing areas with transformative potential to the Indian higher education ecosystem. What began in 2006 with a single School of Biosciences at IIT Madras has grown into a national initiative, creating intellectual talent in cutting-edge domains like Biomedical Engineering, Data Science and AI, and Sustainability.

Under Rahul's leadership, the Mehta Family Foundation is guided by the belief that investment in science drives economic prosperity. This vision has led to the establishment of eight schools across six prestigious Indian Institutes of Technology (IIT) in India, spanning bioscience and bioengineering, data science and artificial intelligence, and sustainability. Together, these programs have created a capacity for more than 3,000 students across 620,000 square feet of infrastructure. The Foundation has also funded more than 100 nonprofits/NGOs, created the Mehta Rice Engineering Scholarship program with Rice University, and supported numerous initiatives at the University of Houston.

Rahul has served on the boards of several Houston-area nonprofits, including DePelchin Children's Center, Ronald McDonald House, Bo's Place, American Leadership Forum, Krist Samaritan Center, and Houston Methodist Hospital. A lifelong learner, he is a 2021 Stanford Distinguished Careers Institute Fellow and a 2024 Oxford Next Horizons Scholar. He has completed executive education programs at Stanford, Wharton, Harvard, the University of Chicago, and Rice.

An avid traveler who has explored every continent, Rahul remains devoted to the development of humanity and endeavors that his lifelong commitment will leave a meaningful legacy in the lives of others.

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ORGANISING COMMITTEE

Sanjib Senapati (HOD, Convenor)

Greeshma Thrivikraman (Co-convener)

S. Mahalingam (Co-convener)

Santhosh Sethuramanujam (Co-convener)

Prof. Amal Kanti Bera

Dr. Arumugam Rajavelu

Prof. Himanshu Sinha

Dr. Mamata Bangera

Dr. Meiyappan Lakshmanan

Dr. Nathiya Muthalagu

Dr. Ninitha AJ

Dr. Richa Karmakar

STAFFS

Jayaraman M

Lekshmi A K

MadhanKumar S

Sneha Sathapathi

Swathy R

Varsha Selvachandran

Vengadeshkumar V

STUDENT VOLUNTEERS

Aadit Mahajan
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Gayathrinanda Prakash
Harikrishna
Ishaan Yadav
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Kavin Chakravarthy
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Ramakrishna Reddy
Roshni Raj
Sandhya N
Satrajit Das
Shankhanava Ghosh
Sharad Js
Shivan Ajay Iyer
Sourav Nandi
Sunita Pradhan
Surya Prakash Tiwari
Tanjot Kaur
Tarun Shyam Mohan
Vikash Gokul

CONTENT

PROGRAMME SCHEDULE

DAY 1 – DEC 4TH 2025

9:00 – 9:30	***Inauguration***	
9:30 – 10:45 **Chairs:** **Athi Naganathan,** *IIT Madras* *&* **Sharad Gupta,** *IIT Gandhinagar*	**SESSION 1: STRUCTURAL BIOLOGY I** **Helmut Grubmuller** Max Planck Institute, Fassberg (Germany) ***Title:*** *Towards Room Temperature Macromolecular Structure Ensembles: Freezing Ribosomes And Single Molecule X-Ray Diffraction* **Giulia Palermo** University of California Riverside (USA) ***Title:*** *Advancing Gene Editing through Computational Methods and Deep Learning* **Shruthi Viswanath** National Center for Biological Sciences (India) ***Title:*** *Integrative Structure Determination of Sub-complexes of the Mitochondrial Contact Site And Cristae Organizing System (MICOS)*	 Plenary Lecture 9:30 – 10:00 Keynote Lecture 10:00 – 10:25 Invited Lecture 10:25 – 10:45
10:45 – 11:15	***Tea Break***	
11:15– 12:30 **Chairs:** **Amal K Bera** *IIT Madras* *&* **Jagadeesh Bayry,** *IIT Palakkad*	**SESSION 2: DISEASE BIOLOGY I** **Vinay Nandicoori** Center for Cellular and Molecular Biology (India) ***Title:*** *Delineating the survival strategies employed by Mycobacterium tuberculosis* **Manidipa Banerjee** Indian Institute of Technology Delhi (India) ***Title:*** *Dynamics of Plasmodium Enzyme Complexes From Native Cryo-EM Structures* **Soham Chanda** Colorado State University (USA) ***Title****: Modeling Synapse Development and Dysfunction with Stem Cell-Derived Human Neurons*	 Plenary Lecture 11:15 – 11:45 Keynote Lecture 11:45 – 12:10 Invited Lecture 12:10 – 12:30
12:30 – 13:15	***Lunch***	
13:15 – 14:30	***Poster Session I***	***Connect to Post-Doc Mentors*** *(Invited Participants Only)*

14:30 – 15:45	**SESSION 3: INTERVENTIONAL BIOLOGY I**	
Chairs: **Vignesh Muthuvijayan** *IIT Madras* *&* **B.S. Balaji** *JNU*	**Abhay Pandit** University of Galway (Ireland) ***Title:*** *Exploring the Regenerative Processes Initiated by Biomaterial Systems: Emphasizing The Significance of Glycosylation*	Plenary Lecture 14:30 – 15:00
	Kristopher Kilian University of New South Wales (Australia) ***Title:*** *A Bioengineering Approach To Next-Gen Micro Physiological Systems*	Keynote Lecture 15:00 – 15:25
	Shamik Sen Indian Institute of Technology Bombay (India) ***Title:*** *Glycocalyx in Cancer Invasion & Drug Resistance*	Invited Lecture 15:25 – 15:45
15:45 – 16:15	***Tea Break***	
16:15– 17:30	**SESSION 4: TRANSLATIONAL BIOLOGY I**	
Chairs: **Nitish Mahapatra** *IIT Madras* *&* **Utpal Bora** *IIT Guwahati*	**Maneesha Inamdar** inSTEM Bangalore (India) ***Title:*** *Guiding Innovation Towards Therapy, With Stem Cells And Organoids*	Plenary Lecture 16:15 – 16:45
	Arumugam Rajavelu Indian Institute of Technology Madras (India) ***Title:*** *Epigenetically Regulated Parasites' RIFINs Contribute To Severe Malaria Pathogenesis*	Keynote Lecture 16:45 – 17:10
	Charlotte Steenblock Technische Universität Dresden (Germany) ***Title:*** *Emerging Cell Therapies for Type-1 Diabetes and Adrenal Insufficiency: From Bench to Bedside*	Invited Lecture 17:10 – 17:30
17:30 -18:30	**Panel Discussion/ Academia-Industry** **Malathi S** *(Pfizer)* **Kaushik Ghosh** *(Bristol-Myers Squibb)* **Girish S. Ratnaparkhi** *(IISER Pune)* **S. Mahalingam** (*IIT Madras)* **Smita Srivastava** *(IIT Madras)- Moderator*	
19:00 – 22:00	**Interaction Session over Dinner**	

DAY 2 – DEC 5TH 2025

9:00 – 10:15	**SESSION 5: SYSTEMS BIOLOGY I**	
Chairs: **Himanshu Sinha** *IIT Madras* *&* **Pinaki Sar** *IIT Kharagpur*	**Shankar Subramaniam** University of California San Diego (USA) ***Title:*** *A spatial and tissue digital landscape of triple negative breast cancer – diagnosis to therapy*	Plenary Lecture 9:00 – 09:30
	Sangram Bagh Saha Institute of Nuclear Physics (India) ***Title:*** *Towards AI Bacteria*	Keynote Lecture 09:30 – 09:55
	Kartik Sunagar Indian Institute of Science Bangalore (India) ***Title:*** *Indian Snake Venoms: Ecological Drivers and Therapeutic Innovation*	Invited Lecture 09:55 – 10:15
10:15 – 10:45	***Tea Break***	
10:45– 12:00	**SESSION 6: STRUCTURAL BIOLOGY II**	
Chairs: **N. Manoj** *IIT Madras* *&* **Rukmankesh** *IIT Bhilai*	**Naoki Sugimoto** Konan University (Japan) ***Title:*** *"To B or not to B" in Nucleic Acids Chemistry*	Plenary Lecture 10:45 – 11:15
	Saikrishnan Kayarat IISER Pune (India) ***Title:*** *Mechanism Of DNA Shredding By The Innate Immune Systems of ATP-Dependent Nucleases*	Keynote Lecture 11:15 – 11:40
	Aravind Penmatsa Indian Institute of Science Bangalore (India) ***Title:*** *NETs and GATs: Countering Pain Through Inhibitory Neurotransmission*	Invited Lecture 11:40 – 12:00
12:00 – 12:30	***Communicating your research outside the lab***	***HOD Meeting (11:30 – 12:30)***
12:30 – 13:15	***Lunch***	
13:15 – 14:30	***Poster Session II***	***Road to Academia*** *(Invited Participants Only)*

14:30 – 15:45	**SESSION 7: SYSTEMS BIOLOGY II**	
Chairs: **Michael Gromiha** *IIT Madras* *&* **Parimal Kar** *IIT Indore*	**Ananth Grama** Purdue University, USA ***Title:*** *Learning Across Modalities: From Digital Pathology to Molecular Design*	Plenary Lecture 14:30 – 15:00
	Anand B Indian Institute of Technology Guwahati (India) ***Title:*** *Bacterial Cell Growth and Dormancy: An Interplay Between Ribosome Biogenesis And Stringent Response Pathway*	Keynote Lecture 15:00 – 15:25
	Tapomoy Bhattacharjee National Centre for Biological Sciences (India) ***Title:*** *TBA*	Invited Lecture 15:25 – 15:45
15:45 – 16:15	***Tea Break***	
16:15– 17:30	**SESSION 8: DISEASE BIOLOGY II**	
Chairs: **Vani Janakiraman** *IIT Madras* *&* **Prosenjit Mondal** *IISER Berhampur*	**Catherina G. Becker** Dresden Technical University (Germany) ***Title:*** *Fishing for Success! Spinal Cord Regeneration in the Zebrafish*	Plenary Lecture 16:15 – 16:45
	Arnab Mukhopadhyay National Institute of Immunology (India) ***Title:*** *A Diet-Gene Interaction That Targets Mitochondria To Modulate Longevity*	Keynote Lecture 16:45 – 17:10
	Nikhil R. Gandasi Indian Institute of Science Bangalore (India) ***Title:*** *Islet Cell Secretory Mechanisms: What Are Drivers That Are Dysregulated During Diabetes*	Invited Lecture 17:10 – 17:30
17:30 -18:30	**Panel Discussion/ Academia-Industry** **Apurva Sarin** *(Wellcome Trust)* **Harpreet Singh** *(ICMR)* **A. V. Balachandar** *(ANRF)* **Suresh Rayala** (*IIT Madras)* **Himanshu Sinha** *(IIT Madras) - Moderator*	
19:00 – 20:00	***Cultural Evening by Kalakshetra***	
20:00 – 22:00	***Dinner***	

DAY 3 – DEC 6TH 2025

9:00 – 10:15	**SESSION 9: INTERVENTIONAL BIOLOGY II**	
Chairs: **V. Kesavan** *IIT Madras* *&* **Jac Fredo Agastinose Ronickom** *IIT BHU*	**Amit Singh** Indian Institute of Science, Bangalore (India) ***Title:*** *Bioenergetic Crosstalk between Host and Pathogen Mediates Drug Tolerance in Mycobacterium tuberculosis*	Plenary Lecture 9:00 – 9:30
	Ramandeep Singh Translational Health Science and Technology Institute, India ***Title:*** *Identification Of Novel Therapeutic Interventions Against Mycobacterium tuberculosis*	Keynote Lecture 9:30 – 9:55
	Arnab Mukherjee University of California Santa Barbara ***Title:*** *Programmable genetic sensors for deep-tissue imaging*	Invited Lecture 9:55 – 10:15
10:15 – 10:45	***Tea Break***	
10:45– 12:00	**SESSION 10: TRANSLATIONAL BIOLOGY II**	
Chairs: **K. Chandraraj** *IIT Madras* *&* **Supratim Datta** *IISER Kolkata*	**Michele Solimena** Dresden Technical University (Germany) ***Title:*** *Advanced Imaging Unveils The Progressive Demise Of Islet Cells From Euglycemia To Type-2 Diabetes And Back*	Plenary Lecture 10:45 – 11:15
	Pradip Kumar Sasmal Morepen Drug Research Pvt. Ltd. (India) ***Title:*** *Molecules to Medicine: Challenges and Opportunities*	Keynote Lecture 11:15 – 11:40
	Meiyappan Lakshmanan Indian Institute of Technology Madras (India) ***Title:*** *Systems Approaches For Animal Cell Culture Media Development In Biomanufacturing*	Invited Lecture 11:40 – 12:00
12:00 – 13:00	***Prize Distribution and Concluding Remarks***	
13:00 – 14:00	***Lunch***	

SPEAKERS

MAIN SPEAKERS

Prof. Helmut Grubmüller

Professor and Director of the Department of Theoretical and Computational Biophysics, Max Planck Institute for Multidisciplinary Sciences, Göttingen, Germany

Dr. Giulia Palermo

Associate Professor, Department of Bioengineering and Chemistry, University of California, Riverside, USA

Dr. Shruthi Viswanath

Assistant Professor, National Centre for Biological Sciences, Tata Institute of Fundamental Research

Prof. Vinay Nandicoori

Director, Centre for Cellular and Molecular Biology, Hyderabad

Prof. Manidipa Banerjee

Professor, Kusuma School of Biological Sciences
PI, Structural and Molecular Virology Group
IIT Delhi

Dr. Soham Chanda

Assistant Professor, Department of Biochemistry and Molecular Biology Colorado State University, USA

Prof. Abhay Pandit

Professor of Biomaterials, Founding Director of CÚRAM (Centre for Research in Medical Devices), University of Galway, Ireland

Prof. Kristopher (Kris) Kilian

Professor, School of Chemistry and Materials Science & Engineering

University of New South Wales, Australia

Prof. Shamik Sen

Department of Biosciences and Bioengineering
IIT Bombay

Prof. Maneesha Inamdar

Director, BRIC Institute for Stem Cell Science and Regenerative Medicine

Professor, JNCASR, Bangalore

Dr. Arumugam Rajavelu

Associate Professor, Department of Biotechnology
IIT Madras

Dr. Charlotte Steenblock

Research group leader, Dept. of Internal Medicine III

Technische Universität Dresden, Germany

Prof. Shankar Subramaniam

Professor, Bioengineering and Computer Science
University of California, San Diego, USA

Dr. Sangram Bagh

Associate Professor, Biophysics and Structural Genomics Division
Saha Institute of Nuclear Physics

Dr. Kartik Sunagar

Associate Professor, Centre for Ecological Sciences
PI, Evolutionary Venomics Lab
Indian Institute of Science, Bangalore

Prof. Naoki Sugimoto

Frontier Institute for Biomolecular Engineering Research (FIBER)
Konan University, Kobe, Japan

Prof. Saikrishnan Kayarat

Professor, Department of Biology
Indian Institute of Science Education and Research Pune

Dr. Aravind Penmatsa

Assistant Professor, Molecular Biophysics Unit
Indian Institute of Science

Prof. Ananth Grama

Professor, Department of Computer Science
Associate Director, Center for Science of Information
Purdue University, USA

Prof. Anand B

Professor, Department of Biosciences and Bioengineering
Principal Investigator, Mechanistic Approaches to Biology Lab
IIT Guwahati

Dr. Tapomoy Bhattacharjee

Assistant Professor, National Centre for Biological Sciences
Tata Institute of Fundamental Research

Prof. Catherina G. Becker

Professor and Chair of Neural Development and Regeneration
Director at the Center for Molecular and Cellular Bioengineering (CMCB) & Deputy Director at the Center for Regenerative Therapies Dresden (CRTD)
Technische Universität Dresden, Germany

Dr. Arnab Mukhopadhyay

Faculty, National Institute of Immunology, Delhi

Dr. Nikhil R Gandasi

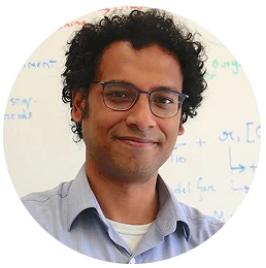
Assistant Professor, Developmental Biology and Genetics
Indian Institute of Science, Bangalore

Prof. Amit Singh

Centre for Infectious Disease Research (CIDR) & Department of Microbiology and Cell Biology (MCBL)
Indian Institute of Science, Bangalore

Prof. Ramandeep Singh

Senior Professor, Translational Health Science and Technology Institute



Dr. Arnab Mukherjee

Associate Professor, Department of Chemical Engineering
University of California, Santa Barbara, USA

Prof. Michele Solimena

Paul Langerhans Institute Dresden
Professor of Molecular Diabetology, Technische Universität Dresden

Dr. Pradip Kumar Sasmal

Chief Scientific Officer
Morepen Proprietary Drug Research

Dr. Meiyappan Lakshmanan

Assistant Professor, Department of Biotechnology
IIT Madras

PANELISTS

Dr. Malathi S *(Pfizer)*

Dr. Kaushik Ghosh *(Bristol-Myers Squibb)*

Dr. Girish S. Ratnaparkhi *(IISER Pune)*

Prof. S. Mahalingam *(IIT Madras)*

Prof. Smita Srivastava *(IIT Madras)*- **Moderator**

Prof. Apurva Sarin *(Wellcome Trust)*

Dr. Harpreet Singh *(ICMR)*

Dr. A. V. Balachandar *(ANRF)*

Prof. Suresh Rayala *(IIT Madras)*

Prof. Himanshu Sinha *(IIT Madras)* – **Moderator**

FLASH TALKS

ROAD TO ACADEMIA

S.No	Poster ID	Name	Title
1	PANIIT20251118	Dr. Pravesh Gupta	Multi-omics immune microenvironment interrogation of human brain malignancies identifies anti-tumor phagocytic immunomodulators.
2	PANIIT20250976	Dr. Bhavani Shankar Sahu	Dense core vesicles- Biogenesis, trafficking, and physiological functions
3	PANIIT20250960	Dr. Tanmay Mitra	Rewiring the Immune Synapse with T-cell Engagers: How Flexibility, Membrane Mechanics, and Synaptic Architecture Shape T-Cell Engager Potency
4	PANIIT20250947	Dr. Pradeep Kumar Singh	Lecanemab blocks the effects of the Aβ/fibrinogen complex on blood clots and synapse toxicity in organotypic culture
5	PANIIT20250943	Dr. Prabahan Chakraborty	From circuits to behaviour: understanding how danger and diet affects cognition
6	PANIIT20250913	Dr. Akhil Pratap Singh	Endosomal Escape of pH-Sensitive Lipid Nanoparticle-Based Drug Carriers: From Poration to Transfection

CONNECT TO POST-DOC MENTORS

S.No	Poster ID	Name	Title
1	PANIIT20250140	Agnibina Paul	Structural Impact of Cancer-Associated Mutations in Calnuc (NUCB1) and Their Functional Consequences
2	PANIIT20250124	M S Ananthakrishna Tantry	Tubulin Isotypes for the Rescue: Decoding 5-Hydroxythalidomide Neurotoxicity and Functional Redundancy Among β-Tubulins in tubb5 Mutants
3	PANIIT20250108	Sambhavi Pattnaik	Tunneling nanotubes as a therapeutic target for glioblastoma
4	PANIIT20250107	Aakriti Singh	G-Quadruplex structures within the hfq gene regulate RNA-protein interactions in Acinetobacter baumannii
5	PANIIT20250100	Digvijay Lalwani Prakash	SuBMIT: A Toolkit for Facilitating Simulations of Coarse-Grained Structure-Based Models of Biomolecules.
6	PANIIT20250959	Dhruv Kumar Chaurasiya	Differential Long-Range Thermodynamic Coupling Governs Slow and Fast Protein Dynamics
7	PANIIT20250956	Avinash Dakshinamoorthy	Understanding the mechanism of action of HIV Protease dimerization inhibitors
8	PANIIT20250925	Meenakshi Kandpal	From Gut to Brain: Helicobacter pylori-Mediated Dysbiosis in the Pathogenesis of Neurodegeneration
9	PANIIT20250921	Beauty Rani Koch	Title- ERK-mediated phosphorylation controls stability of cardiac poly(A) polymerase Star-PAP to regulate myocyte hypertrophy
10	PANIIT20250906	Sreepadmanabh M	Physico-chemical regulation of living matter across scales
11	PANIIT20250105	Monica Alfred	Rapid somatic HCN dysfunction in epileptogenesis induces hyperexcitability in subicular burst firing neurons
12	PANIIT20250938	Pramod Patidar	TIRAP–c-JUN Signaling Links Macrophage–Hepatic Stellate Cell Crosstalk in Alcohol-Induced Liver Fibrosis

POSTER SESSION

Day 1

S.No	Poster ID	Name	Title
1	PANIIT20250928	Aynal Hoque	A quadrangle π-π and π-H interdomain interaction contributes to the voltage-dependent facilitation (VDF) of L-type voltage-gated calcium channels
2	PANIIT20250930	Tejaswini Ganapathy	Genome-scale reconstruction of the chicken enables cell culture media design and toxicity assessment in cultivated meat manufacturing
3	PANIIT20250932	Seemanti Aditya	PIGBOS1 is a Novel Regulator of Calcium Dynamics and Mitochondrial Function
4	PANIIT20250939	Thasni Fazil	Impact of TUBB4B Tubulin Isotype Mutation on Microtubule Dynamics.
5	PANIIT20250940	Abhijith K	Preclinical Safety Studies With Gene-Edited Hematopoietic Stem And Progenitor Cells For The Treatment Of B-Hemoglobinopathies
6	PANIIT20250941	Prasmita Paul	Identification and characterisation of small RNA regulators in *Helicobacter pylori* infection and pathogenesis
7	PANIIT20250944	Nitya Nandkishore	From Embryos to Organoids: Modelling Development to Decode Disease
8	PANIIT20250949	Shilpi Laha	Tug-of-War between Stability and DNA-Binding Modulates Phase Separation in Yeast HMG Proteins
9	PANIIT20250951	Ms. Yashvi Soni	Next-Generation β-Glucosidases : Enhancing Stability and Glucose Tolerance Therapeutic Biocatalysts.
10	PANIIT20250952	Purvi Trivedi	WGS driven predictive combination of antibiotics for precision-based therapy in Drug-resistant *Acinetobacter baumannii*
11	PANIIT20250954	Aravindha Anjana P	Evaluating the effect of CHO endogenous promoters and enhancers to improve expression efficiency and stability
12	PANIIT20250955	Ashutosh Sahoo	Comparative genomics guided discovery of pathway targets against Multidrug-Resistant *Mycobacterium tuberculosis* and screening of essential oil phytochemicals
13	PANIIT20250957	Sudipta Nandi	Mechanistic Insights and Fragment-Based Evolutionary Design: Next-Generation CETP Inhibitors for Cardiovascular Therapeutics
14	PANIIT20250958	Radhika K	HOCl-Induced Oxidative Stress for Improvement of Phycocyanin Production in *Synechococcus elongatus UTEX2973* under Simulated Microgravity Conditions

15	PANIIT20250961	Ananya Asmita	Ionic Liquids as Molecular Stabilizers for Nucleic Acids: Achieving Long-Term Structural and Functional Stability at Room Temperature
16	PANIIT20250962	Amit Kumar	Success rate and variability of Diabetes induction by High-Fat Diet in male C57BL/6J Mice
17	PANIIT20250963	Tamanna Saini	Role of VPS52 in Neurodevelopment And Membrane Trafficking
18	PANIIT20250972	Supratim Saha	Temporal Shear Dynamics as an Independent Regulator of Epithelial Mechanotransduction in Straight Microchannels
19	PANIIT20250973	Puyam Milan Meitei	Crude root extracts of *Sida cordifolia* rescues motor, cognitive, and neuropathological deficits in Huntington's Disease Mouse Model by ameliorating mHTT protein aggregates
20	PANIIT20250974	Shreya Kumari	Identification of a novel pathway against HD disease using a naturally inspired compound
21	PANIIT20250975	Palki Chauksey	Beyond the Vesicular trafficking: Novel role of Chromogranin B in regulation of Autophagy and Proteostasis
22	PANIIT20250978	Mohima Mukherjee	Syntaxin 6 orchestrates dense-core vesicle function through coordinated regulation of vesicle biogenesis, cargo sorting, and exocytosis.
23	PANIIT20250980	Krittika Biswas	VAMP-2 SUMOylation causes impaired dense-core vesicle exocytosis in obesity-associated metabolic stress
24	PANIIT20250982	Souradipta Chakraborty	Melatonin Protects Cardiac Tissue from Chemotherapy-Induced Oxidative and Structural Damage in Wistar rats
25	PANIIT20250983	Sarmistha Sarkar	Melatonin as a protective adjuvant against doxorubicin-induced cardiotoxicity and ensuing perturbations in cardiac riboflavin metabolism
26	PANIIT20250984	Narendra Pratap Singh	Inhibitory Circuits in Retina Are Critical For Image Stabilization During Global Motion
27	PANIIT20250985	Alisha Wadud Mondal	Harnessing Intrinsic Glutaraldehyde-Protein Fluorescence for Breakthrough Sensing
28	PANIIT20250987	Subashree Anand	The Role of Lactate in Tunneling Nanotube Formation in Glioblastoma Cells
29	PANIIT20250989	Hanna Fathima	Dietary Trans Fatty Acids Exacerbate Chronic Stress–Induced Cognitive and Behavioral Impairments in Zebrafish
30	PANIIT20250990	Roshini Mohan	ROS-responsive hydrogel targeting Intervertebral Disc Degeneration

31	PANIIT20250991	Bhavana Ramachandran	Analysis of Eccrine Sweat in Women During Menstrual Cycle: An Exploratory Proteomic Study
32	PANIIT20250992	Diksha Mall	Lab-on-chip device for rapid isolation and detection of bacteria for early sepsis detection
33	PANIIT20250993	Subhranwita Mallik	Dendritic integration mechanisms preferentially enhance the OFF signals in ON-OFF direction-selective retinal ganglion cells
34	PANIIT20250994	Neha Rani Das	Rapid DNA Extraction Method for Point-of-Care Diagnostics
35	PANIIT20250995	Dr. Subhashree Subhasmita Nayak	Computer-aided drug designing strategies for adjuvant therapy to eliminate Mycobacterium tuberculosis and in vitro validation
36	PANIIT20250996	Amrutha Arjunan	Clinicopathological and Functional Characterization of RASSF9 in Gastric Cancer
37	PANIIT20251125	Pon Yazhine Tamilselvan	Zidovudine Delivery and Mitigation of Hepatotoxicity through Glycan-Engineered Stem Cell–Derived Exosomes: An In Vitro Study
38	PANIIT20251693	Akhilesh Kumar Mishra	Deciphering the role of tyrosine phosphorylation sites within the catalytic domain of PTP-PEST on its activity and endothelial cell functions.
39	PANIIT20250109	Gautam Mohapatra	Possibility of Using Lactate Receptor (GPR81) Agonist for Treating Drug Resistant Epilepsy

Day 2

40	PANIIT20250101	Sunanda Samanta	Syk in Motion - Loop-Guided Control of the Conformational Landscape and Its Modulation by Mutations
41	PANIIT20250102	Lopamudra Parida	Comprehensive characterization and functional insights into septin genes of *Labeo rohita*: molecular basis of immune modulation and development
42	PANIIT20250103	Aparna M	From Cells to Meat: Integrating Cells and Metabolic Modelling for Cultivated Mutton Manufacturing
43	PANIIT20250106	Rituparna Sahu	Selective activator of calcium-activated potassium channels, NS-309 acts as effective seizure reducing agent by restricting the hyperexcitability phenomenon in acquired epilepsy
44	PANIIT20250110	Kashmiri M Lande	Role of GTPase in adrenergic modulation of sAHP-generating IKCa and L-type Cav1.3 channels in hippocampal pyramidal neurons
45	PANIIT20250111	Ramya D	In Situ Mineralized Bone Microenvironment For Drug Screening Applications
46	PANIIT20250112	Arthi N	Twisted ECM and polymer yarn for cardiac tissue regeneration
47	PANIIT20250113	Evan Debnath	Reformulating drugs for enhanced solubility & lower dose requirements
48	PANIIT20250115	Srutimanjari Parida	To study if PAK1 (p21 activated kinase) phosphorylation of AHNAK protein modulates blood-brain barrier integrity
49	PANIIT20250121	Tripti Sharma	Aluminium-Induced Reproductive Toxicity and Estrogen Receptor Gene Expression Alterations: A Possible Forerunner to Neurodegenerative Pathology in Female Wistar Rats
50	PANIIT20250122	Velvili S	Investigation of the Function of Epigenetic Reader Proteins in Malarial Parasites
51	PANIIT20250123	Jissmole Lukose	Elucidating the role of unique Epigenetic modifications in *Plasmodium falciparum*
52	PANIIT20250126	Dhanaraj R	Investigating the deregulated miRNAs and their expression in differentiated thyroid cancer of South Indian ethnicity
53	PANIIT20250127	Nachiket Joshi	Identification of Novel ATP-site GSK-3β Inhibitors by Employing Molecular Modeling Approaches and Free-Energy Calculations

54	PANIIT20250128	Raghul Kannan S	N-terminal acetylation affects mitochondrial function and mitochondrial biogenesis in alcohol-induced neurotoxicity
55	PANIIT20250129	Anandhi Kalaiyazhagan	Molecular Screening of Genes Associated with Differentiated Thyroid Cancer in the South Indian Population: A Case Control Study.
56	PANIIT20250132	Agnes Mary S	Studies on Crosslinking Efficiency of Bovine Pericardium Using Terminalia Chebula Extract for tissue engineering applications
57	PANIIT20250133	Rajesh Das	RAS Effector, RASSF10, Modulates Cancer Cell Metabolism
58	PANIIT20250134	Jithin P. V.	RASSF7: A novel transcription factor orchestrating oncogenic signalling and tumour dynamics
59	PANIIT20250135	Sujoy Sow Mondal	RAS Effectors Orchestrating MYC Activity: Molecular Insights Into Tumorigenesis
60	PANIIT20250136	Bhatt Baby Bhavana	Computational profiling of Chromosome 1 – A comprehensive search for cancer biomarkers
61	PANIIT20250138	Dhyaneshwar M	Targeted Nisosomes protect the heart from doxorubicin-induced injury: Evidence from Oxidative stress, pyroptosis and apoptosis pathways
62	PANIIT20250139	Paras Gautam	Bioinformatic and Biochemical Characterization of Putative Esterase/Lipase SsoEst5 from *Sulpholobus solftaricus*
63	PANIIT20250141	Sachin Mishra	Molecular and Biophysical Insights into the Structure and Function of the Single-stranded DNA-binding Protein from *Pseudomonas aeruginosa*
64	PANIIT20250143	Sunanda Gautam	Structure and function insights into the DnaB helicase of *Pseudomonas aeruginosa*
65	PANIIT20250144	I P Latha Laxmi	Metabolic profiling in human postmortem brain under the influence of alcohol use
66	PANIIT20250146	Anushka Banerjee	Natural Killer cell immunotherapy for augmentation of immune synapse formation and degranulation against leukaemia.
67	PANIIT20250147	Srayasi Majee	Therapeutic potential of human macrophage Piezo1 ion channel modulation ameliorating atherosclerosis progression.
68	PANIIT20250148	Razia Khatoon	Melatonin Protects Against T3-Induced Cardiac Hypertrophy Through Restoration of Antioxidant Defence and Mitochondrial Integrity

69	PANIIT20250909	Kokilaramani Seenivasan	Rust Meets Roots: Plant-Derived Inhibitors Against the Protein Language of Corrosion
70	PANIIT20250910	Roshan Balaji	NovoMolGen: Rethinking Molecular Language Model Pretraining
71	PANIIT20250911	Sukanya Naik	Advancing Protein-Ligand Binding Site Prediction Using Attention-Enhanced 3D Graph Neural Networks
72	PANIIT20250914	Dr Souradyuti Ghosh	Investigating a bioconjugation-free glucometer-enabled readout for hemin DNAzyme transducer and application in a biosensor
73	PANIIT20250918	Aishwarya Murali	Exploring the ribosomal protein gene expression signatures as lineage-dependent markers of gastric cancer progression
74	PANIIT20250919	Amrish P	Unlocking fungal pharmacogenomics: A systems-level approach to mapping the genetic basis of multiple drug-drug interactions
75	PANIIT20250920	Puja Laxmanrao Shinde	Traditional Medicine Meets Modern Mechanism: Amalaki Rasayana Modulates Galectin-3-C-Epitope Oligomerization in Cardiac Hypertrophy
76	PANIIT20250922	Namrata Britto	Computational Screening And Molecular Docking Of Phytochemicals For Therapeutic Intervention In Arthritis
77	PANIIT20250923	Prachi Bhargava	Binding-induced transitions in intrinsically disordered regions in protein-protein and protein-nucleic acid complexes
78	PANIIT20250927	Vijaya P.P	Cellular and chromosomal interaction of bio-synthesized copper oxide nanoparticles - Induced nano-cytotoxicity and genotoxicity
79	PANIIT20251694	Punita Kumari	Membrane Lipids as Key Regulators of GPCR Dynamics

ABSTRACTS

MAIN SPEAKERS

Towards room temperature macromolecular structure ensembles: Freezing ribosomes and single molecule X-ray diffraction

Lars Bock[1], Steffen Schultze[1], Helmut Grubmüller[1]

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Abstract:

We explore two routes to room biomolecular structure ensembles. First, non-equilibrium shock-freeze simulations of solvated ribosomes reveal how much of the room temperature structural ensemble of these RNA/protein complexes is preserved during plunge-freezing in single particle cryo-electron microscopy experiments [1,2]. For these specimen, we estimate that ca. 20-30% of their room temperature structural heterogeneity is lost upon plunge freezing. Second, using a rigorous Bayesian approach, we show how protein structures and structure ensembles can be determined from single molecule X-ray scattering experiments using ultrashort free electron laser pulses; we show from synthetic scattering simulations that it should be possible to resolve medium-sized proteins *de novo* to 2Å resolution with as few as 15 recorded photons per scattering image [3,4]. Scattering experiments on DNA origami demonstrate the feasibility of this approach, albeit at present at lower resolution.

References:

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[2] Bock LV, Igaev M, Grubmüller H. Curr. Op. Struct. Biol. 86, 102825 (2024)
[3] Schultze S, Grubmüller H. Science Adv. 10, eadp4425 (2024)
[4] Schultze S, Luke DR, Grubmüller H. J. Chem. Theory Comput. 21, 8227 (2025)

Advancing Gene Editing through Computational Methods and Deep Learning

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Abstract:

Future medicines will increasingly rely on editing DNA and RNA to treat a wide range of diseases, with broader applications in agriculture and sustainability. Designing next-generation genome editing tools requires not only biological insight but also advanced computational approaches to probe their molecular mechanisms. I will present recent and unpublished advances that combine molecular simulations with deep learning to dissect and engineer CRISPR systems. Classical and ab-initio molecular dynamics, deep learning–based structure prediction, and Graph Neural Networks (GNNs) are used to capture the conformational dynamics and engineer these complex assemblies. In particular, deep learning combined with free-energy simulations reveals the dynamic behavior of large CRISPR complexes such as Cascade and informs the engineering of compact RNA-targeting Cas proteins. Linear Discriminant Analysis explains how conformational selection and induced fit enhance the efficiency of base-editing enzymes. Finally, Graph Attention Networks (GATs) coupled with free-energy simulations uncover the mechanism of DNA filament formation in CRISPR-associated transposons. Together, these findings underscore how cutting-edge computational methods drive new biological discoveries, paving the way for the rational design of improved genome editing technologies.

Integrative structure determination of sub-complexes of the mitochondrial contact site and cristae organizing system (MICOS)

Muskaan Jindal[1], Rakesh Mahato[2], Arko Guha[2], Kartik Majila[1], Shreyas Arvindekar[1], Anand Vaidya[2], Shruthi Viswanath[1]

[1] *National Centre for Biological Sciences, TIFR, Bangalore*
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Presented by: Shruthi Viswanath

Abstract:

Integrative structure determination is a method of determining structures of large macromolecular assemblies by combining data from complementary experimental methods with physical principles, statistical inference, and prior models. It is particularly useful for assemblies which are recalcitrant to direct observation by experiments such as cryo-electron microscopy and X-ray crystallography. We are characterizing the mitochondrial contact site and cristae organizing system (MICOS) complex, an inner mitochondrial membrane (IMM) assembly. MICOS is present at the cristae junction (CJ) and regulates cristae formation and remodeling. Recently, the structures of two domains of the MICOS subunits Mic60 and Mic19 have been characterized by X-ray crystallography. However, the overall architecture of the MICOS complex is yet to be determined, and the functions of several subunits are not known. Here, we applied Bayesian integrative structure determination to characterize the structures of sub-complexes of the MICOS complex, including Mic60, Mic19, Mic10, and Mic13, which are critical for CJ formation. We integrated information from in vivo crosslinking mass spectrometry (XLMS), biochemical assays, electron tomography (ET), homology modeling, AlphaFold predictions, and sequence alignments. The integrative structure was validated by information from biochemical studies and XLMS that were not used in the modeling. Our integrative structures suggest that the two MICOS sub-complexes are bridged by Mic13. We uncovered novel protein-protein interactions between Mic13, Mic10, and Mic60, which are being validated by biochemical experiments. Finally, we rationalized mutations using the integrative structure revealed the structural basis of the associated diseases. Overall, our approach provided insights into the structure and function of the MICOS complex. If time permits, I will also discuss recent methods developed by our group; specifically, deep learning methods for sequence-based identification of binding sites for intrinsically disordered regions, and methods for localizing macromolecules in cryo-ET (cryo-electron tomography) data.

Delineating the survival strategies employed by *Mycobacterium tuberculosis*

Vinay Nandicoori

Center for Cellular and Molecular Biology, Hyderabad, India

Abstract:

Mycobacterium tuberculosis (Mtb), the causative agent of TB, causes approximately 1.2 million deaths per year worldwide. Mtb is capable of surviving in the hostile host environment and thrives when the opportune moment arises. To survive in the host, Mtb employs multiple mechanisms, including regulation of host signaling, apoptosis, virulence, transcriptome, and autophagy, to name a few. We are interested in elucidating the mechanisms employed by Mtb. Mtb encodes about 4000 genes, of which 800 are essential for its survival in complete growth medium. My lab studies various cellular processes such as regulation of transcription, translation, cell wall synthesis, amino acid metabolism, and secretion pathways. We are particularly interested in how Mtb serine/threonine kinases regulate these processes. I will be presenting our lab's recent work on these aspects.

Dynamics of Plasmodium enzyme complexes from native cryoEM structures

Manidipa Banerjee

Indian Institute of Technology Delhi (India)

Abstract:

Structural biology generally requires homogeneous sample preparation, however, the workflow of cryoelectron microscopy allows computational sorting of heterogeneous samples. We are utilizing the single particle reconstruction pathway for structural analysis of protein complexes from highly heterogeneous samples of the malarial parasite *Plasmodium falsiparum* in its merozoite stage. The blood stage of malaria is the most damaging, as Plasmodium parasites multiply in Red Blood Cells (RBCs) which can cause severe complications. Protein complexes secreted/displayed by the parasite were subjected to chromatography and density gradient ultracentrifugation for size based separation. Fractions with substantial protein content were cryofrozen and images captured in near native stage, which resulted in highly heterogeneous populations of particles. The particles were subjected to reiterative 2D and 3D classifications and refinement protocols to obtain atomic resolution structures of protein complexes such as proteasomes and aminopeptidase enzyme complexes. The identity of the complexes were determined from the cryoEM density by bottom-up structural proteomics. We find that the native structures have significant differences compared to recombinant protein complexes. Extended all atom simulations of the cryoEM structures suggested molecular pathways of substrate entry into the enzyme active site. Our results capture dynamic cellular processes involved in the blood stage of the malarial parasite, which may aid in the development of more effective inhibitors.

Modeling Synapse Development and Dysfunction with Stem Cell - Derived Human Neurons

Soham Chanda

Associate Professor, Biochemistry & Molecular Biology Department, Colorado State University

Abstract:

Neurons in our brain communicate with each other via highly specialized subcellular compartments called synapses. These delicate structures process and transmit information across the nervous system using electrochemical signaling, and pathogenic mutations in synaptic proteins can cause severe mental disorders. However, at a cell biological level, it remains mostly unclear how synapses assemble, properly organize their pre- and postsynaptic components, and align their molecular machineries across the transsynaptic space. My lab utilizes stem cell -derived neurons to explore the fundamental mechanisms of synapse development and model neurological diseases associated with synapse malfunction, especially in human cellular environment. In my talk, I will discuss our recent efforts to better understand the contributions of presynaptic neurotransmitter release and postsynaptic cell adhesion molecules, which modulate synapse formation and shape the functional properties of human synapses. This work employs interdisciplinary techniques, e.g. cellular reprogramming, electrophysiology, high-resolution imaging, molecular biology, gene-expression, and biochemical assays.

Exploring the Regenerative Processes Initiated by Biomaterial Systems: Emphasizing the Significance of Glycosylation

Abhay Pandit

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Abstract:

Our contemporary research paradigm has advanced beyond viewing biomaterials as mere passive structures, now focusing on the application of functionalisation and biofabrication techniques to elicit specific responses. Central to this approach is a comprehensive understanding of the host response, contextualised within the underlying pathology at both local and systemic levels, which is essential for developing effective treatment strategies. Our research team has made significant progress in the development of glycomic tools, which have yielded valuable insights in this domain. We adhere to the principle that the intrinsic therapeutic potential of biomaterials must be integrated into the design process. By leveraging this understanding, we have successfully employed appropriate functionalisation methods to link biomolecules with various structural components. The systematic organization of biomolecules into intricate, self-organizing systems is essential for various biological functions and is pivotal in the development of therapeutic scaffolds. In our recent scientific investigations, we employed a glycobiology-focused approach to explore and manipulate supramolecular structures, providing a transformative perspective on the design and application of biomaterials.

A bioengineering approach to next-gen micro physiological systems

Kristopher A Kilian

School of Materials Science and Engineering, School of Chemistry, Australian Centre for Nanomedicine, University of New South Wales, Sydney NSW, Australia

Abstract:

The assembly of cells into tissue arises from a tight coordination of multivariate signals, where dynamic materials properties and cellular activities converge to define form and function. Over the past decade, bio-fabrication techniques have enabled avenues to precisely define soft and hard materials in 3D constructs often in the presence of living cells. However, mimicking the dynamic aspects of natural materials while simultaneously directing complex cellular morphogenesis remains challenging. Here I will present our work combining cell and tissue engineering with supramolecular materials chemistry towards bio-fabrication of reproducible organotypic model systems. First, I will describe a new class of peptide-based hierarchically structured hydrogels that are self-healing, have tunable viscoelasticity and support a wide range of cell and organoid cultures. Next, I will demonstrate how these materials can be used to direct morphogenesis in vitro, thereby providing control over 3D cellular heterogeneity. Finally, I will present our integration of advanced hydrogels into microfluidic systems to study circulating cells and extravasation events. Microphysiological systems are changing the way in which fundamental biological questions can be probed, which will aid our understanding of biological processes while revealing new design parameters for pre-clinical testing to aid precision medicine.

Glycocalyx in Cancer Invasion & Drug Resistance

Shamik Sen

Department of Biosciences and Bioengineering, Indian Institute of Technology (IIT) Bombay, Powai, Mumbai 400076, India

Abstract:

The majority of the eukaryotic cell surface is decorated with a layer of membrane-attached polysaccharides and glycoproteins collectively referred to as the glycocalyx. While the formation of a bulky glycocalyx has been associated with cancer progression, the mechanisms by which the glycocalyx regulates cancer invasiveness are incompletely understood. We show that a bulky glycocalyx increases invasiveness by via increased traction generation and by modulating substrate-specific adhesion. Remarkably, we show that the glycocalyx also protects the cell from undergoing nuclear rupture by buffering invasion-associated stresses. Using AFM experiments, we show that the glycocalyx behaves as a poroelastic layer on the cell surface that dissipates stresses through outflow of water. In addition to physically buffering stresses, we show that then glycocalyx also inhibits drug uptake, with cells possessing a bulky glycocalyx being more drug resistant. Together, our results establish the glycocalyx as a "physico-chemical barrier" that drives cancer invasiveness and drug resistance.

Guiding innovation towards therapy, with stem cells and organoids

Maneesha S Inamdar

Director, BRIC-Institute for Stem Cell Science and Regenerative Medicine, Bengaluru, India;
Professor, Jawaharlal Nehru Centre for Advanced Scientific Research, Bangalore, India

Abstract:

Stem cells possess extraordinary abilities for self-organization, lineage specification, and tissue regeneration—traits that have driven significant progress in therapeutic innovation. Despite these advances, the molecular and cellular processes that guide their developmental choices and sustain tissue integrity remain only partially understood. Deciphering the inherent regulatory frameworks of stem cells could unlock new approaches to tissue repair and regeneration, inspired by the principles of embryogenesis. Recent findings highlight the pivotal role of early metabolic signals in shaping developmental outcomes. Harnessing these metabolic cues presents a compelling strategy for fine-tuning stem cell fate, with far-reaching implications for regenerative medicine. Organoids—three- dimensional constructs derived from stem cells that mimic tissue architecture and morphogenesis—enable detailed exploration of developmental pathways and responses to environmental changes. Together, stem cells and organoid systems offer unprecedented insight into human tissue formation, establishing themselves as powerful tools for assessing toxicity and teratogenic potential. The convergence of stem cell biology and organoid technology holds immense promise for advancing regenerative therapies and refining our understanding of human development.

Epigenetically regulated parasites' RIFINs contribute to severe malaria pathogenesis

Rajina BR[1] and Arumugam Rajavelu[1]*

[1,] *Department of Biotechnology, Bhupat & Jyoti Mehta School of Biosciences, Indian Institute of Technology Madras, Chennai - 600 036, Tamil Nadu, India.*

Abstract:

The epigenetic players contribute significantly to the fine-tuning of gene expression in eukaryotic organisms. The plasticity of genome organization and its functional outcomes are tightly regulated for the successful development of organisms. The epigenetic players that mediate gene silencing are conserved from lower eukaryotic organisms to multicellular organisms. The intracellular lower eukaryotic organisms, like protozoan pathogens, exploit host machinery to establish a successful infection. Similarly, the human malaria parasite is an obligate intracellular pathogen that extensively modulates its gene expression program in response to the host cells, including the liver and RBCs. One such mechanism adapted by the malaria parasite is antigen switching to escape from host immunity, which paves the way to the development of severe malaria. In my talk, I will highlight our recent work on epigenetic reader proteins that regulate the differential expression of variable surface antigenic genes in P. falciparum through selective interaction with modified chromatin. Such interaction tightly regulates the RIFIN (one of the VSA family), which is linked to the severe malaria pathologies.

Emerging Cell Therapies for Type 1 Diabetes and Adrenal Insufficiency: From Bench to Bedside

Charlotte Steenblock

Molecular Endocrinology, Dept. of Internal Medicine III, Technische Universität, Dresde

Abstract:

Type 1 diabetes and congenital adrenal hyperplasia (CAH) represent paradigmatic endocrine disorders where lifelong hormone replacement remains the cornerstone of therapy but falls short of restoring physiological function and quality of life. Recent advances in regenerative medicine offer new hope through cell-based therapies, aiming for durable, functional cures. In my talk, I will provide a concise overview of the current state and prospects of cell therapy for type 1 diabetes, focusing on the transplantation of human islets and stem cell-derived β-like cells. Key challenges, including immune rejection, donor cell scarcity, and the need for scalable, cost-effective production, will be discussed alongside innovative solutions such as encapsulation technologies and alternative islet sources (e.g., xenogeneic and bioengineered cells). I will then highlight recent progress in cell-based approaches for adrenal insufficiency, specifically the transplantation of in vitro-derived steroidogenic cells in models of CAH, and their potential translation to clinical application. Finally, I will briefly touch on gene therapy strategies as complementary or alternative approaches. This presentation will emphasize translational hurdles, ongoing clinical trials, and the critical need for multidisciplinary collaboration to bring these therapies from the laboratory to patients.

A spatial and tissue digital landscape of triple negative breast cancer – diagnosis to therapy

Shankar Subramaniam

Department of Bioengineering, University of California, San Diego, USA

Abstract:

Triple negative breast cancer (TNBC) is a highly aggressive breast cancer (BC) subtype, characterized by a clinical absence of HER2 and <1% expression of hormone receptors, and shows significant heterogeneity across patients. Prior efforts at characterizing this heterogeneity into TNBC subtypes to offer specific treatments have had only partial success. In a study with a large cohort from LSU of circa 250 TNBC patients (with nearly equal numbers of those with European and African ancestry), we show that cellular heterogeneity composition in the tumor tissue, derived from bulk tissue transcriptomics, can aid subtyping TNBC patients and suggest potential therapeutic interventions. Furthermore, the markers of these subtypes can aid in predicting survival outcomes. To explore the spatially resolved mechanisms associated with these subtypes and to derive mechanisms of poor prognosis, we carried out spatial transcriptomics of several FFPE tissue sections. A detailed investigation of spatial transcriptomics, parsing the regions involving invasive epithelia and the tumor microenvironment, shows several mechanisms associated with atypia that can be prognostic. Delineating the cellular states defined by the transcriptome shows the aggressive biology associated with poor and good prognosis outcomes. We identified mechanisms dysregulated in poor-prognosis atypia associated with complement pathways, with implications for prognosis and therapy. I will also present results from Pregnancy-Associated TNBC and exploration of mechanisms that persist beyond involution.

Towards AI Bacteria

Sangram Bagh

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Abstract:

Performing cellular computations with engineered bacteria has enormous importance in biocomputer technology development at the micron scale, where microprocessor-based computers have limitations due to energy, cost, and technological constraints. Here, we designed and built artificial neural networks with genetically engineered bacteria that can identify prime numbers, vowels, and even determine the maximum number of pieces of pizza or pie that can be obtained from a given number of straight cuts. In addition, the 'intelligent' bacteria can answer mathematical questions such as whether a number n's factorial is divisible by $n \times (n+1)/2$ OR whether a number n's square can be expressed as the sum of three factorials. All those problems are classic abstract computational problems and are solved by a computer by writing codes in Python or C. Introducing such abstract computational capability in living cells, will be a step forward in biocomputer technology development and may help understanding the biochemical nature of 'intelligence'.

Indian Snake Venoms: Ecological Drivers and Therapeutic Innovation

Kartik Sunagar

Evolutionary Venomics Lab, Centre for Ecological Sciences, Indian Institute of Science, Bangalore 560012, Karnataka, India.

Abstract:

Molecular innovations in snake venom have been central to the evolutionary success of snakes across terrestrial and aquatic habitats. Venom composition is thought to be shaped by ecological,environmental, and evolutionary pressures—including diet, geographic isolation, ontogeny, sex, and underlying molecular mechanisms. This diversity drives adaptation but also complicates snakebite treatment. Yet, the ecological and environmental determinants of venom variation in Indian snakes remain largely unexplored, hindering the development of effective therapies. In this talk, I will present key findings from my lab, demonstrating how ecology and environment drive striking differences in venom composition, pharmacology, and potency among Indian snakes—and the serious consequences of this variation for antivenom efficacy. I will also outline our ongoing efforts to develop pan-India, effective snakebite therapies aimed at safeguarding the lives, limbs, and livelihoods of the hundreds of thousands of people affected by snakebite in India each year.

“To B or not to B” in Nucleic Acids Chemistry

Naoki Sugimoto

Frontier Institute for Biomolecular Engineering Research (FIBER), Konan University

Abstract:

In this lecture, I will provide an overview of the basic concepts, methods, and recent applications of predicting the stabilities of nucleic acid structures. I explain the theory of the most successful prediction method based on a nearest-neighbor (NN) model. To improve the versality of prediction, corrections for various solution conditions considered hydration have been investigated. I also describe advances in the prediction of non-canonical structures of G-quadruplexes and i-motifs. Finally, studies of intracellular analysis and stability prediction are discussed for the application of NN parameters for human health and diseases. Acknowledgment. The author is grateful to the colleagues named in the cited papers from my laboratory, institute (FIBER), and others. This work was supported by the Ministry of Education, Culture, Sports, Science and Technology (MEXT) and the Japan Society for the Promotion of Science (JSPS) (Grant No. JP17H06351, 18KK0164, 19H00928, and 20K21258), especially for Grant-in-Aid for Scientific Research (S) (22H04975), JSPS Core-to-Core Program (JPJSCCA20220005), and The Chubei Itoh Foundation

Mechanism of DNA shredding by the innate immune systems of ATP-dependent nucleases

Saikrishnan Kayarat

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Abstract:

ATP-dependent restriction enzymes are one of the most common bacterial immune systems, and the first bacterial immune system to be discovered. These enzymes prevent phage infection, and regulate horizontal gene transfer, including acquisition of genetic elements harboring pathogenic islands and antibiotic resistance genes. Though common, studies on the mechanism and physiological role of ATP-dependent restriction enzymes have been limited and poorly understood. In contrast, the functioning of ATP-independent or Type II restriction enzymes, which has powered recombinant DNA technology and genetic engineering, is well-known. Why bacteria harbor the complex and energy-inefficient ATP-dependent restriction enzymes is a mystery. Biochemical and structural studies carried out on ATP-dependent restriction enzymes SauUSI (a Type IV restriction enzyme) and LlaBIII (a Type ISP restriction enzyme) by my laboratory have revealed that these enzymes cleave DNA in a manner distinct from other nucleases. These enzymes, which have a nuclease coupled to an ATPase belonging to the Superfamily 2 helicases, make multiple nicks on the entire length of the DNA in between two of their DNA target sequences. Nicks at multiple locations on the DNA away from the target sequence are made possible by the translocase activity of the ATPase that propels the nuclease along the DNA. The nicks result in the DNA being shredded between the two target sequences. The shredded DNA is difficult to repair, making the ATP-dependent restriction enzymes a potent barrier to entry of foreign DNA.

NETs and GATs: Countering pain through inhibitory neurotransmission

Aravind Penmatsa, PhD, FNASc, FASc

Associate Professor, Molecular Biophysics Unit, Indian Institute of Science, Bangalore

Abstract:

Neurotransmitter transporters are the synaptic workhorses that recycle released neurotransmitters at the neural synapses and enforce spatiotemporal control. These sodium/chloride-coupled neurotransmitter sodium symporters (NSS) facilitate the uphill movement of neurotransmitters across the presynaptic and glial cell membranes. Due to the central role played by these symporters in the control of neurotransmitter levels, they are also attractive targets for diverse drug classes including antidepressants and psychostimulants. Using structural studies through X-ray crystallography, cryoEM and biochemical analyses, we explored the contribution of noradrenaline transporter in pain relief as increased noradrenaline in the dorsal horn of the spine facilitates pain inhibition. We explore how commercially used pain inhibitors and natural toxins interact specifically with NSS members. Noradrenaline and GABA transporters have very similar structures but have a very different inhibitor interactions primarily affected by difference in the binding sites that dictate specificities. We have also delved into the substrate interactions with the GABA transporter that modulates inhibitory neurotransmission through GABA uptake, has allowed us to reconstruct the GABA reuptake cycle.

Learning Across Modalities: From Digital Pathology to Molecular Design

Ananth Grama

Professor, Department of Computer Science;
Associate Director, Center for Science of Information;
Purdue University, USA

Abstract:

Advances in digital pathology and high-content molecular profiling are generating unprecedented volumes of multimodal biological data, creating new opportunities and bottlenecks in understanding cellular behavior, disease mechanisms, and therapeutic responses. In this talk, I will present results from a unified research program that develops scalable AI frameworks to bridge histopathology, spatial transcriptomics, perturbation biology, and molecular design. I will first introduce AnnotateAnyCell, an open-source semi-supervised platform that accelerates expert annotation of whole-slide histopathology images through active contrastive learning and human-in-the-loop model refinement. Building on the integration of cellular imaging and molecular readouts, I will then describe GeneFlow, a rectified-flow-based framework that learns a continuous, bijective mapping between spatial transcriptomic profiles and cellular morphology, enabling high-resolution generation of diverse stain images and revealing spatially resolved molecular–phenotypic relationships. Extending these ideas to perturbation biology, I will present PertFlow, a cross-modal predictive model that jointly forecasts treatment-induced transcriptomic changes and synthesizes corresponding cellular morphologies by learning aligned drug-conditioned representations across RNA-seq and microscopy. Finally, I will discuss Pert2Mol, the first multimodal phenotype-to-structure generator that leverages paired control-treatment transcriptomic and imaging data to design candidate molecular structures using a rectified-flow transformer augmented with Student-Teacher Self-Representation learning. Together, these frameworks illustrate a path toward deeply integrated, data-driven discovery pipelines that connect cellular morphology, gene expression, and chemical structure—advancing both mechanistic insight and therapeutic design.

Bacterial Cell Growth and Dormancy: An Interplay Between Ribosome Biogenesis And Stringent Response Pathway

B. Anand

Department of Biosciences and Bioengineering, Indian Institute of Technology Guwahati, Guwahati 781039 (E-mail: banand@iitg.ac.in)

Abstract:

Cellular homeostasis is maintained by balancing the cell growth and quiescence in response to specific environmental cues. However, the regulatory mechanism that modulates cellular homeostasis is not understood in bacteria at appreciable details. Here, we investigated this phenomenon by studying the cross-talk between two antagonistic pathways, ribosome biogenesis that promote cell growth and stringent response that induce dormancy. In E. coli, controlled depletion of Era –an essential protein– halts protein synthesis leading to cell growth arrest and dormancy. This phenotype is reminiscent of of persister cells that evade the impact of antibiotic stress. Intriguingly, ribosomes isolated from Era-depleted cells were competent in protein synthesis in vitro, suggesting that ribosomes are downregulated in the absence of Era. This cell growth arrest can be rescued by deletion of relA that synthesizes (p)ppGpp, which serves as an alarmone in E. coli, suggesting epigenetic interaction between Era and RelA. In this talk, I will highlight how the regulatory mechanism that controls the molecular interaction between Era and RelA governs how cellular decision that modulates cell growth and dormancy is made.

Fishing for Success! Spinal Cord Regeneration in the Zebrafish

Catherina G. Becker

FRSB, Alexander-von-Humboldt Professor
Professor of Neural Development and Regeneration;
Administrive Director, Center for Molecular and Cellular Bioengineering (CMCB); Center for Regenerative Therapies at the Technische Universität Dresden, Germany;
Honorary Professor University of Edinburgh

Abstract:

Adult and larval zebrafish can repair lesions to their spinal cords and regain swim function within days to weeks. This goes hand in hand with regrowth of axons and the formation of new neurons, in part from neural stem cells. Functional recovery depends on the intricate interplay between neurons and stem cells in the central nervous system and different non-neural cell types that invade the spinal cord after injury. The roles of the interactions between neural and non-neural tissues in successful spinal cord regeneration is not well understood. In our latest study, we use scRNA-seq and in vivo experiments to show that sema4ab, mainly expressed by lesion-reactive microglia, attenuates regenerative neurogenesis by changing the complex lesion environment. After spinal injury, disruption of sema4ab doubles the number of newly generated progenitor cells and neurons but attenuates axon regrowth and recovery of swimming function. Disruption of the plxnb1a/b receptors, selectively expressed by neural progenitor cells, increases regenerative neurogenesis. In addition, disruption of sema4ab alters activation state and cytokine expression of microglia, such that fibroblasts increase expression of the cytokine tgfb3, which strongly promotes regenerative neurogenesis. Hence, sema4ab in microglia attenuates regenerative neurogenesis in multiple ways, likely directly through plxnb1a/b receptors and indirectly, by controlling the inflammatory milieu and tgfb3 levels. Targeting Sema4A-dependent signalling in non-regenerating vertebrates may be a future strategy to improve regenerative outcomes.

A diet-gene interaction that targets mitochondria to modulate longevity

Arnab Mukhopadhyay

PhD, FNASc, FASc, FNA
Molecular Aging Laboratory, National Institute of Immunology

Abstract:

Normal life history traits of an organism are shaped by its ability to adapt to the dynamic environmental challenges within its niche, including fluctuations in food availability and quality. This adaptive capacity is maintained by intricate diet-gene interactions, many of which remain poorly understood. Our study demonstrates that the conserved mTORC2 catalytic subunit, RICTOR/RICT-1, plays a critical role in maintaining adaptability to variations in the bacterial diet of C. elegans. When fed a nutrient-rich diet, the rict-1 mutant shows enhanced tolerance to osmotic stress and an extended lifespan. These phenotypic changes depend on appropriate mitochondrial function and metabolic outputs. Notably, the enhanced phenotypes of the rict-1 mutant are modulated by bacterially derived metabolites. Our findings reveal a novel mechanism by which RICTOR/TORC2 prevents bacterially derived metabolites from impacting host cellular functions and lifespan.

Islet cell secretory mechanisms: what are drivers that are dysregulated during diabetes

Nikhil Gandasi

Developmental Biology and Genetics (DBG), Indian Institute of Science, Bangalore

Abstract:

The diabetes prevalence in India stands at 9.3% according to 2018 and is rising at a fast rate. A well-functioning pancreas preserves glucose homeostasis and prevents diabetes. Glucose homeostasis is maintained by secretion of insulin from pancreatic beta cells that lowers blood glucose after a meal. Although mechanisms behind secretion of insulin have been studied for years a clear subcellular view on secretion had remained elusive. My talk will focus on molecular drivers that show subcellular changes in diabetes in both the young and the older people. We have been pursuing high-resolution microscopy approaches to understand the islet environment to understand the functional changes brought about by these molecular drivers. This can provide novel insights into islet dysfunction during diabetes at multiple levels such as single cells of islet, whole islet, islet cell interactions and pancreas functioning.

Bioenergetic Crosstalk between Host and Pathogen Mediates Drug Tolerance in *Mycobacterium tuberculosis*

Amit Singh

Professor, Department of Microbiology and Cell Biology, Indian Institute of Science (IISc)

Abstract:

Successful treatment of tuberculosis (TB) depends on eradicating its causative agent, Mycobacterium tuberculosis (Mtb), in the host. However, the emergence of phenotypically drug-resistant Mtb in the host environment tempers the ability of antibiotics to cure disease. Host immunity produces diverse micro environmental niches that Mtb exploits to mobilize adaptation programs. Such differential interactions amplify pre-existing heterogeneity in the host-pathogen milieu to influence disease pathology and therapy outcome. Therefore, comprehending the intricacies of phenotypic heterogeneity can be an empirical step forward in potentiating drug action. With this goal, we discovered the interconnectedness between macrophage bioenergetics and bacterial heterogeneity underlying phenotypic drug resistance. We further examined a few clinically-approved host- directed pharmacological agents that manipulate macrophage metabolism to collapse heterogeneity in bacterial physiology, thereby potentiating the lethal activity of anti-TB drugs. Our findings suggest targeting heterogeneity in host-pathogen encounters to shorten TB therapy time.

Identification of novel therapeutic interventions against *Mycobacterium tuberculosis*

Ramandeep Singh

Senior Professor, Centre for Tuberculosis Research, Tuberculosis Research Laboratory, BRIC-Translational Health Science and Technology Institute, Faridabad, Haryana, India

Abstract:

To address the growing threat of drug-resistant TB, there is an urgent need to identify small molecules that possess a novel mechanism of action and show synergy with first-line TB drugs. Inorganic polyphosphate (polyP), an inorganic linear polymer consisting of hundreds of phosphate residues, is synthesised by Polyphosphate kinase – 1 (PPK-1). Exopolyphosphatases are enzymes involved in the degradation of polyP. Our group has shown that polyP homeostasis is essential for M. tuberculosis virulence. These findings suggest that targeting PPK-1 could be a promising approach for developing novel anti-TB therapies. We have performed high-throughput screening against the PPK-1 enzyme and identified small molecules that possess activity against intracellular M. tuberculosis. We have also performed phenotypic screening to identify small molecules that possess activity against M. tuberculosis. We have screened nearly 25,000 molecules and identified several molecules that possess anti-tubercular activity in the low micromolar range. The pyridine carboxamide-based scaffold identified from phenotypic screening requires AmiC for activation and inhibits M. tuberculosis growth in a mouse model of infection. The molecules identified from both target and phenotypic-based screening show activity against drug-resistant strains and synergy with first-line TB drugs. Experiments are in progress to further optimise these lead molecules.

Programmable genetic sensors for deep-tissue imaging

Arnab Mukherjee, Ph.D

Associate Professor of Chemical Engineering & Chemistry, University of California, Santa Barbara, CA

Abstract:

Innovations in protein engineering have revolutionized fluorescent proteins, transforming them into versatile, genetically encoded biosensors that can be easily adapted to monitor a wide range of user-defined biological events with molecular specificity. However, many critical biological processes, such as cancer metastasis, brain function, and the efficacy of cell- and gene-based therapies, still require study within the intact physiological context of experimental animal models, as opposed to cultured cells. This creates a fundamental scientific impasse: sensors based on fluorescent proteins are ineffective at depth, while gold-standard clinical modalities like MRI excel at deep-tissue penetration but lack sensitivity to molecular- level events. To break this impasse, my research explores the convergence of synthetic biology and advanced biomedical imaging. In this talk, I will discuss our recent efforts in this domain, focusing on the development of modular aquaporin-based protease-activatable probes for enhanced reporting (MAPPER) — an all-genetic platform to create modular and easily programmable sensors for MRI. By harnessing the clinical capabilities of MRI for noninvasive, molecular-level reporting within deep, optically opaque tissues, MAPPER provides the crucial link between cellular events and whole-organism physiology.

Advanced imaging unveils the progressive demise of islet cells from euglycemia to type 2 diabetes and back

Michele Solimena, MD, PhD

Chair of Molecular Diabetology, Univ. Hospital and Medical Faculty, TU Dresden

Abstract:

Pancreatic islet beta cells are responsible for producing and secreting insulin, the hormone that lowers blood glucose and maintains energy balance. The loss or dysfunction of beta cells — leading to insufficient insulin to meet physiological demands — underlies all forms of diabetes. In this talk, I will discuss our recent findings on the cellular and molecular mechanisms that enable beta cells to adjust insulin synthesis and release in response to changing metabolic needs. I will also highlight how studying human islets in situ from living donors is advancing our understanding of the molecular alterations that occur in beta cells throughout the progression of hyperglycemia — from normoglycemia to prediabetes, type 2 diabetes, and its remission.

Molecules to Medicine: Challenges and Opportunities

Pradip Kumar Sasmal

Chief Scientific Officer, Morepen Proprietary Drug Research Pvt Ltd

Abstract:

The talk will focus on the multifaceted journey required for a molecule to successfully qualify as a medicine. The journey from a New Chemical Entity (NCE) to an approved medicine is a complex, high-risk endeavour characterized by significant challenges. The primary obstacle is the low success rate and high attrition of NCEs, largely due to translational challenges. Preclinical models often fail to predict human efficacy and safety accurately, leading to many failures in clinical trials. Even when a drug reaches the market, rare or idiosyncratic safety concerns that emerge only with broad population use can lead to costly and disruptive drug withdrawals. These issues underscore the limitations in current predictive toxicology and human disease modelling. Despite these hurdles, numerous opportunities exist to enhance efficiency and success rates. Innovative formulation solutions offer a powerful strategy to overcome poor pharmacokinetics, improve bioavailability, and enable targeted delivery, effectively transforming the potential of existing molecules. Furthermore, drug repositioning and repurposing provide an accelerated pathway to market. By leveraging the extensive safety data of approved drugs for new indications, researchers can bypass early-stage testing and mitigate some of the inherent risks of novel development. Collectively, focusing on these strategic approaches, alongside improved predictive science, can help navigate the "valley of death" and improve the delivery of safe and effective medicines to patients.

Systems approaches for animal cell culture media development in biomanufacturing

Meiyappan Lakshmanan

Department of Biotechnology, Bhupat and Jyoti Mehta School of Biosciences, Indian Institute of Technology Madras, India

Abstract:

Cell culture media is one of the inevitable components in cell-based biomanufacturing. It functions as the life support system by supplying all requisite chemical signals and nutrients for cellular sustenance, proliferation, and differentiation ex vivo, and thus effectively replacing the animal's physiological conditions. To cultivate animal cells, researchers commonly use a two-part system, comprising a basal medium such as DMEM or RPMI 1640 and a complex, undefined supplement, such as fetal bovine serum (FBS). While this two-part cultivation system serves best for lab-scale cultivations, the use of serum is still undesirable in large-scale cultivations. Driven by these limitations, the development of serum-free media happened, where serum is replaced with a cocktail of proteins and small molecules. Commercial biomanufacturing now happens on completely chemically defined media (CDM), which are built based on these early predecessors. Development of cell line-specific CDMs typically begins by cultivating the target cells in a generic base medium and subsequently, concentrations of individual components are optimized using traditional experimental strategies such as One-Factor-at-a-Time (OFAT) or Design of Experiments (DOE). The use of systems biology tools such as multi-omics profiling and genome-scale metabolic models (GEMs) has gained prominence in the last decade to better understand the nutritional requirements of individual cells. In this talk, I will present how these multi-omics profiling and GEMs can be effectively utilized for developing cell culture media with applications in biotherapeutics and cultivated meat manufacturing.

ROAD TO ACADEMIA

Multi-omics immune microenvironment interrogation of human brain malignancies identifies anti-tumor phagocytic immunomodulators.

Pravesh Gupta[1,#], Minghao Dang[2], Mekenzie Peshoff[3], Silvana Valdebenito-Silva[4] Shivangi Oberai[3], Simona Migliozzi[5], Gayatri Kumar[1], Rakesh Trivedi[1], Prashanth Chakrapani[3], Tuan M. Tran[6], Joy Gumin[7], Carlos Kamiya-Matsuoka[8], Jason Huse[3,9], Atul Maheshwari[11], Nicholas E. Navin[6], Amy B. Heimberger[12], Frederick F. Lang[7], Eliseo Eugenin[4], Antonio Iavarone[5], Karen Clise-Dwyer[10], Linghua Wang[2], Krishna P. Bhat[1,#]

[1]Cancer Biology, Mayo Clinic, USA, Departments of [2]Genomic Medicine, [3]Translational Pathology, [6]Systems Biology, [7]Neurosurgery, [8]Neuro-Oncology, [9]Pathology, [10]Hematopoietic Biology at MD Anderson Cancer Center, USA, [4]Neurobiology, The University of Texas Medical Branch, USA, [5]Neurological Surgery at the University of Miami Miller School of Medicine, USA, [11]Neurology and Neuroscience at Baylor College of Medicine, USA, [12]Neurosurgery, Northwestern University, USA (#) Correspondence.

Presenting Author: Dr. Pravesh Gupta

Abstract:

Glioma-associated macrophages (MAC) are largely considered immunosuppressive, despite their anti-tumor potentials, which remains elusive. In this pursuit, our transcriptomics (n=145,00) and cytometry studies of glioma-associated leukocytes (n=54) revealed 22 distinct cell types mediate brain immunity. Microglia decreased with glioma recurrence, whereas monocytic derivatives increased with glioma severity. MAC acquired antigen presentation-like phenotype upon tumor recurrence, with a concomitant increase in CD8+ T cells. Besides dissecting relapse associated immunity patterns, we provide a faithful clinical genomics framework for redefining macrophage polarization beyond M1/M2 states such as palmitic- and oleic-acid modules. Beyond canonical LM22 (leukocyte gene matrix), we curated glioma specific leukocyte signatures termed GlioTIME-36 (glioma tumor immune microenvironment-36) for deconvolution of brain transcriptomic datasets. Furthermore, we discovered that TREM2 and Galectin-9 expression in microglia correlated with enrichment of phagocytosis pathways. Using ex vivo and in vivo brain tumor models, we confirmed that TREM2+ and Galectin-9+ microglia efficiently phagocytosed glioma cells. In summary, besides providing the advanced optics of pan-glioma immune contexture for downstream translational and clinical applications, our reverse translational approach also identifies actionable anti-glioma phagocytic circuits for developing brain tumor immunotherapies.

Dense core vesicles- Biogenesis, trafficking, and physiological functions

Bhavani Shankar Sahu[1]

[1] *National Brain Research Centre, NH-8, Manesar, Gurugram, Haryana*

Presenting Author: Dr. Bhavani Shankar Sahu

Abstract:

Secretory vesicles are fundamental for neuronal and endocrine communication, ensuring the timely release of neuropeptides, hormones, and other bioactive molecules. Disruptions in their biogenesis or exocytosis are increasingly recognised as drivers of metabolic, cardiovascular, and neuropsychiatric disorders. We investigated this theme across multiple molecular and physiological contexts, including ER stress, high-fat diet-induced metabolic stress, and loss-of-function. The talk will focus on how neuroendocrine secretion drives various metabolic and cellular physiological functions, and how metabolic function drives neuroendocrine secretion. By linking molecular mechanisms with organismal outcomes, this integrated approach highlights a unifying principle—that disturbances in secretory vesicle dynamics underlie a range of human pathologies, and their study presents new therapeutic avenues for addressing metabolic, cardiovascular, and neurological diseases.

Rewiring the Immune Synapse with T-cell Engagers: How Flexibility, Membrane Mechanics, and Synaptic Architecture Shape T-Cell Engager Potency

Tanmay Mitra [1], Alexander Leithner [1], Shengpan Zhang [1], Salvatore Valvo [1], Michael L Dustin [1];

[1] *Kennedy Institute of Rheumatology, University of Oxford*

Presenting Author: Dr. Tanmay Mitra

Abstract:

Introduction: Conventional T cell engager (TcE) design focuses on affinity and epitope targeting yet often neglects how conformational flexibility and membrane mechanics influence T cell signaling. Objectives: We sought to investigate how structural flexibility and target membrane properties shape the performance of bispecific T cell engagers. By integrating SAXS-informed structural analysis, quantitative imaging of CD8 T-cell immune synapse on tunable supported lipid bilayers (SLBs), we examined how conformational flexibility and paratope spacing of the engagers, and diffusivity of integrin ligands (ICAM-1) affect T cell activation, synapse patterning, and cytotoxic output. Key Findings: Rigid formats outperformed flexible ones, even when epitope spacing was matched. Excessive flexibility introduced spatial uncertainty, compromising synapse fidelity and cytotoxic efficacy. Shannon entropy of the target-bound engagers and pPLCgamma-1 emerged as the best predictors of cytotoxic potency. Furthermore, anchored ICAM-1 enhanced perforin release in comparison with mobile ICAM-1. Outlook: Our ongoing work explores computational modeling of immune synapse in response to engagers and explores trispecific engagers that combine CD3, tumor antigens, and co-stimulatory targets to enhance function at low CD3 affinity. Conclusion: Engager potency is not just encoded in molecular affinity but in how an immune engager sculpts the synapse.

Lecanemab blocks the effects of the Aβ/fibrinogen complex on blood clots and synapse toxicity in organotypic culture

Pradeep Kumar Singh [1], Elisa Nicoloso Simões-Pires[1], Zu-Lin Chen [1], Sidney Strickland [1], Erin H Norris [1];

[1] *Rockefeller University, New York, NY, USA*

Presenting Author: Dr. Pradeep Kumar Singh

Abstract:

Proteinaceous brain inclusions, neuroinflammation, and vascular dysfunction are common pathologies in Alzheimer's disease (AD). Vascular deficits include a compromised blood-brain barrier, which can lead to extravasation of blood proteins like fibrinogen into the brain. Fibrinogen's interaction with the amyloid-beta (Aβ) peptide is known to worsen thrombotic and cerebrovascular pathways in AD. Lecanemab, an FDA-approved antibody therapy for AD, clears Aβ plaque from the brain and slows cognitive decline. Here, we show that lecanemab blocks fibrinogen's binding to Aβ protofibrils, preventing Aβ/fibrinogen-mediated delayed fibrinolysis and clot abnormalities in vitro and in human plasma. Additionally, we show that lecanemab dissociates the Aβ/fibrinogen complex and prevents fibrinogen from exacerbating Aβ-induced synaptotoxicity in mouse organotypic hippocampal cultures. These findings reveal a possible protective mechanism by which lecanemab may slow disease progression in AD.

From circuits to behaviour: understanding how danger and diet affects cognition

Prabahan Chakraborty [1,2], Freddy Jeanneteau [1];

[1] *Institut de Genomique Fonctionnelle, Montpellier, France,* [2] *SRM Institute of Science and Technology, Kattankulathur, Tamil Nadu*

Presenting Author: Dr. Prabahan Chakraborty

Abstract:

Introduction - Genetic and environmental factors both cause neuropsychiatric disorders, drastically affecting cognition. Understanding neural circuit mechanisms that underlie such behavioural deficits could reveal novel mechanisms for precise, early and effective interventions. Objective- We explored the neural correlates of behaviour across two disease models in mice. Brain circuits underlying behavioural deficits were investigated in the Magel2 knockout (KO) mouse model of Prader-Willi syndrome which shows comorbidity with autism, as well as a murine model of high-fat-diet induced obesity. Key findings – Magel2 KO mice showed impairments in overcoming social fear, which was identified to be associated with dysfunction in the hypothalamus-lateral septum (LS) circuit crucially involved in social cognition. Using in vivo circuit manipulation strategies like optogenetics, we identified a hyperactivity of LS-somatostatin neurons underlying such behavioural deficit. On the other hand, prolonged consumption of high-fat diet from adolescence led to longitudinal disruption of morphological plasticity in vivo in corticohippocampal circuits revealed by two-photon microscopy, and impaired long-term memory – both reversed by time-restricted feeding. Building on these findings, my independent research program aims at discovering key, novel neuronal circuits mechanistically linked with cognitive outcomes across neuropsychiatric disorders.

Endosomal Escape of pH-Sensitive Lipid Nanoparticle-Based Drug Carriers: From Poration to Transfection

Akhil Pratap Singh [1,2], Kana Shibata [1], Yusuke Miyazaki [2], and Wataru Shinoda [1,2];

[1] Department of Materials Chemistry, Nagoya University, Furo-cho, Chikusa-ku, Nagoya 464-8603, Japan, [2] Research Institute for Interdisciplinary Science, Okayama University, 3-1-1 Tsushima-naka, Kita-ku, Okayama 700-8530, Japan

Presenting Author: Dr. Akhil Pratap Singh

Abstract:

Lipid nanoparticles (LNPs) have transformed nucleic acid delivery, significantly impacting vaccines and gene therapies. However, their effectiveness is often limited by obstacles to escaping endosomes after cell uptake. While studies suggest that endosomal membrane (EM) disruption can enhance cytosolic release, the exact molecular mechanisms underlying this process remain unclear. In this study, we utilize molecular simulations with the SPICA force field to explore the fusion of LNPs with the EM and the release of their cargo. SPICA, based on a coherent coarse-grained framework, allows us to uncover multiple fusion pathways, with stalk-pore formation being the primary mechanism. This process begins as LNPs approach the EM, forming a stalk that leads to pore creation and the release of nucleic acids. Our findings emphasize the importance of ionizable lipids in facilitating nucleic acid reorganization and stalk formation. Additionally, we identify key factors affecting endosomal escape efficiency, such as lipid shape, pH sensitivity, EM tension, and the arrangement of encapsulated nucleic acids. Overall, this research enhances our understanding of intracellular delivery systems and paves the way for more effective LNP designs to improve gene therapy and RNA vaccine outcomes.

CONNECT TO POST-DOC MENTORS

#PANIIT20250140 - Connect to Postdoc Mentors

Tittle- Structural Impact of Cancer-Associated Mutations in Calnuc (NUCB1) and Their Functional Consequences

Agnibina Paul[1], Dr. Gopala krishna A[1]

[1]*Department of Biotechnology, IIT Madras*

Presenting Author: Agnibina Paul

Abstract:

Calnuc is a multi-domain Ca^{2+}-binding protein whose expression is elevated in various cancers, suggesting a role in tumorigenesis. Calnuc interacts with multiple partners, notably Gαi, and exhibits serine protease activity, both of which are relevant to cancer cell physiology. Our study shows that cancer cell–derived exosomes contain calnuc, which can induce tumorigenic properties in normal cells. To understand the impact of cancer-associated mutations in calnuc, we used computational analysis and site-directed mutagenesis to examine their effects on structure and function. The mutants displayed alterations in secondary structure and metal ion binding, while maintaining similar thermal stability and reversibility to the wild-type. Notably, one mutation in the second EF hand 8th position (V312M) has reduced calcium binding affinity as expected and displayed changes in serine protease activity as well as Gαi activation. Overall, our findings suggest that V312M modulates calnuc structure and Ca^{2+} interactions, affecting its functional aspects without majorly affecting stability, potentially influencing its role in cancer progression and highlighting its therapeutic relevance.

#PANIIT20250124 - Connect to Postdoc Mentors

Tubulin Isotypes for the Rescue: Decoding 5-Hydroxythalidomide Neurotoxicity and Functional Redundancy Among β-Tubulins in tubb5 Mutants

M S Ananthakrishna Tantry[1,2], Kirankumar Santhakumar[1]

[1]Zebrafish Genetics Laboratory, Department of Genetic Engineering, SRM Institute of Science and Technology, Kattankulathur 603 203, Tamil Nadu, INDIA; [2]Department of Biotechnology, Bhupat and Jyoti Mehta School of Biosciences, Indian Institute of Technology Madras, Chennai 600 036, Tamil Nadu, INDIA (Present affiliation)

Presenting Author: M S Ananthakrishna Tantry

Abstract:

Thalidomide (Thal), once infamous for its teratogenicity and later repurposed for leprosy and multiple myeloma, is linked to peripheral neuropathy. We propose that its major metabolite, 5-hydroxythalidomide(5OHThal), induces neurotoxicity by disrupting microtubule dynamics. Zebrafish embryos exposed to 400µM Thal or 5OHThal from 2hpf exhibited reduced head and eye size, and malformed pectoral fins. In Tg[her4.3:EGFP] embryos, neuronal progenitors were markedly reduced, suggesting impaired neurogenesis. Secondary motor neuron innervation and neuromast hair cell formation were disrupted, indicating deficits in axon guidance and neuromast development. Molecular docking and dynamics simulation studies revealed stable 5OHThal binding near the colchicine pocket of tubulin heterodimer, with the highest binding affinity observed for the heterodimer containing tubb5, indicating differential affinity. Consistently, gene expression analysis showed the overexpression of tubb5, tubb2 and tubb6 in Thal and 5OHThal treated larvae. To validate this mechanism, we generated tubb5 mutants via CRISPR-Cas9 mutagenesis producing C-terminal truncations (Δ47 and Δ31aa). Although homozygous mutants showed no morphological defects, tubb2 expression was markedly upregulated, reflecting compensatory β-tubulin isotype regulation. These results reveal tubulin isotype compensation mechanism and establish zebrafish as an effective model to study functional diversity and redundancy between tubulin isotypes.

#PANIIT20250108 - Connect to Postdoc Mentors

Tunneling nanotubes as a therapeutic target for glioblastoma

Sambhavi Pattnaik[1], Gautam Mohapatra[1], Amal Kanti Bera[1]

[1]*Department of Biotechnology, Bhupat and Jyoti Mehta School of Biosciences, Indian Institute of Technology Madras, Chennai-600036, India*

Presenting Author: Sambhavi Pattnaik

Abstract:

Glioblastoma multiforme (GBM) is a highly aggressive brain tumor accounting for 48% of malignant gliomas. Despite multimodal interventions, GBM remains incurable. GBM cells establish extensive intercellular networks through tunnelling nanotubes (TNTs), actin-based cytoplasmic bridges that facilitate the exchange of cellular cargoes. Increasing evidences suggest that TNTs enhance tumor survival and promote drug resistance, contributing to disease progression. However, destabilizing TNTs have not been clinically explored. Thus, targeting TNTs represents an underexplored avenue for GBM therapy. In this study, we developed a strategy to destabilize TNTs by increasing intracellular Ca^{2+} levels in GBM cells. We identified that the FDA-approved antidepressant nortriptyline elevates intracellular Ca^{2+}, disrupts TNT formation, and exerts potent anti-glioblastoma activity. Quantitative analysis demonstrated that nortriptyline markedly reduced TNT density and structural stability. In both monolayer and 3D spheroid GBM models, nortriptyline elicited a more pronounced loss of cell viability than the standard therapeutic agent temozolomide (TMZ). Furthermore, zebrafish xenograft assays confirmed that nortriptyline treatment significantly suppressed tumor proliferation in vivo. Collectively, these findings establish TNT disruption as an effective anti-GBM mechanism and support the potential repurposing of nortriptyline as a clinically translatable therapeutic candidate.

#PANIIT20250107 - Connect to Postdoc Mentors

G-Quadruplex structures within the hfq gene regulate RNA-protein interactions in Acinetobacter baumannii

Aakriti Singh[1], Mansee Patel[1], Tarun Kumar Sharma[2], Amit Kumar[1]

[1]Mehta Family School of Biosciences and Biomedical Engineering, Indian Institute of Technology Indore, Simrol, 453552, Madhya Pradesh, India; [2]Department of Medical Devices, National Institute of Pharmaceutical Education & Research (NIPER), S.A.S. Nagar, Mohali - 160062, Punjab, India

Presenting Author: Aakriti Singh

Abstract:

G-quadruplexes (G4s) are non-canonical nucleic acid structures with emerging roles in gene regulation. While extensively studied in eukaryotes, the roles of G4S, especially two-tetrad (2G) G4s, in prokaryotic systems remain largely underexplored. This work explores G4-mediated regulation within the hfq gene of Acinetobacter baumannii, which reveals a previously unknown regulatory layer in bacterial gene expression. In this study, utilizing a combination of in-silico, biophysical, and biochemical approaches we demonstrated the ability of hfq RNA and DNA sequences to form two-tetrad G4 structures. These G4 motifs were recognized and stabilized by the G4 ligand BRACO-19, which displayed favorable binding affinity. Importantly, both RNA and DNA G4s interacted with the Hfq protein, with a notable preference for RNA G4s by the full-length protein. Stabilization of these G4 motifs by several G4-binding ligands resulted in downregulation of hfq transcript and Hfq protein levels. This work highlights the functional versatility of non-canonical nucleic acid structures and positions G4-Hfq interactions as dynamic regulatory switches that may serve as scaffolds for modulating gene expression in bacteria. Overall, this study provides a foundation for targeting G4 motifs as innovative antimicrobial intervention strategies against the multidrug-resistant pathogen, A. baumannii.

SuBMIT: A Toolkit for Facilitating Simulations of Coarse-Grained Structure-Based Models of Biomolecules

Digvijay Lalwani Prakash [1], Arkadeep Banerjee [1], Shachi Gosavi[1]

[1]*National Centre for Biological Sciences, Tata Institute of Fundamental Research, Bengaluru 560065, India*

Presenting Author: Digvijay Lalwani Prakash

Abstract:

Coarse-grained structure-based models (CG-SBMs) are simplified potential energy functions of biomolecules or their complexes, that encode their structure. Molecular dynamics simulations of such SBMs have been successfully used to study long time scale dynamics such as protein and RNA folding, and large conformational transitions of their complexes. Moreover, SBMs are easy to modify and can be adapted for the specific biomolecular problem that needs to be investigated. However, the force-fields of SBMs are not usually included in commonly used simulation packages resulting in a barrier to their use. Here, we present SuBMIT (Structure Based Models Input Toolkit), a toolkit for generating coarse-grained SBM input files for performing MD simulations with GROMACS and OpenSMOG. The different flavors of SBMs included in the software can be used to simulate systems such as folding and conformational ensembles of proteins with intrinsically disordered regions, 3D-domain swapping of proteins, assembly of large homogeneous or heterogeneous RNA and protein multimer complexes. We describe our study on dynamics and assembly of simple icosahedral RNA viruses, as an example of multimeric assembly systems that can be easily set up using SuBMIT.

#PANIIT20250959 - Connect to Postdoc Mentors

Differential Long-Range Thermodynamic Coupling Governs Slow and Fast Protein Dynamics

Dhruv Kumar Chaurasiya[1], Athi N Naganathan[1]

[1]*Department of Biotechnology, IIT Madras, Chennai, 600036, India*

Presenting Author: Dhruv Kumar Chaurasiya

Abstract:

Proteins display a range of conformational motions spanning the micro (μs) - to millisecond (ms) timescales. The resulting dynamics enable access to transient, sparsely populated states that are critical for stability, function, and regulation. Here, we demonstrate that thermodynamic coupling free energies derived from a structure-based statistical mechanical model accurately capture the residues that undergo conformational exchange from a curated RelaxDB dataset of 133 proteins. Specifically, protein residues that display fast dynamics (ps-ns) are characterized by lower local stabilities and coupling free energies, while those that undergo slow exchange (μs-ms) display proportionately higher stability and coupling free energies with an average mean difference of ~14 kJ/mol. While the residues exhibiting fast and slow dynamics are distributed across the structure, the mean shortest distance between them is just ~10 Å. Cross-validation with specific model systems supports the robustness of our findings. We thus establish a quantitative link between dynamics, local stability, and thermodynamic coupling, with strong implications for understanding how the sequence-structure connection determines macromolecular dynamics.

#PANIIT20250956 - Connect to Postdoc Mentors

Understanding the mechanism of action of HIV Protease dimerization inhibitors

Avinash Dakshinamoorthy[1], Sanjib Senapati[1]

[1]*Department of Biotechnology, IIT Madras, Chennai, 600036, India*

Presenting Author: Avinash Dakshinamoorthy

Abstract:

HIV Protease is a vital target in the treatment of AIDS, with ten FDA approved drugs currently in use. All of these protease inhibitors (PIs) function by competitively binding to the active site of the protease enzyme. However, two of the PIs, Darunavir (DRV) and Tipranavir (TPV) also exhibit a secondary mode of action where it inhibits the dimerization of the protease. The mechanism by which these two PIs portray the dual mode of action is not yet understood. In this study, we utilized molecular docking, all-atom MD and accelerated MD simulation to uncover this mechanism. We identified an alternative pocket in the tail region of the protease monomer where DRV and TPV could bind stably. Ligand Gaussian Accelerated Molecular Dynamics was further used to elucidate the mechanism for their dual mode of action. Structural analysis revealed that binding to tail pocket induces a conformation change in the C-terminal region of protease monomer that make it less favorable for dimerization. These findings were further validation by in-silico mutagenesis studies. The results from this study could guide the development of new drugs that specifically target and disrupt the dimerization of HIV protease.

From Gut to Brain: *Helicobacter pylori*-Mediated Dysbiosis in the Pathogenesis of Neurodegeneration

Meenakshi Kandpal[1], Pranit Hemant Bagde[1], Sidharth Singh[1], Tarun Prakash Verma[1], Manivannan Elangovan[2], Hem Chandra Jha[1]

[1]*Infection Bioengineering group, Mehta Family School of Biosciences and Biomedical Engineering, Indian Institute of Technology Indore, MP, India;* [2]*School of Pharmacy, Devi Ahilya Vishwavidyalaya, Indore, MP, India*

Presenting Author: Meenakshi Kandpal

Abstract:

Helicobacter pylori, a common gastric pathogen, is increasingly recognized for its role in gut–brain axis disruption and neurodegeneration. Our studies demonstrate that H. pylori infection in gastric epithelial cells triggers inflammation and STAT3 activation, while its secretome elevates Alzheimer's disease (AD)-related markers in neuronal cells. Inhibition of pSTAT3 reduces neuroinflammation and amyloid pathology. Exposure to H. pylori secretome induces mitochondrial fission in neurons via upregulation of Drp1, Fis1, MFF, and Mid51 and downregulation of MFN1, resulting in decreased mitochondrial potential and mass, elevated intracellular Ca^{2+}, and excitotoxicity. In vivo, mouse brains show disrupted mitochondrial dynamics, altered lipid metabolism, and impaired peroxisomal function. Both antibiotic-sensitive and -resistant strains induce cognitive deficits, anxiety-like behaviours, and motor impairments, accompanied by gastric inflammation, leaky gut markers, neuronal damage, microgliosis, STAT3 activation, and upregulation of AD-related proteins (APP, ApoE4, p-tau, S100β, NEFL) in the mouse model. LC–MS-based metabolomics revealed strain-specific perturbations in steroid/fatty acid metabolism and neuronal glucose oxidation, while elevated N-methyl salsolinol, salsoline, and dopaminochrome indicate neurotransmitter dysregulation. Bioinformatic analysis of ~230 secreted proteins identified four proteins and eight peptides with amyloidogenic potential leading to AD.

ERK-mediated phosphorylation controls stability of cardiac poly(A) polymerase Star-PAP to regulate myocyte hypertrophy

Beauty Rani Koch[1,2], Rakesh S Laishram[1,2]

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Presenting Author: Beauty Rani Koch

Abstract:

Star-PAP is a non-canonical poly(A) polymerase located in nuclear speckles, regulated by PIPKIα, which polyadenylates select mRNAs involved in oxidative stress and cardiovascular diseases. Our lab uncovered key role of Star-PAP in cardiac condition such as cardiac hypertrophy where inherent downregulation of Star-PAP compromised anti-hypertrophy gene program leading to pathological condition. Yet, how Star-PAP is downregulated during hypertrophy or how protein expression is controlled is unknown. In this study, we show that Star-PAP functions downstream of ERK signal in hypertrophic myocyte and that ERK signaling negatively controls Star-PAP levels. Specifically, when ERK is active, Star-PAP is reduced. We discovered that ERK directly phosphorylates Star-PAP, especially within its zinc finger (ZF) domain. This phosphorylation event appears to be a critical signal for Star-PAP's degradation. Our experiments show that Star-PAP is degraded through a ubiquitin-mediated process. These findings were consistent in both cell line and animal model of cardiac hypertrophy, where we observed increased ERK activity and reduced Star-PAP level. Consistently, inhibition of ERK or the use of a Star-PAP phospho-mutant stabilized the protein and led to significant protection against hypertrophic induction in cardiomyocytes. In conclusion, our work reveals ERK-mediated phosphorylation leads to degradation of Star-PAP, crucial event in cardiac hypertrophy.

#PANIIT20250906 - Connect to Postdoc Mentors

Physico-chemical regulation of living matter across scales

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Presenting Author: Sreepadmanabh M

Abstract:

How physical environments regulate living matter remains an extensively explored, yet perpetually intriguing question. While biochemical approaches target specific molecular pathways, physics-of-life approaches focus on the non-linear dynamics of biological systems. My PhD research pioneers a new perspective establishing physical microenvironments as active regulators of living matter across scales. Towards this, we have innovated custom-engineered 3D culture platforms mimicking the spatial architecture of habitats such as soil, mucus, and tissues – resulting in universally-adaptable scaffolds for bioengineering and 3D bioprinting. Leveraging these, we have explored how biophysical constraints regulate growth, motility, morphology, and cellular states across scales, viz., demonstrating that 3D confinement acts as a potent selective pressure on bacterial communities; identifying a unique mode of physical control over cell division in budding yeast; establishing how viscoelastic regimes govern transitions in worm motility; characterizing environmental cues regulating the organization and plasticity of structurally-heterogeneous ovarian cancer spheroids; and discovering that the cellular state is an emergent property resulting from regulatory crosstalk between oxygen signalling and mechanical regimes. Together, our work pioneers generalizable physical principles describing the physico-chemical regulation of fundamental biological phenomena across complex 3D microenvironments.

#PANIIT20250105 - Connect to Post-Doc Mentors

Rapid somatic HCN dysfunction in epileptogenesis induces hyperexcitability in subicular burst-firing neurons

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Presenting Author: Monica Alfred

Abstract:

The subiculum, owing to its bursting nature, plays a crucial role in Temporal Lobe Epilepsy (TLE). Studying subicular neuronal subtypes can elucidate the mechanisms underlying epileptiform firing. In epilepsy pathophysiology, hyperpolarization-activated cyclic nucleotide-gated (HCN) channels display paradoxical conditions of up- and downregulation. However, the functional role of HCN channels in subicular neuronal subtypes in epileptogenesis remains unclear, which could drive targeted therapeutic interventions for TLE. We investigated the contribution of HCN to epileptiform activity in subicular neurons using patch-clamp electrophysiology and dynamic clamp in a 4-Aminopyridine (4-AP-0Mg) in vitro rat epilepsy model. We observed that epileptogenic 4AP-0Mg in a brain slice induced different patterns of epileptiform discharges in subicular burst firing neurons and interneurons. We further show that enhancing HCN conductance reduces the epileptogenic activity in subicular bursters, but not in interneurons. We further demonstrate that HCN in bursters undergo modulation during the initiation and progression of epileptiform activity within minutes, unravelling a novel mechanism of rapid homeostatic dysfunction. These changes appeared irreversible post 4AP-0Mg washout, culminating in neuronal hyperexcitability. We suggest that rapid HCN dysregulation is a critical modulator in epileptogenesis, and enhancing HCN function can counteract neuronal hyperexcitability and mitigate seizures.

#PANIIT20250938 - Connect to Post-Doc Mentors

TIRAP–c-JUN Signaling Links Macrophage–Hepatic Stellate Cell Crosstalk in Alcohol-Induced Liver Fibrosis

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Presenting Author: Pramod Patidar

Abstract:

Alcoholic liver disease (ALD) is characterized by chronic hepatic injury and excessive extracellular matrix (ECM) deposition leading to fibrosis. Activation of hepatic stellate cells (HSCs) represents a key fibrogenic event largely driven by macrophage-derived inflammatory mediators. This study elucidates the macrophage–HSC signalling mechanisms underlying HSC activation under alcohol-induced stress. THP-1-derived macrophages exposed to ethanol and lipopolysaccharide (LPS) exhibited heightened inflammatory activation, with increased secretion of pro-inflammatory and fibrogenic cytokines (IL-1β, TNF-α, IL-6, TGF-β, and PDGF-α). Mechanistically, ethanol and LPS exposure enhanced Toll-Interleukin-1 Receptor (TIR) Domain-Containing Adaptor Protein (TIRAP) phosphorylation, leading to activation of MAPKs and Nuclear Factor-kappa B (Nf-kB). Conditioned media from ethanol and LPS treated macrophages induced fibrogenic activation of HSCs, as evidenced by increased expression of α-smooth muscle actin (α-SMA) and collagen. Furthermore, the upregulation of α-SMA and collagen correlated with enhanced expression of HSC c-JUN, suggesting that c-JUN serves as a regulator of ECM production and the progression of ALD. Collectively, these findings demonstrate that macrophage–HSC crosstalk plays a pivotal role in driving alcohol-induced liver fibrosis, underscoring the importance of intercellular communication in ALD pathogenesis.

POSTER SESSION
DAY 1

A quadrangle π-π and π-H interdomain interaction contributes to the voltage-dependent facilitation (VDF) of L-type voltage-gated calcium channels

Aynal Hoque[1], Vishal Tanaji Khade[2], Abhijeet Kate[2], Giriraj Sahu[1]

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Presenting Author: Aynal Hoque

Abstract:

L-type Voltage-gated calcium (Cav1) channels possess a peculiar Voltage-dependent facilitation (VDF), in which depolarized prepulse increases the inward current by ~ 60-80%. Notably, it contributes to increased calcium entry during repetitive action potential firing in neuronal, cardiac, and endocrine systems. However, the molecular and biophysical mechanisms that generate Cav1 channel VDF remain unexplored. Coincidentally, we observed solvents stored in plastic tubes for 7-30 days abolished the VDF, enabling us to identify the leachable compounds that plays role behind VDF inhibition. Using whole-cell and single channel current recordings, we could document a drastic reduction in the Cav1 channel VDF by the identified phenolic leachable compounds in nanomolar range. In addition, docking and MD simulations revealed the compound stabilization in the domain I-II fenestration of the channels. The MSA indicated that interdomain quadrangle interaction exists between P loop and S6 segments of fenestration, which are absent in non facilitable Cav2.1 channels. Importantly, mutation of critical amino acids of quadrangle in Cav1.2 resulted in complete reduction of VDF. Alternatively, introduction of a single amino acid supporting quadrangle interaction in Cav2.1 displayed robust VDF. In conclusion, the study identified a novel interdomain quadrangle interaction that plays a crucial role in the development of activity-dependent VDF of Cav1 channels.

Genome-scale reconstruction of the chicken enables cell culture media design and toxicity assessment in cultivated meat manufacturing

Tejaswini Ganapathy[1,2], Yi Fan Hong[3,6], Kannan Ramesh[1], Jun Ping Quek[3], Yin Ying Ho[3], Dawn Leong[3], Tessa Tan[3], Esther Peh[3], Say Kong Ng[3], Ying Swan Ho[3], Kuin Tian Pang[3,4,5,6], Meiyappan Lakshmanan[1,2,3,7]

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Presenting Author: Tejaswini Ganapathy

Abstract:

Cultivated meat is an elegant source of sustainable protein with significant environmental, ethical, and sustainability benefits. Unlike traditional livestock farming, which contributes to greenhouse gas emissions, land degradation, and water usage, cultivated meat reduces ecological impact and eliminates animal slaughter. However, its commercial production faces challenges, particularly the high cost of cell culture media, which limits large-scale production. In this study, we reconstructed a comprehensive chicken genome-scale metabolic model and investigated the metabolism of DF-1 cells, an immortalized chicken embryonic fibroblast line, as a potential candidate for cultivated meat production. The maximum viable cell density attained by DF-1 cells is approximately six times lower than CHO cells, suggesting growth limitations. To understand the metabolic bottlenecks underlying reduced growth, we used genome-scale metabolic models along with transcriptomics and spent media analysis data, providing a comprehensive understanding of cellular metabolism. Beyond growth limitations, we used the chicken GEM to analyse the potential production of toxic metabolites by DF-1 cells as byproducts, enabling the design of purification processes to ensure safety for human consumption. Our findings support that media reformulation can not only increase VCD but also reduce toxic byproducts in cell culture, and enable more sustainable, cost-effective, and scalable cultivated meat production.

PIGBOS1 is a Novel Regulator of Calcium Dynamics and Mitochondrial Function

Seemanti Aditya[1], Amal Kanti Bera[1]

[1] *Department of Biotechnology, Bhupat and Jyoti Mehta School of Biosciences, Indian Institute of Technology Madras, India*

Presenting Author: Seemanti Aditya

Abstract:

PIGBOS1 is a recently discovered microprotein of 54 amino acids localized to the outer membrane of mitochondria and has been shown to modulate the endoplasmic reticulum (ER) stress response. PIGBOS1 knockout cells exhibit an enhanced ER stress response when exposed to ER stress inducing agents. Dysregulation of PIGBOS1 has been proposed in various diseases, including neurodegenerative disorders and cancer. In the present study, we investigated the role of PIGBOS1 in cellular calcium homeostasis by altering its expression levels in HEK293T cells. Overexpression of PIGBOS1 augmented calcium signaling, as evidenced by increased Ca^{2+} release from the ER through the IP3 receptor and enhanced mitochondrial Ca^{2+} uptake upon histamine stimulation. Conversely, siRNA-mediated knockdown of PIGBOS1 and PIGBOS1 knockout cells exhibited the opposite effect. The interaction between PIGBOS1 and the ER-localized chloride channel CLCC1 through its C-terminus was found to be crucial for its regulatory function. Deletion of the C-terminus abolished PIGBOS1's modulatory activity. In addition, loss of PIGBOS1 reduced mitochondrial oxygen consumption and ATP synthesis while increasing reactive oxygen species production. Overall, our findings identify PIGBOS1 as a key component of the cellular Ca^{2+} signaling network, linking ER-mitochondrial communication to energy metabolism and stress responses.

Impact of TUBB4B Tubulin Isotype Mutation on Microtubule Dynamics

Thasni Fazil [1], Kathiresan Natarajan *[1]

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Presenting Author: Thasni Fazil

Abstract:

Background: Microtubules have an exceptionally high degree of structural homology across all eukaryotic organisms, making them an important cytoskeleton component. They perform essential roles in critical cellular functions and thereby control cell dynamics and trafficking. During cell division, they play a pivotal role in separating DNA into daughter cells. These vital roles made microtubules a potential anti-cancer target. Several TUBB4B tubulin isotype mutations have been identified among lung cancer patients. The consequences of these mutations in lung cancer progression remain unclear. Thus, TUBB4B isotype mutations may play a significant role in lung cancer progression and potentially modify microtubule dynamics, impacting cancer. Methodology: The study examined the effect of lung cancer-related TUBB4B tubulin isotype mutation on microtubule dynamics using molecular dynamic simulations, site-directed mutagenesis, FACS, and microscopy studies. Results and Conclusion: The study reveals a surprising role of the TUBB4B isotype in lung cancer. TUBB4B mutation alters tubulin structure and dynamics of β tubulin and allosterically amplifying the mutational effects in the tubulin dimer simulations. Experimental studies confirm these mutations alter the polymerization dynamics of microtubules, revealing how subtle changes in TUBB4B tubulin isotype can affect microtubule dynamics and lead to pathological conditions.

Preclinical Safety Studies With Gene-Edited Hematopoietic Stem And Progenitor Cells For The Treatment Of B-Hemoglobinopathies

Abhijith K [1,2], Saravanabhavan Thangavel [1]

[1] *Centre for Stem Cell Research, A unit of BRIC - inStem, Bengaluru, Christian Medical College Campus, Bagayam, Vellore*

[2] *Manipal Academy of Higher Education, Manipal*

Presenting Author: Abhijith K

Abstract:

Reactivation of fetal hemoglobin (HbF) is a well-recognized therapeutic strategy for ameliorating β-hemoglobinopathies. However, the persistence of aberrant adult hemoglobin continues to limit HbF tetramer formation, thereby reducing therapeutic efficacy. We identified an 11-kb core region, extending from the putative repressor region (PRR) to β-globin exon 1 (βE1), as a critical regulatory sequence. Deletion of this region completely ablated HbA expression and, induce HbF compared with naturally occurring HPFH mutations or other established genomic targets. Since Cas9 induced double-strand breaks can lead to unintended genome-wide consequences such as chromosomal rearrangements, large deletions, or translocations it is necessary that to check the genomic integrity and safety of the approach for clinical translation. The objective of this study was to comprehensively evaluate the genomic stability and safety of PRR-βE1–edited HSPCs. To assess the genomic integrity of PRR-βE1–edited HSPCs, we performed a series of complementary safety assays like nuclear stability analysis like micronuclei formation, array-based KaryoStat, CAST-Seq and copy number variation. The result suggesting that minimal genotoxic stress and no detectable loss of DNA and chromosomal translocation. Collectively, these results indicate that PRR-βE1 deletion is a potent strategy for HbF induction with a strong preliminary safety profile.

Identification and characterisation of small RNA regulators in Helicobacter pylori infection and pathogenesis

Prasmita Paul [1], Bivash Sinha [1], Ahana Sarkar [1], Shrihari D. Katti [1], Oindrilla Mukherjee [1]

[1] *Department of Biotechnology, National Institute of Technology, Durgapur, Mahatma Gandhi Rd, A-Zone, Durgapur, West Bengal 713209, India*

Presenting Author: Prasmita Paul

Abstract:

Helicobacter pylori is a gram-negative, microaerophilic bacteria causing persistent inflammation leading to chronic gastritis and gastric adenocarcinoma. Recent studies highlight small non-coding RNAs as important regulators of bacterial virulence. These short transcripts regulate gene expression at the transcriptional and post-transcriptional levels, thereby significantly influencing bacterial adaptation and pathogenicity. Our study aims to identify putative small RNAs involved in H. pylori infection and elucidate their regulatory functions. An integrative bioinformatics workflow was employed, wherein CopraRNA was used for the comparative prediction of small RNA targets and their mRNA targets were further refined through IntaRNA-based interaction mapping. Targets with p-value < 0.05 were considered significant. Three sibling sncRNAs were identified as key candidates which were associated with the deregulation of several bacterial adhesins, ABC transporters, and outer membrane proteins. Functional analysis revealed their significance in bacterial adhesion, colonisation, stress tolerance and persistent inflammation. Further validation of these findings using sncRNA-deficient H. pylori mutants is being employed to establish the targets as potential regulators of host colonisation, immune modulation, and infection persistence. Establishing this sncRNA–mRNA axis may reveal a novel mechanistic layer in bacterial pathogenesis and open new avenues for RNA-based therapeutics.

From Embryos to Organoids: Modelling Development to Decode Disease

Nitya Nandkishore [1,2,3], Ramkumar Sambasivan [2,4], Fabienne Lescroart [3]

[1] *SRM Institute of Science and Technology, Kattankulathur*
[2] *inStem, Bengaluru*
[3] *Marseille Medical Genetics, Aix-Marseille University*
[4] *Department of Biology, Indian Institute of Science Education and Research Tirupati, Andhra Pradesh, India*

Presenting Author: Nitya Nandkishore

Abstract:

Gastrulation is a critical early developmental stage when a single layer of cells generates three primary germ layers and establishes the body axes that specify the coordinates for the adult body plan. Environmental, metabolic or genetic perturbations at gastrulation lead to long-lasting impacts that manifest as congenital diseases and/or developmental defects. Studying gastrulation in vivo is challenging, but crucial, to examine intricate cell migratory patterns and complex signaling environments. This information can then be used to construct in vitro models such as 3D gastruloids, that can mimic the key processes of gastrulation, and model disruptions that occur in disease. Using mouse embryo cultures, we uncovered signaling pathways essential for normal development of the different chambers of the heart, and that specify the cardiopharyngeal mesoderm which contains progenitors for the both the heart and skeletal muscles of the head, face and neck. Using directed differentiation assays coupled with gene and protein expression studies, we derived this population in vitro, both in 2D and 3D, and single-cell sequencing was used to characterize the various progenitor populations present. With the initiation of my independent research group, I am interested to establish in vitro models that explore the environmental influences that underlie congenital defects.

Tug-of-War between Stability and DNA-Binding Modulates Phase Separation in Yeast HMG Proteins

Shilpi Laha[1], Athi N Naganathan[1]

[1] *Department of Biotechnology, Bhupat and Jyoti Mehta School of Biosciences, Indian Institute of Technology Madras, India*

Presenting Author: Shilpi Laha

Abstract:

High Mobility Group proteins (HMG) act as central modulators of chromatin architecture and nucleosome dynamics. Apart from histones, they are the most abundant genome-associated proteins in Saccharomyces cerevisiae and function at high macromolecular crowding density within nucleus. They are also subjected to extensive post-translational modifications, including phosphorylation and acetylation. In this work, we employ Nhp6A and Nhp6B, a class of paralogous non-specific DNA-binding proteins with similar stabilities and folding relaxation rates, to explore the role of crowding and phospho-mimetic mutations on stability and phase separation tendencies. These paralogs are characterized by intrinsically disordered N-terminal tail enabling multivalent interactions with DNA. We find that phospho-mimetic mutations act as a rheostat to progressively destabilize the native ensemble. On the other hand, the effect of phosphorylation is context-dependent leading to no change or weakened binding to DNA, irrespective of the effects on stability. This observation is carried over to phase separation - mutations that weaken DNA binding lead to less phase separation, irrespective of the degree of structural order, with Nhp6B and mutants displaying a higher tendency to phase separate. Our work uncovers molecular mechanisms through which access to DNA can be tuned by HMG proteins through an intricate interplay between disorder and phase-separation.

Next-Generation β-Glucosidases : Enhancing Stability and Glucose Tolerance Therapeutic Biocatalysts

Ms. Yashvi Soni[1], Dr. Ankit Sudhir[2]

[1] *Department of Biotechnology, School of Science, GSFC University, Vadodara, Gujarat, India*
[2] *Senior Assistant Professor, Department of Biotechnology, School of Science, GSFC University, Vadodara, Gujarat, India*

Presenting Author: Ms. Yashvi Soni

Abstract:

β-Glucosidases (BGLs) (EC 3.2.1.21) hydrolyse β-glycosidic linkages, releasing glucose and bioactive aglycones. These enzymes act as crucial molecular bridges supporting environmental biorefineries and human health. BGLs are essential for Enzyme Replacement Therapy (ERT), treating lysosomal storage disorders such as Gaucher disease (defective glucocerebrosidase/GCase activity). They also facilitate drug activation in prodrug cancer therapies, detoxification of cyanogenic glycosides, bioconversion of therapeutic glycosides (e.g., ginsenosides, isoflavones) into readily absorbed aglycones, and modulate gut microbiota via oligosaccharide (GOS) synthesis. Sources like Aspergillus niger, Trichoderma reesei, Thermotoga maritima, and Lactobacillus spp. are utilized. Notably, Bacillus subtilis is key for producing thermostable BGLs, with production often optimized using signal peptide strategies. However, native BGL utility is constrained by limitations: low catalytic activity under physiological conditions, poor thermal stability and severe product inhibition (glucose/cellobiose). Present study focuses on applies protein engineering, specifically rational design, to enhance BGL catalytic efficiency, stability, and glucose tolerance. This approach aims to generate robust BGL variants, promising improved bioactivity for pharmaceutical synthesis and advanced health- oriented products.

#PANIIT20250952- Poster Session, Day 1

WGS driven predictive combination of antibiotics for precision-based therapy in Drug-resistant *Acinetobacter baumannii*

Purvi Trivedi[1]*, Siddhi Darji[1], Devjani Banerjee[1]

[1] *School of Sciences, GSFC University, Vadodara, Gujarat, India*

Presenting Author: Purvi Trivedi

Abstract:

Acinetobacter baumannii is an opportunistic pathogen increasingly implicated in severe nosocomial infections in immunocompromised patients. With its multi drug resistant nature, it poses global threat with approximately 20% of ICU-acquired infections. The World Health Organization has designated carbapenem-resistant *A. baumannii* (CRAB) as a critical priority pathogen for novel antibiotics development. Resistance in *A. baumannii* arises through diverse mechanisms, including efflux pump overexpression, porin alteration, target modification, and enzymatic degradation, facilitated by the acquisition of mobile genetic elements carrying carbapenemases such as NDM, VIM, and OXA variants. As new antibiotic development is challenging, the combination drug therapy is preferred approach.

Here, 50 WGS of *Acinetobacter baumannii* isolates from Gujarat were analysed as part of GSBTM sponsored state-wide AMR surveillance under the One Health initiative. Typing analysis was done using Pasteur and Oxford scheme and Resistance profiling was done using ResFinder pipeline. Absence of specific resistance genes across antibiotic classes enabled the identification of potential therapeutic agents. A rational triple and double antibiotic combination therapy was proposed based on comparative genomic resistance profiling for optimized clinical application.

Based on the WGS analysis, 5 different double-antibiotic combinations were finalised for testing. 1 out of these 5 showed significant results. The result analysis will be shared during presentation.

Evaluating the effect of CHO endogenous promoters and enhancers to improve expression efficiency and stability

Aravindha Anjana Padmanaban[1], Debabrata Mohanty[1,2], Meera Alagu Sundari P M[1], Meiyappan Lakshmanan[1,2,3,4]

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Presenting Author: Aravindha Anjana P

Abstract:

Chinese Hamster Ovary (CHO) cells are the predominantly used mammalian cells in biotherapeutics production, creating an inevitable need for higher gene expression rates, because of which strong viral-regulatory elements are being used. As the cells tend to protect their genomic integrity, they silence such foreign genes by epigenetic modifications, causing a drop in productivity levels. This could be overcome by replacing them with endogenous promoters and enhancers. Previous studies have reported a few potential endogenous promoters and enhancer candidates of highly expressed genes, found by RNA seq data which can be used in this context. Our objective is to identify novel promoters and enhancers using a bioinformatic pipeline and to investigate the effect of novel endogenous promoters in comparison with a strong viral promoter like CMV, and the effect of novel endogenous promoters+ enhancers combinations in comparison with CMV promoter+ enhancer, by assessing the expression of GFP reporter. In addition, their effects will be evaluated by assessing the expression levels of recombinant proteins like human tissue plasminogen activator, interferon, and finally with mAbs. Thus, this study will provide us with novel and highly efficient endogenous promoters and enhancers, which will ensure prolonged and higher transgene expression levels.

#PANIIT20250955- Poster Session, Day 1

Comparative genomics guided discovery of pathway targets against Multidrug-Resistant *Mycobacterium tuberculosis* and screening of essential oil phytochemicals

Ashutosh Sahoo[1], Shagun Shagun[1], Nidhi YadavY[2], Rithika Pai[1#], Amay Sandilya[1#], Ranjan Kumar Nanda[2]*, Shyam Kumar Masakapalli[1]*

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Presenting Author: Ashutosh Sahoo

Abstract:

The emergence of multidrug-resistant *Mycobacterium tuberculosis* (MDR-Mtb) poses a major challenge to global tuberculosis control, underscoring the urgent need for novel therapeutic strategies. This study employed comparative genome and metabolic pathway analyses of six clinical MDR-Mtb strains from North-East India, alongside twenty reference strains representing diverse Mtb lineages, to identify conserved metabolic vulnerabilities suitable for drug discovery. SNP based conservation analyses were carried for validation of the targets across the MDR strains. The study identified the methylerythritol phosphate and biofilm biosynthesis pathways as conserved and could be promising drug targets across Mtb lineages. Further, selected Essential oils (EOs) catalogued in an inhouse repository of Himalayan Medicinal & Aromatic Plant and Phytochemicals database were screened against *Mycobacterium smegmatis* to assess antimycobacterial and antibiofilm activities. Phytochemical profiling using GCMS and NMR (1H/13C) was carried as well as ADMET properties of the identified small molecules were predicted. Among the tested oils, Ajowan oil demonstrated potent antimycobacterial and biofilm inhibitory activity, highlighting its phytochemicals as promising candidates for antimycobacterial drug development. Ongoing efforts focus on isolating and characterizing the active components and assessing their effects on MDR-Mtb strains to advance lead optimization for therapeutic applications.

Mechanistic Insights and Fragment-Based Evolutionary Design: Next-Generation CETP Inhibitors for Cardiovascular Therapeutics

Sudipta Nandi[1], Sanjib Senapati[1]

[1] *Department of Biotechnology, Indian Institute of Technology Madras, Chennai, Tamil Nadu*

Presenting Author: Sudipta Nandi

Abstract:

Cardiovascular diseases remain a leading cause of mortality globally. Increasing HDL levels by inhibiting Cholesteryl Ester Transfer Protein has become a promising therapeutic strategy. Over the past few decades, extensive researches have focused on discovering effective CETP inhibitors that advanced to clinical trials yet failed. To address these, we performed docking and molecular dynamics simulations comparing CETP bound complexes of Torcetrapib and its derivative Obicetrapib, an ongoing clinical candidate demonstrating improved therapeutic potential. Obicetrapib exhibited superior affinity and reduced protein flexibility, emphasizing the importance of favourable electrostatic and hydrophobic interactions. Concurrently, 'good' and 'bad' structural moieties influencing CETP inhibition, are identified using QSAR and ML analysis. Leveraging these insights, an innovative fragment based evolutionary design pipeline was implemented, followed by a regression based ML and genetic algorithms to redesign existing scaffolds and generate structurally diverse inhibitors. Docking and simulations of top compounds validated their potential efficacy, offering key insights into inhibitory mechanisms and further investigation via energy profile analysis using advanced MD. This integrative computational strategy provides an enhanced understanding of CETP inhibition at molecular level and establishes a robust platform for generating novel inhibitors for therapeutic advancements in CV medicine.

HOCl-Induced Oxidative Stress for Improvement of Phycocyanin Production in Synechococcus elongatus UTEX2973 under Simulated Microgravity Conditions

Radhika K[1], G K Suraishkumar*[1]

[1] *Department of Biotechnology, Indian Institute of Technology Madras, Chennai, India*
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Presenting Author: Radhika K

Abstract:

In-space biomanufacturing provides a sustainable platform for space-mission by reducing the dependency on Earth-based resources. The microgravity environment in space is known to alter various microbial characteristics, such as growth, metabolism, and gene expression. Further, under microgravity, the oxidative stress results from an imbalance in the rates of reactive oxygen species (ROS) production and utilization within a cell, which can lead to cellular and tissue damage. However, ROS are also essential for cell signalling, metabolism, and maintaining cell homeostasis. Algae can be promising candidates for space-mission because they produce food, oxygen, and water through photosynthesis. Also, they can produce valuable biomolecules and bioactive compounds, with applications in various industries. Although microgravity has shown to alter algal growth and photosynthesis, experimental data on these effects remain limited, and the mechanisms underlying the microgravity-induced ROS effects on algae remains unclear. This study investigates the effects of ROS on Synechococcus elongatusUTEX2973 under simulated microgravity conditions and the effects of induced ROS on the production of phycocyanin, high-value compound used in pharmaceuticals, therapeutics, and food industries. We found that inducing ROS with an optimum HOCl level of 45µmole HOCl/ g cell in inoculum increased phycocyanin specific yield by 37% under simulated microgravity compared to the control.

Ionic Liquids as Molecular Stabilizers for Nucleic Acids: Achieving Long-Term Structural and Functional Stability at Room Temperature

Ananya Asmita[1], Kavin Chakravathy[1], Sanjib Senapati[1]

[1] *Department of Biotechnology, Indian Institute of Technology Madras, Chennai, Tamil Nadu*

Presenting Author: Ananya Asmita

Abstract:

The reliable preservation of nucleic acids under ambient conditions remains a critical challenge for molecular biology, diagnostics, and RNA-based therapeutics. Their inherent susceptibility to enzymatic degradation and thermal instability necessitates energy-intensive cold-chain systems that hinder large-scale deployment. This work explores a novel class of green solvents, Ionic liquids (ILs), and their aqueous binary mixtures as alternative media for room-temperature stabilization of DNA and RNA without compromising biological functionality. Spectroscopic and electrophoretic analyses show that select ILs maintain DNA structural integrity for over one year and RNA stability for three months. Functional assays confirm that IL-stored plasmid DNA retains activity in bacterial transformation and mammalian transfection, while total RNA recovered from IL mixtures remains fully amplifiable by RT-PCR. All-atom molecular dynamics simulations reveal that IL cations form dynamic hydrogen-bond and electrostatic networks with the nucleic acid backbone, constraining structural fluctuations and preventing denaturation. This study establishes IL-water binary mixtures as a robust platform for cold chain-free, long-term preservation of functional nucleic acids, providing mechanistic insight and a foundation for developing ambient-stable genetic materials and mRNA therapeutics.

Success rate and variability of Diabetes induction by High-Fat Diet in male C57BL/6J Mice

Amit Kumar[1], Sivapriya Sivagurunathan[1], and Madhulika Dixit*[1]

[1] *Department of Biotechnology, Bhupat and Jyoti Mehta school of Biosciences, Indian Institute of Technology Madras, Chennai 600036, India*

Presenting Author: Amit Kumar

Abstract:

Type 2 diabetes is a complex metabolic disorder often driven by obesity, insulin resistance, and dietary factors. High-fat diet (HFD) feeding is a common strategy to model type 2 diabetes in rodents (Burcelin et al. 2002; Stott and Marino 2020; Weichhart 2013). To establish this model, male C57BL/6J mice were randomized to either a normal-fat diet (NFD) (10 kcal% fat) or HFD (60 kcal% fat) for 20 weeks. HFD-fed mice showed significant body weight gain from week 6 onward compared to control. We also observed a significant increase in fasting body weight from week 12 onward in HFD-fed mice. Oral glucose tolerance tests (OGTT) revealed impaired glucose tolerance in a subset of HFD mice in comparison to NFD mice. Based on OGTT AUC and body weight, 38.89% of HFD mice developed prediabetes (including one lean-prediabetic) and 22.22% progressed to overt diabetes, while 38.89% remained metabolically normal; thus, the overall success rate of diabetes/prediabetes induction was ~61.11%. Although monthly fasting glucose remained unchanged, total insulin ($p=0.0497$) and total cholesterol ($p=0.0371$) level were significantly elevated in HFD-fed mice. These findings demonstrate that 20 weeks of HFD feeding induces obesity, dyslipidemia, and impaired glucose tolerance in a majority of C57BL/6J mice, establishing a reliable diet-induced model of type 2 diabetes.

Role of VPS52 in neurodevelopment and membrane trafficking

Tamanna Saini[1], Krittika Biswas[1] , DVS Sudhakar[2], Iyengar Vaishnavi Venkatachari[3], Tanya Jain[4], S. Gopalan Sampathkumar[4], Bhavani Shankar Sahu[1]*

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Presenting Author: Tamanna Saini

Abstract:

VPS52 is a common subunit of the GARP and EARP complexes, and mutations in these complexes have been linked to several neurodevelopmental defects. Previously, VPS52 mutation caused embryonic lethality in mice, highlighting its critical role in development. We identified a patient with VPS52 deficiency presenting with persistent microcephaly, cerebellar hypoplasia, delayed speech and social development, autism spectrum disorder, and subclinical seizures, consistent with severe neurodevelopmental impairment. To investigate underlying mechanisms, we generated VPS52 knockout in PC12 and HT22 cells using CRISPR/Cas9. Because glycosylation is essential for cell–cell signaling in brain development, we examined key glycosylation enzymes. VPS52 loss caused mislocalization of Mannosidase II and Galactosyltransferase, indicating impaired glycan processing. Furthermore, regulated secretion of NPY paradoxically increased while constitutive secretion decreased, as seen by decreased HSP90 release. However, NPY and CgB accumulated in lysosomes, indicating that the cargo of dense-core vesicles was missorted and that this was accompanied by impaired calcium influx, reflecting disruption of both calcium-dependent exocytosis and trafficking. Together, our results reveal a previously unrecognized role of VPS52 in maintaining glycosylation, calcium signaling and secretory pathway integrity, providing mechanistic insight into how membrane trafficking defects underlie rare neurodevelopmental disease.

#PANIIT20250972- Poster Session, Day 1

Temporal Shear Dynamics as an Independent Regulator of Epithelial Mechanotransduction in Straight Microchannels

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Presenting Author: Supratim Saha

Abstract:

Mechanical forces such as fluid shear stress (FSS) critically regulate epithelial physiology, yet how their temporal patterns influence cell behavior in simple geometries remains unclear. Here, we investigate renal epithelial mechanotransduction in a straight microfluidic channel designed to apply identical mean shear magnitudes under distinct flow regimes: continuous flow (500 $\mu L\ h^{-1}$) and intermittent flow (500 $\mu L\ h^{-1}$ with 1 h ON/1 h OFF cycles), compared against static culture. Using live-cell calcium imaging and fixed assays for F-actin, ZO-1, and nuclear morphology (DAPI), the study explores how time-varying FSS modulates intracellular signaling, cytoskeletal remodeling, and junctional organization. The simplified channel geometry allows isolation of temporal shear effects from curvature-induced spatial gradients, enabling a fundamental analysis of epithelial mechanoadaptation. We hypothesize that intermittent FSS provides periodic mechanical recovery that alters calcium oscillation dynamics and actin-junction coupling compared to steady shear. This approach introduces a minimal yet mechanistically rich model to dissect how temporal shear dynamics alone can regulate barrier integrity and nuclear mechanotransduction advancing the design of physiologically realistic kidney-on-chip platforms for One-Health–aligned bioengineering applications.

Crude root extracts of *Sida cordifolia* rescues motor, cognitive and neuropathological deficits in Huntington's Disease Mouse Model by ameliorating mHTT protein aggregates

Puyam Milan Meitei[1], Shreya Kumari[1], Chandramouli Mukherjee[1], Ankita Roy[1], Ankit Sharma[1], Prasanna K Simha[2], Ashwini Godbole[3], Sanjeev K Upadhyay[2,3], Bhavani Shankar Sahu[1]

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Presenting Author: Puyam Milan Meitei

Abstract:

Introduction: Huntington's Disease (HD) is an autosomal dominant neurodegenerative disease, caused by a mutation in exon 1 of huntingtin (HTT) gene, producing mutant HTT (mHTT) proteins, which form insoluble aggregates in nucleus and cytoplasm and cause neuronal death. *Sida cordifolia (S. cordifolia)* is a tropical perennial shrub, widely used as a medicinal plant in traditional medicinal system. It has been shown to have therapeutic effects in neurodegenerative diseases such as Parkinson's Disease. Objectives: 1. To investigate the effects of *S. cordifolia* in motor and cognitive deficits of R6/2 mice by assessing various behavioural assays 2. To investigate its therapeutic effects on the anatomical defects, mHTT protein aggregates load and various key physiological defects in R6/2 mice Key Findings: 1. *S. cordifolia* prevented motor deficits in R6/2 mice such as prevented hindlimb clasping and gait defects, improved rotarod and balance beam performance as well as prevented cognitive deficits such as improved performance in novel object location and recognition tests 2. *S. cordifolia* ameliorated mHTT protein aggregates in the cortex, hippocampus and striatal brain regions of R6/2 mice. It also significantly prevented striatal atrophy. It significantly reduced mitochondrial fragmentation, neuroinflammation, restored expression of synaptic proteins and health of medium spiny neurons.

Identification of a novel pathway against HD disease using a naturally inspired compound

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Presenting Author: Shreya Kumari

Abstract:

Huntington's disease is an autosomal-dominant polyQ disorder caused by a mutation in the HTT gene. It is characterised by motor dysfunction, cognitive decline and emotional disturbances. Expanded CAG repeats lead to the formation of protein aggregates primarily in medium spiny neurons of the striatum region, causing neuronal death. Plant derivatives or their modified forms have shown promising neuroprotective effects. β-phenylethylamine, a naturally occurring trace amine abundantly present in medicinal plants, is a natural ligand for the TAAR-1 receptor and is decreased in HD. The dysregulation of trace amines has been linked to various neurodegenerative and psychiatric disorders such as schizophrenia, depression, Parkinson's disease, attention deficit hyperactivity disorder (ADHD) and migraine. We aimed to study the neuroprotective role of β-PEA in pre-clinical HD model cellular and animal models. We found that β-PEA was effective in reducing GFP-linked tNHTT in Neuro-2a cell lines with 150 polyQ repeats. It has also been shown to improve motor function and to ameliorate mHTT protein aggregates in the brains of R6/2 mice. We further aim to study the molecular mechanism of β-PEA-mediated neuroprotection, thereby uncovering novel therapeutic targets for HD.

Beyond the Vesicular trafficking: Novel role of Chromogranin B in regulation of Autophagy and Proteostasis

Palki Chauksey[1], Chandramouli Mukherje[1], Sourav Chatterjee[1], Bhavani Shankar Sahu[1]

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Presenting Author: Palki Chauksey

Abstract:

Chromogranin B (ChgB), a key dense core vesicle protein, is well recognized for its role in the regulated secretory pathway. Early studies have indicated that the loss of granin family proteins can lead to cytoplasmic accumulation of glycogen granules, suggesting defects in glycogen catabolism. However, the mechanisms underlying this phenomenon remain largely unexplored, and the potential link between granins, metabolism, and pathogenic consequences at the organismal level is still enigmatic. Our study provides critical insights into this gap by demonstrating that ChgB's absence disrupts autophagic processes. Using ChgB-knockout neuroendocrine cell lines as a model, we observed a pronounced impairment in overall autophagic flux, highlighting a previously unexplored role for this well-known granin in autophagy regulation. Extending our investigation to a cellular model of Huntington's disease, we found that CgB overexpression protects against mutant huntingtin aggregation, likely by facilitating autophagic clearance and restoring cellular homeostasis. Further analysis in CgB KO cells revealed potential mechanisms involving lysosomal stability and autophagosome formation. To our knowledge, this represents a novel study linking a classical secretory granin to autophagic regulation and metabolic homeostasis. These findings hold promising translational potential for treating conditions such as metabolic or neurodegenerative diseases.

Syntaxin 6 orchestrates dense-core vesicle function through coordinated regulation of vesicle biogenesis, cargo sorting, and exocytosis

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Presenting Author: Mohima Mukherjee

Abstract:

Neuroendocrine secretion is a key regulator of metabolic physiology and function. STX6 is a t-SNARE linked to secretory granule release. Although studies have suggested its role in intracellular trafficking, its precise function in neuroendocrine secretion remains unclear. We have generated Stx6 knockout cells in PC12 and INS1 using CRISPR/Cas9 and characterized their secretory status. Loss of Stx6 impaired regulated secretion of CGB and NPY. Interestingly, STX6 mutants lacking the C terminus could not reverse this secretory defect. RUSH assays revealed a delay in NPY exit from the TGN, while constitutive VSVG cargo trafficking was unaltered. Ultrastructural analysis revealed smaller, denser DCV cores, positioned farther from the plasma membrane, suggesting defective maturation and docking. STX6 deficient cells exhibited reduced levels of SNAREs and granins, with the latter being missorted to lysosomal degradation. Additional experiments ruled out calcium flux and fusion pore defects, indicating that Stx6 doesn't regulate these key steps of DCV function. Together, our findings suggest that Stx6 is a key regulator of DCV biogenesis, cargo sorting, and exocytosis, implicating its role in governing the basic pathophysiology of neurodegenerative diseases linked to secretory dysfunction, such as insulin granule exocytosis.

VAMP-2 SUMOylation causes impaired dense-core vesicle exocytosis in obesity-associated metabolic stress

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Presenting Author: Krittika Biswas

Abstract:

Obesity is associated with an overload of free fatty acids (FFAs) in the body, leading to altered metabolic homeostasis. The adrenal medulla (AM) maintains metabolic homeostasis by releasing catecholamines from dense-core vesicles (DCV), but its functions are often impaired in obesity. We found that mice fed high-fat diet (HFD) had reduced DCV exocytosis. When PC12 cells were treated with palmitate (a fatty acid increased in obesity) to mimic obesity-induced metabolic stress, we observed a similar reduction in DCV release. HFD-fed mice or palmitate-treated PC12 cells showed fewer docked vesicles at plasma membrane. Using global SUMOylation inhibitor, we could reverse the impaired DCV secretion in palmitate-treated PC12 cells. We identified VAMP2 as novel target of SUMOylation which showed a different subcellular distribution. Expressing a SUMO-resistant VAMP2 in both chromaffin cells from high-fat-diet-fed animals and palmitate-treated PC12 cells rescued impaired DCV secretion. These results suggest that VAMP2 is hyper SUMOylated in obesity and that SUMO-VAMP2 negatively regulates DCV release, thus identifying a plausible mechanism for the blunted AM function in obesity. These findings will advance our knowledge in metabolic physiology, and offers potential therapeutic solutions for clinical conditions contributed by the neuroendocrine and sympathetic systems, such as diabetes, obesity and hypertension.

Melatonin Protects Cardiac Tissue from Chemotherapy-Induced Oxidative and Structural Damage in Wistar rats

Souradipta Chakraborty[1,3], Swaimanti Sarkar[1], Razia Khatoon[1], Arefa Jasmin[2], Sarmistha Sarkar[1], Debasish Bandyopadhyay[1,4]

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Presenting Author: Souradipta Chakraborty

Abstract:

Cyclophosphamide, a chemotherapeutic used for cancers such as sarcoma and breast cancer, has strong anti-tumor effects but is limited by dose-dependent cardiotoxicity that can raise mortality. Melatonin (MEL), an endogenous antioxidant synthesized by the pineal gland, has demonstrated protective effects in various cardiovascular disorders. The present study investigated the potential cardioprotective effects of melatonin against CP-induced injury in male Wistar rats. Rats were treated with melatonin via oral gavage, followed by a single intraperitoneal dose of CP. Cardiac tissues were analyzed for oxidative stress, apoptosis, and structural changes using biochemical assays, flow cytometry, histology, and SEM. CP caused marked oxidative, apoptotic, and structural cardiac damage. Melatonin co-treatment promoted antioxidant status, and reduced apoptotic alterations. Histological, Masson's trichrome, and SEM analyses demonstrated that melatonin preserved myocardial architecture and collagen organization. Flow cytometry data supported a reduction in cellular oxidative and apoptotic alterations in melatonin-treated groups. These key findings suggest that melatonin offers protective effects against CP-induced cardiotoxicity by attenuating oxidative stress, inhibiting apoptosis, and preserving cardiac structure. Further studies are ongoing to delve deeper into the molecular mechanisms underlying this protection.

Melatonin as a protective adjuvant against doxorubicin-induced cardiotoxicity and ensuing perturbations in cardiac riboflavin metabolism

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Presenting Author: Sarmistha Sarkar

Abstract:

Doxorubicin (Dox) is one of the most extensively used antineoplastic drug in treating breast cancer, leukemia, pediatric cancer etc. However, its wide clinical applications are restricted due to its acute cardiotoxicity leading to increased risks of mortality in cancer patients. Increased oxidative stress is one of the several molecular mechanisms of Dox-induced cardiotoxicity. In this study the effects of Dox in cardiac riboflavin metabolism have been investigated in murine model. Riboflavin metabolism is crucial for maintaining metabolic homeostasis in the body. The protective efficacy of melatonin, an endogenous indole-amine has been explored in mitigating the deleterious effects of Dox in cardiac tissue. Dox administration resulted in alterations in cardiac riboflavin status and inhibition in the activities of riboflavin metabolizing enzymes in rats. These changes were accompanied with elevation in oxidative stress biomarkers and altered activities of antioxidant enzymes along with tissue-morphological changes and collagen deposition in heart. Isothermal titration calorimetric study revealed the spontaneous binding of Dox with FMN and FAD, the cofactors of riboflavin. Melatonin significantly protected the cardiac tissue from Dox-induced perturbations in riboflavin metabolism. Melatonin also mitigated antioxidant imbalance, disruption of mitochondrial membrane potential and tissue-morphological changes proving its efficacy as a cardio-protective therapeutic intervention.

Inhibitory Circuits In Retina Are Critical For Image Stabilization During Global Motion

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Presenting Author: Narendra Pratap Singh

Abstract:

To resolve objects in a constantly changing visual environment, it is essential to stabilize the image on the retina. Mechanisms for image stabilization include head and eye movements that cancel out the global motion in the visual scene. However, the neuronal circuitry involved in image stabilization is still not fully understood. In this study, we evaluated the image stabilization mechanisms in mice by monitoring their head movements in response to global motion within a custom-built behavioral arena. We found that head movements were triggered by globally moving naturalistic stimuli at speeds of up to 30º/sec, beyond which responses decreased rapidly. This stabilization is facilitated by circuits tuned to speed, which operate independently of the spatio-temporal characteristics of the visual scene. We altered the activity of these circuits under two conditions: a) by adjusting ambient light to simulate night-time conditions, and b) by pharmacologically blocking the retinal glycinergic receptors through intravitreal injections. In both scenarios, we observed a significant reduction in head movements in response to global motion. These findings led to two key conclusions: i) glycinergic inhibitory circuits in the retina are crucial for driving image-stabilizing head movements, and ii) these circuits are modulated by ambient light conditions.

Harnessing Intrinsic Glutaraldehyde-Protein Fluorescence for Breakthrough Sensing

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Presenting Author: Alisha Wadud Mondal

Abstract:

Recent studies on fluorescence-based imaging offer a better alternative to conventional immunohistochemistry diagnosis. However, most fluorescence systems depend on external dyes or contrast agents, with possible risks of photobleaching and toxicity. Considering these challenges, we have developed a self-reporting, label-free system that integrates identification and signal generation in a single, biocompatible platform without needing nanofabrication or external fluorophores. Our work introduces a novel self-illuminating bio-interface that harnesses the intrinsic properties of glutaraldehyde-protein conjugate, which is further used for selective biological recognition. The application is executed by properly functionalizing the prepared bio-interface and using it to target CTCs from the analyte. The binding triggers localized fluorescence into the cells. This results in distinct, cell-specific fluorescence, enabling real-time visualization and quantification of targeted cells without external labels under a standard fluorescence microscope. We observed strong, distinct membrane-localized fluorescence in MCF7 cells but negligible background in mixed populations of white blood cells and normal HEK293 kidney cells. Moreover, spectroscopic studies were in confirmation with the fluorescence emission results and showed changing intensities with different concentrations. Future work will focus on detecting other diseases and therapeutic delivery by integrating drug to the interface.

The Role of Lactate in Tunneling Nanotube Formation in Glioblastoma Cells

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Presenting Author: Subashree Anand

Abstract:

Tunneling nanotubes (TNTs) form dynamic intercellular networks that facilitate the direct exchange of cellular contents and signaling molecules, thereby contributing to tumour progression and therapy resistance. The Warburg effect, a metabolic hallmark of cancer cells, results in elevated lactate levels within the tumour microenvironment (TME). This environment, characterized by hypoxia, nutrient limitation, and metabolic stress, has been shown to promote TNT formation. However, the specific role of lactate in regulating TNT biogenesis and the underlying mechanisms are not known. In this study, we quantitatively analyzed lactate-induced TNT formation under both glucose-deprived and glucose-enriched conditions. Pharmacological inhibition of lactate transporters or lactate dehydrogenase markedly reduced TNT formation, highlighting the dependence of this process on lactate metabolism. Using a three-dimensional glioblastoma spheroid model, we further demonstrate that lactate treatment enhances resistance to temozolomide (TMZ), suggesting that TNT-mediated intercellular connectivity confers protection against TMZ-induced cytotoxicity. Seahorse extracellular flux analysis revealed that lactate-driven TNT formation occurs independently of alterations in mitochondrial respiration. We propose that lactate facilitates TNT formation through epigenetic mechanisms involving histone and/or protein lactylation, which may reprogram the expression of genes governing TNT assembly and dynamics.

Dietary Trans Fatty Acids Exacerbate Chronic Stress–Induced Cognitive and Behavioral Impairments in Zebrafish

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[1] *Department of Biotechnology, Indian Institute of Technology Madras, Chennai, India*

Presenting Author: Hanna Fathima

Abstract:

Dietary trans fatty acids (TFAs) are known contributors to cardiovascular disease and metabolic disorders such as diabetes. However, their potential involvement in cognitive dysfunction remains poorly understood. We investigated how elaidic acid, the most abundant dietary TFA, influences depression-like, anxiety-like, and social behaviors in zebrafish under chronic unpredictable stress (CUS) and non-stress conditions. CUS was induced by randomly applying a series of mild stressors, in an unpredictable manner for ten days. Fish exposed to CUS exhibited cognitive impairments as well as anxiety- and depression-like behaviors, which were assessed through a battery of behavioral tests such as the novel tank test, light–dark preference test, and shoal cohesion assay. Zebrafish receiving a TFA-supplemented diet displayed more severe behavioral and cognitive deficits than those fed a control diet. In contrast, oleic acid, the cis isomer of elaidic acid, did not significantly affect CUS-induced behavioral abnormalities. Gene expression analysis revealed that TFA intake altered the expression of several brain genes associated with depression, inflammation, oxidative stress, and fatty acid metabolism. Histological examination further demonstrated greater neuronal damage in the CUS group fed TFAs. Taken together, these findings suggest that dietary TFAs aggravate cognitive and behavioral disturbances, especially under chronic stress.

ROS-responsive hydrogel targeting Intervertebral Disc Degeneration

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Presenting Author: Roshini Mohan

Abstract:

Chronic low back pain is a multi-factorial disease associated with intervertebral disc degeneration (IVDD) in 40% of cases, which involves the regulated death of disc cells responsible for maintaining ECM homeostasis. This is linked to extensive oxidative stress in the intervertebral disc(IVD), which leads to inflammation, senescence, mitochondrial dysregulation, and apoptosis, resulting in the accumulation of reactive oxygen species (ROS). A prospective approach for IVDD therapeutics encompasses the combined strategy of scavenging this ROS to prevent oxidative damage and providing an ECM mimetic environment to promote the regeneration of disc cells. In this regard, we have fabricated a hyaluronic acid-based photo-responsive hydrogel that can release the antioxidant resveratrol in the redox-imbalanced IVD microenvironment. The targeted release of resveratrol through the cleavage of ROS-labile linker supports the enhancement of resveratrol's bioavailability while retaining its bioactivity. The significant antioxidant capacity of the fabricated hydrogel was confirmed through an in vitro study conducted on primary nucleus pulposus (NP) cells. We demonstrate that IVDD can be managed by leveraging the mechanical support provided by the injectable photo-crosslinking hydrogel and the dynamic properties of resveratrol, ultimately regulating mitochondrial respiration and protecting against oxidative damage.

Keywords: Hyaluronic acid, photo-crosslinking, Resveratrol, ROS, antioxidant

Analysis of Eccrine Sweat in Women During Menstrual Cycle: An Exploratory Proteomic Study

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Presenting Author: Bhavana Ramachandran

Abstract:

There is a growing need for the development of non-invasive methods for disease detection and prognosis. This is an exploratory study aimed at individually characterizing the proteins in the eccrine sweat of women in different phases of their menstrual cycle. Although sweat is not a primary hormonal biofluid, hormonal influences in physiology across the menstrual cycle may indirectly reflect in sweat proteome. The aim was to validate the potential use of sweat analysis to differentiate between physiological states, laying the foundation for future work in disease biomarkers. Analysis of LC–MS protein intensity data from 30 sweat samples revealed 7–88 proteins detected per sample, with 12 proteins (5 %) consistently detected in more than 80 % of samples, (Dermicidins and Keratins) and most proteins were detected in less than 20 % of samples. Principal Component Analysis showed inter-individual differences account for majority of the variance, with a smaller contribution of menstrual cycle phase. Over representation analysis showed terms related to skin development, keratinocyte proliferation, epithelium development and epithelial differentiation in ovulatory phase compared to menstrual and mid-follicular phase. Our analysis therefore provides a foundational framework for the use of sweat as a non-invasive source to study physiological states.

Lab-on-chip device for rapid isolation and detection of bacteria for early sepsis detection

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Presenting Author: Diksha Mall

Abstract:

Rapid and sensitive antibiotic susceptibility testing (AST) is essential for timely management of high-morbidity sepsis, especially amid the growing threat of antimicrobial resistance (AMR). Conventional culture-based AST methods are labour-intensive and require 48–72 hours, delaying appropriate treatment. Early in sepsis, the bacterial load in blood is extremely low. Bacterial separation from whole blood is required for rapid and sensitive bacteria detection.This work aims to develop a rapid, affordable, and point-of-care microfluidic device for bacterial detection and AST from blood. A Lab-on-Chip platform integrating acoustofluidic separation and electrochemical impedance spectroscopy (EIS)-based detection will be designed to achieve this goal. The acoustofluidic module enables bacterial separation from red and white blood cells with 60% efficiency and 65% purity. The EIS-based AST (ε-μD) allows sensitive detection of bacterial growth inhibition at a limit of 105 CFU/mL (~500 CFU immobilized) without sample pretreatment. Analytical validation using urine as a sample matrix demonstrated reliable detection performance. The EIS based device can perform AST within approximately 3 hours, providing a rapid, sensitive, and point-of-care solution for early sepsis detection and effective antimicrobial therapy guidance. Increasing the separation efficiency and integration of separation module and detection module is the future goal of this study.

Dendritic integration mechanisms preferentially enhance the OFF signals in ON-OFF direction-selective retinal ganglion cells

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Presenting Author: Subhranwita Mallik

Abstract:

ON-OFF Direction-Selective Ganglion Cells contain bistratified dendrites that extend into both ON and OFF sublaminae of the inner plexiform layer of the retina. Our lab previously found that the OFF arbors are larger, denser and have higher density of bipolar synapses compared to the ON arbors. However, the potential functional consequences of these structural variations are unknown. Here, using NEURON simulations, we show that structural differences preferentially enhance the OFF signals. Our modeling studies revealed that the OFF arbor generates weaker signals in the ganglion cell, as its inputs are 30-345 microns further from the soma and are attenuated ~10% more than corresponding inputs in the ON arbor. We found two mechanisms by which the ganglion cells can compensate for the preferential attenuation of the OFF signals. First, the addition of sodium conductances in the dendrites, and second, increasing the number of excitatory synapses. Given the larger/denser OFF arbor, both these mechanisms preferentially enhanced the OFF signals in our simulations, leading to a higher sensitivity of OFF signals to visual contrast. These results provide the first evidence showing how ganglion cells differentially process signals from ON and OFF arbors in the same ganglion cell.

Rapid DNA Extraction method for Point of Care Diagnostics

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Presenting Author: Neha Rani Das

Abstract:

Cervical cancer, primarily caused by persistent infection with high-risk human papillomavirus (HR-HPV) genotypes such asHPV16 and HPV18, remains a significant global health burden, especially in low-resource settings. Early detection of HPV DNA is essential for effective disease management; however, the implementation of point-of-care (POC) diagnostics is often limited by the lack of rapid, affordable, and user-friendly DNA extraction methods compatible with downstream molecular assays. In this study, we developed a simple, rapid, and equipment-free DNA extraction protocol using cellulose-based filter papers. The compatibility of the extracted DNA was evaluated across three amplification platforms such as conventional PCR, quantitative PCR (qPCR), and loop-mediated isothermal amplification (LAMP). Strong and consistent amplification signals were obtained in all three assays, confirming the broad applicability of our method for both thermocycling and isothermal amplification techniques. Furthermore, we analyzed the physical and chemical properties of the filter papers to determine DNA yield variability.

Computer aided drug designing strategies for adjuvant therapy to eliminate *Mycobacterium tuberculosis* and in vitro validation

Subhashree Subhasmita Nayak*[1], Hassan Abdul Basheer K.V*[1], Tejan Lodhiya[1], Akshada Jagdale[1], Kiran Kumar[2], Hussain Bhukya[2], Raju Mukherjee[$2]

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Presenting Author: Dr. Subhashree Subhasmita Nayak

Abstract:

Tuberculosis (TB), caused by *Mycobacterium tuberculosis* (Mtb), affects millions of lives every year and continues to pose a major global health threat due to prolonged treatment regimens and emergence of multidrug-resistant strains. Therefore, it is essential to identify novel therapeutic-strategies that can enhance efficacy of existing drugs to improve treatment efficacy. In this study, we explore metabolic intoxication as an adjuvant therapeutic strategy to target the GlgE, a key enzyme in the α-glucan biosynthesis pathway. GlgE is responsible for converting maltose-1-phosphate (M1P) into linear α-glucans, and the inhibition of GlgE causes accumulation of toxic metabolite (M1P), leading to bacterial death. Herein, we employed both structure-based and pharmacophore-model-based drug design approaches to identify potential small-molecule inhibitors targeting GlgE. Virtual screening, molecular docking was conducted to shortlist lead molecules with favourable binding affinity and drug-like properties. Further, the stability of the shortlisted docked complexes was validated by molecular-dynamics simulations. For in vitro validation, the GlgE gene was successfully cloned, expressed, and purified to homogeneity. Ongoing work includes establishing enzymatic-assays, determining kinetic-parameters, and performing inhibition studies to assess the potency of the lead molecules. The finding of this research highlights GlgE-mediated metabolic intoxication as promising adjuvant therapy.

Clinicopathological and Functional Characterization of RASSF9 in Gastric Cancer

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Presenting Author: Amrutha Arjunan

Abstract:

Gastric cancer remains a significant health challenge in India, characterized by late diagnosis, limited therapeutic options, and poor clinical outcomes. Ras Association Domain Family Member 9 (RASSF9), a member of the N-terminal RASSF family, has been implicated in cell signaling and tumor-suppressive pathways, but its role in gastric carcinogenesis remains poorly defined. In this study, we analyzed RASSF9 expression across the pathological stages of gastric carcinogenesis and investigated its functional significance in gastric cancer cell lines. Immunohistochemical and transcript analyses revealed a marked reduction of RASSF9 expression in tumor tissues compared to adjacent gastric mucosa. Functional assays demonstrated that RASSF9 overexpression significantly inhibited cellular proliferation, migration, and transformation in vitro, whereas it's silencing enhanced proliferative potential. These findings establish RASSF9 as a potential tumor suppressor with anti-proliferative and anti-migratory functions, underscoring its relevance in the molecular pathogenesis and progression of gastric cancer.

Zidovudine Delivery and Mitigation of Hepatotoxicity through Glycan-Engineered Stem Cell–Derived Exosomes: An In Vitro Study

Pon Yazhine Tamilselvan[1], Swantish Singh[1], Mamello Sekhoacha.[2], Sandhya Tantuan.[3], Anand Krishnan[1]

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Presenting Author: Pon Yazhine Tamilselvan

Abstract:

Zidovudine (AZT), which is essential in PMTCT of HIV, is hepatotoxic; this study designs a glycan-engineered exosome-AZT bioconjugate as a novel biomolecule to mitigate AZT-induced hepatic injury. Adipose-derived stem cells (ADSCs) were metabolically labelled with 50 µM Ac4GalNAl to introduce alkyne functionalities on surface glycans and underwent CuAAC-click chemistry conjugation with 100 µM AZT. TEM characterization revealed surface morphology of exosomes and ζ-potential change from −2.47 mV to −7.79 mV, reflecting effective surface modification and enhanced colloidal stability. Effective conjugation was ascertained, and concentration of AZT in the bioconjugate was profiled by HPLC. Bioconjugate treatment (2.5 µg/mL) effectively minimized oxidative stress in HepG2 cells as compared to treatment with AZT-alone. Intracellular (0.1807 → 0.1409 µM; 22.0%) and extracellular (0.6292 → 0.05735 µM; 90.9%) levels of MDA decreased greatly, intracellular (186.93 → 173.22 µM) and extracellular (173.49 → 157.27 µM) levels of GSH enhanced, and SOD activity (2.54 → 5.88 U/mL; 2.31-fold) increased in biomolecule-treated cells than in AZT, which illustrates enhanced antioxidant defence. These findings indicate that glycan-engineered ADSC exosomes effectively reverse AZT-induced oxidative stress by increasing antioxidant defence and lipid peroxidation inhibition, providing a potential nanotherapeutic strategy to reduce antiretroviral drug toxicity while maintaining pharmacologic efficacy.

#PANIIT20251693- Poster Session, Day 1

Deciphering the role of tyrosine phosphorylation sites within the catalytic domain of PTP-PEST on its activity and endothelial cell functions.

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Presenting Author: Akhilesh Kumar Mishra

Abstract:

Endothelium, a specialized single layer of squamous epithelial cells lining the innermost surface of blood vessels, is crucial for maintaining overall vascular homeostasis. Endothelial homeostasis is tightly controlled by a complex network of signalling pathways involving kinases and phosphatases, which modulate protein phosphorylation states to maintain cellular balance. Among protein tyrosine phosphatases, PTP-PEST (also known as PTPN12, Protein Tyrosine Phosphatase Non-receptor Type 12) is a cytosolic protein that plays critical roles in embryonic development and embryonic vascular formation. Previous studies have shown that both global knockout and endothelial-specific deletion of PTP-PEST in mouse models result in embryonic lethality due to severe developmental defects. Recent studies reveal that the PTP-PEST catalytic domain contains several tyrosine phosphorylation sites that undergo phosphorylation in endothelial cells, as confirmed by mass spectrometry analysis. These identified tyrosine phosphorylation sites are located within important loops of the catalytic domain. However, the significance of these sites in regulating PTP-PEST activity and the PTP-PEST mediated mechanism remains unknown. Therefore, our study aims to elucidate the functional relevance of these tyrosine phosphorylation sites within the catalytic domain of PTP-PEST in endothelial cells to provide a more comprehensive understanding of its regulatory role and its impact on endothelial cell function.

Possibility of Using Lactate Receptor (GPR81) Agonist for Treating Drug Resistant Epilepsy

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Presenting Author: Gautam Mohapatra

Abstract:

Epilepsy, one of the most common neurological disorders, arises from an imbalance between neuronal excitation and inhibition that drives hyperexcitability. About 40% of patients show poor response to standard anti epileptic drugs, creating a major therapeutic gap. In this study, we tested the potential of targeting the lactate receptor GPR81 using its agonist, 3 chloro 5 hydroxybenzoic acid (CHBA), in cultured neurons and a zebrafish model of drug resistant epilepsy. CHBA markedly reduced neuronal hyperexcitability and calcium oscillations in rodent hippocampal and cortical neurons under low Mg^{2+} conditions. In glial cultures, CHBA suppressed glutamate spillover triggered by the same conditions. In Scn1lab mutant zebrafish that model refractory epilepsy, CHBA significantly decreased seizure scores. Mechanistically, activation of the Gi coupled GPCR GPR81 triggered G protein activated inward rectifying potassium (GIRK) channels, leading to neuronal hyperpolarization and inhibition. To demonstrate this coupling, we co expressed GPR81 with GIRK1/2 subunits in Xenopus oocytes, where CHBA induced robust GIRK currents. These results identify GPR81 agonists as promising therapeutic candidates for refractory epilepsy and related neuronal hyperexcitability disorders.

POSTER SESSION
DAY 2

Syk in Motion - Loop-Guided Control of the Conformational Landscape and Its Modulation by Mutations

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Presenting Author: Sunanda Samanta

Abstract:

Spleen tyrosine kinase (Syk) functions as a pivotal enzyme in immune cell signaling and has been associated with diverse pathways in cancer and neurodegenerative disorders, establishing it as a protein of significant therapeutic relevance. Although computational studies on Syk have focused on drug discovery, its molecular regulatory dynamics remain largely unexplored. Here, we used microsecond-scale all-atom MD and GaMD simulations of full-length Syk and its kinase domain to examine phosphorylated states and mutation-induced dynamic changes. Our results suggest that electrostatic interactions around the activation segment modulate its dynamics, while the N and C-terminal anchors are stabilized by nearby helical regions. The P-loop exhibited conformational fluctuations toward and away from the conserved "HRD" motif, with its motions strongly correlated with the β3-αC loop, together regulating the size of the adenine-binding pocket in the C-spine region. Furthermore, in the mutated systems P342T and A353T, the central portion of the SH2-kinase linker displayed a pronounced shift in motion toward the C-SH2 domain, accompanied by alterations in inter-residue connectivity as well as changes in macrostate formation and transition dynamics. Overall, these potential molecular mechanisms illuminate the functional dynamics of Syk regulation and their relevance for therapeutic intervention.

Comprehensive characterization and functional insights into septin genes of *Labeo rohita*: molecular basis of immune modulation and development

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Presenting Author: Lopamudra Parida

Abstract:

Septins are conserved GTP-binding cytoskeletal proteins that function as molecular scaffolds and diffusion barriers, and involved in cytokinesis, vesicle trafficking, and defence in mammals, but largely unexplored in fish. A comprehensive set of thirteen septin genes was in silico identified and in vivo characterized in *Labeo rohita* (rohu), with a major emphasis on septins 2, 6, and 8a. All septin genes exhibited differential expression across tissues, with higher levels in mucosal tissues and brain, suggesting roles in mucosal immunity and neural regulation. Developmental expression analyses indicated elevated transcription during early ontogeny, highlighting their involvement in embryogenesis and immune maturation. Upon exposure to three diverse pathogen models, septins 2, 6, and 8a showed significant upregulation, indicating their participation in pathogen-responsive signaling pathways. Functional characterization of produced recombinant septin 2, 6 and 8a proteins revealed modulation of immune-related genes, including cytokines, antimicrobial effectors, antioxidants, apoptosis regulators, and TLRs, and protection against microbial infections. Developed ELISAs for three septins indicated their elevated levels in liver, spleen, and brain following bacterial infection. A septin 8a knock-out line was generated for further functional validation. Collectively, these findings suggest septins' role in development and innate immunity, and highlight their potential applications in disease management in aquaculture.

Keywords: *Labeo rohita*, septin genes, immune modulation, recombinant protein, infection response, aquaculture immunology

From Cells to Meat: Integrating Cells and Metabolic Modelling for Cultivated Mutton Manufacturing

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Presenting Author: Aparna M

Abstract:

Cultivated meat (CM) is a sustainable alternative to traditional meat production since it addresses key challenges including animal welfare, greenhouse gas emissions, excessive land and water consumption, rising demand for protein-rich foods, and spread of zoonotic diseases. This work aims to establish stable goat muscle satellite cell line and goat muscle fibroblast cell line of Indian indigenous goat species. Primary muscle satellite cells and fibroblasts were isolated from goat muscle biopsies through enzymatic dissociation and purified by pre-plating method. Since we aim at animal component free CM, a gradual serum reduction strategy is employed to adapt the cell lines to serum-free medium. Considering the complex nature of cell culture media, we propose a model based media design strategy over conventional DoE approach. A genome-scale metabolic model(GEM) for Capra hircus(goat) was developed by integrating metabolic information from well-established models of chicken, Chinese Hamster Ovary(CHO), and human. Homologous genes between goat and the three reference species were identified using bidirectional BLASTp, and the corresponding gene-protein reaction (GPR) associations were systematically curated using metabolic subsystems. Overall, this work utilises the model to identify essential nutrients and metabolic pathways specific to goat cells, to formulate an optimized, robust cell-culture medium for CM production.

Selective activator of calcium-activated potassium channels, NS-309 acts as effective seizure reducing agent by restricting the hyperexcitability phenomenon in acquired epilepsy

Rituparna Sahu[1], Giriraj Sahu[1]

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Presenting Author: Rituparna Sahu

Abstract:

Temporal lobe epilepsy (TLE) is characterized by uncontrolled and synchronized firing of the cortical and hippocampal pyramidal neurons, mainly due to an imbalance in the excitatory-inhibitory (E/I) homeostatic processes. Post-burst slow afterhyperpolarization (sAHP), an intrinsic inhibitory neuronal excitability regulator is dramatically suppressed in cortical and hippocampal pyramidal neurons in epileptic human patients and status epilepticus (SE) induced experimental TLE animal models. Recently Cav1.3-RyR-KCa3.1 protein complex was identified for sAHP generation. However, the mechanisms underlying sAHP suppression in TLE remained unexplored, hindering therapeutic intervention. To address this, super-resolution dSTORM imaging in TIRF mode was used to characterise Cav1.3–RyR–KCa3.1 complex organization in control and 0-Mg^{2+}–induced in vitro epileptic hippocampal neurons. A decrease in KCa3.1 clusters in the epileptic neurons was detected from dSTORM. This was supported by decreased KCa3.1 mRNA and protein levels by RTPCR and western blot-mediated protein quantification in epileptic hippocampi of pilocarpine induced chronic epileptic rats, respectively. To evaluate the therapeutic potential of KCa3.1 activator, NS309, as a novel anti-epileptic candidate, its ability to restore sAHP was studied using whole-cell current-clamp recordings in 0-Mg^{2+} epileptic cultured neurons and 4-AP/0-Mg^{2+} acute epileptic rat brain slices,along with seizure monitoring during status epilepticus

Role of GTPase in adrenergic modulation of sAHP-generating IKCa and L-type Cav1.3 channels in hippocampal pyramidal neurons

Kashmiri M Lande[1], Mandip P Gadpayle[1], Aynal Hoque[1], Raja Aravind Y[1], Giriraj Sahu[2]

[1]IISc Bangalore, [2]Assistant Professor, IISc Bangalore

Presenting Author: Kashmiri M Lande

Abstract:

Adrenergic neurotransmission plays a crucial role in cognitive processes by fine-tuning the excitability pattern of principal pyramidal neurons of the brain. Its dysregulation contributes to epilepsy, traumatic brain injury, and neurodegenerative diseases. Adrenergic agonists enhance neuronal excitability by downregulation of slow afterhyperpolarization (sAHP) via Protein Kinase A (PKA) activation. However, the molecular target of PKA activation and sAHP downregulation remained elusive. Since sAHP current is generated by synergistic activation of L-type Cav1.3 and IKCa channels, it prompted us to study the potential intermediate proteins that modulate adrenergic neurotransmission in hippocampal pyramidal neurons. We found a significant increase in expression levels of the novel GTPase in the hippocampus upon treatment with adrenergic agonists. Super-resolution imaging, FRET, and pull-down studies confirmed the interaction between IKCa, Cav1.3, and identified GTPase in HEK293T cells. Additionally, GTPase induced a decrease in IKCa and Cav1.3 expression and current amplitude. We also observed that PKA stimulation induced increased association of GTPase with IKCa and decreased association with Cav1.3, which validates differential modulation. In conclusion, the results suggest that the identified GTPase differentially regulates the association pattern and functional activation of Cav1.3 and IKCa channels, which depend on PKA activation, the byproduct of adrenergic stimulation.

IN SITU MINERALIZED BONE MICROENVIRONMENT FOR DRUG SCREENING APPLICATIONS

Kadambari Sathyanarayanan[1], Shantanu Pradhan[1]

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Presenting Author: Ramya D

Abstract:

Bone metastases affect approximately 70% of advanced breast cancer patients, severely impacting survival and quality of life. Traditional animal and in vitro models fail to fully replicate human bone physiology or the complexity of the extracellular matrix (ECM), limiting insights into tumor–bone interactions. To address this, we developed a metastasis-on-a-chip platform incorporating in situ mineralized GelMA (MG300) within a bone-mimetic chamber to co-culture preosteoblasts (MC3T3-E1) and breast cancer cells (MDA-MB-231). The in situ mineralization process enabled uniform hydroxyapatite deposition, with controlled mineral content (~24%) and bone-like nanostructure confirmed by XRD analysis. Preosteoblasts cultured in this matrix exhibited high viability over 14 days, extended dendritic morphology, and increased osteopontin expression, indicating osteogenic differentiation and functional bone-like behavior. This platform recapitulates the physical, mechanical, and biochemical properties of native bone, providing a versatile system to study colonization, osteomimicry, MET and dormancy, while offering significant potential for targeted therapy development and personalized medicine applications

Twisted ECM and polymer yarn for cardiac tissue regeneration

Arthi N[1], Praveen Kumar Vemula[2]

[1,2]*Institute of Stem Cell Science and Regenerative Medicine, Bengaluru*

Presenting Author: Arthi N

Abstract:

Myocardial Infarction (MI) is an alarming global health issue with high mortality, and it is estimated that by 2030, 23.3 million deaths occur worldwide. MI, also called a heart attack, causes obstruction of coronary arteries, deterioration of extracellular matrices of the myocardium, and thinning of the left ventricular wall, eventually resulting in heart failure. Apart from medications and surgery, heart transplantation is the final solution with major drawbacks such as donor scarcity and immune rejection complications. To overcome this, the concept of scaffold-based tissue engineering committed to the idea of using a combination of cells/biomolecules with a scaffold that promotes tissue repair and regeneration has gained attention in in field of regenerative medicine. We propose to incorporate ECM sheets and forcespun threads together as a scaffold to have the combined advantage of nanofiber morphology and the ECM such as high surface to volume ratio, increased mechanical strength, cell adhesion to promote tissue regeneration. Objective 1: Synthesis of Forcespun polymer threads Objective 2: Synthesis of Decellularised cardiac cell derived ECM into strip form Objective 3: Invitro studies of combined ECM sheets and polymer threads. Key findings: The ECM strips combined with polymer threads showed differentiation of stem cells to cardiac lineage

Reformulating drugs for enhanced solubility & lower dose requirements

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Presenting Author: Evan Debnath

Abstract:

Active pharmaceutical ingredients (APIs) form the foundation of modern therapeutics, yet nearly 60–70% of marketed drugs exhibit poor aqueous solubility, resulting in low bioavailability and inconsistent therapeutic outcomes. Conventional solubilization approaches rely heavily on excipients, which often introduce toxicity, instability, or formulation challenges. To overcome these limitations, we have developed a reformulated API (rAPI) platform based on biocompatible ionic liquids (ILs)- organic salts with tunable physicochemical properties and exceptional solubilizing ability. In this work, we focus on Propofol, a widely used intravenous anesthetic whose marketed lipid emulsion formulation is associated with injection-site pain and stability issues. We synthesize Propofol–IL conjugates using pharmaceutically acceptable organic counterions and characterize them via NMR, HRMS, TGA, and DSC. We demonstrate a >500-fold enhancement in aqueous solubility, improved thermal stability, and faster dissolution compared to the commercial formulation. Preliminary pharmacokinetic and pharmacodynamic studies indicate enhanced bioavailability, faster onset, and prolonged anesthetic duration. These results establish IL-based rAPI technology as a safe, scalable, and versatile reformulation strategy, paving the way for next-generation, excipient-free anesthetic formulations with superior performance and patient compliance.

To study if PAK1 (p21 activated kinase) phosphorylation of AHNAK protein modulates blood-brain barrier integrity

Srutimanjari Parida[1], Suresh Kumar Rayala[1]

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Presenting Author: Srutimanjari Parida

Abstract:

The Blood-Brain Barrier (BBB) maintains central nervous system (CNS) homeostasis, and its disruption is a hallmark of glioblastoma (GBM) progression. PAK1, a serine/threonine kinase, has been implicated in BBB dysregulation under hypoxic tumor microenvironments, potentially through phosphorylation of structural proteins. AHNAK, a scaffolding protein essential for maintaining BBB integrity, may be a novel substrate of PAK1. This study investigates the mechanistic basis of the PAK1–AHNAK interaction and its role in BBB disruption during glioma progression. Three domains of AHNAK (N-AHNAK, AHNAK-4CRU, and C-AHNAK) were expressed in HEK cells to identify the specific region interacting with PAK1. Co-immunoprecipitation and in vitro kinase assays revealed that PAK1 interacts with and phosphorylates the N-terminal domain of AHNAK. These findings suggest a regulatory role of PAK1-mediated post-translational modification of AHNAK in modulating tight junction proteins and endothelial permeability. Future work will evaluate the functional impact of this phosphorylation on BBB integrity in vitro and in vivo, and test PAK1 modulators to enhance chemotherapeutic and nanoparticle delivery across the BBB. Overall, this study identifies the PAK1–AHNAK axis as a potential therapeutic target for improving drug delivery and limiting BBB disruption in glioblastoma.

Aluminium-Induced Reproductive Toxicity and Estrogen Receptor Gene Expression Alterations: A Possible Forerunner to Neurodegenerative Pathology in Female Wistar Rats

Tripti Sharma[1], Amit Kumar Ghosh[4], Hare Krishna[2], Abhinav Dixit[1], Shilpi G Dixit[2], Kamla K Shukla[3], Mithu Banerjee[3], Amal Kanti Bera[4], Prasunpriya Nayak[1]

[1]Department of Physiology, [2]Department of Anatomy, [3]Department of Biochemistry, All India Institute of Medical Sciences, Jodhpur, Rajasthan, India; [4]Department of Biotechnology, Indian Institute of Technology, Madras, Chennai, India

Presenting Author: Tripti Sharma

Abstract:

Aluminium, a ubiquitous metalloestrogen, has no known physiological effect. Nevertheless, it enters through multiple routes and accumulates in various organs, and demonstrates biotoxicity. Given aluminium's neurotoxic properties and the neuroprotective role of estrogen, it is plausible that aluminium-induced disturbances in the reproductive cycle may also be linked to its neurotoxicity. This study aimed to elucidate the mechanistic link between aluminium-induced reproductive and neurotoxicity. Twelve adult female rats with regular estrous cycles were randomly assigned to the experimental group (4 weeks of orogastric aluminium exposure at 100 mg/kg bw) and the control group (only vehicle exposure). Estrous cyclicity was monitored throughout the exposure period. Post-exposure neurobehavioral evaluations were conducted before sacrifice, followed by the collection of ovarian and brain tissues. Neurobehavioral assessments revealed significant alterations in anxiety-related behaviors compared to controls, with additional modifications in motor coordination, locomotor activity, and exploratory behaviors. Significant estrous cycle perturbations in aluminium-treated groups, accompanied by differential estrogen receptor gene expression in the tested organs, suggested aluminium-induced reprotoxicity. Relatively lesser alterations in neurobehavioral parameters with significant reprotoxicity suggest aluminium – estrogen interplay may serve as a forerunner to neurodegeneration.

Investigation of the Function of Epigenetic Reader Proteins in Malarial Parasites

Velvili Saravan[1], Arumugam Rajavelu[1]

[1]*Department of Biotechnology, Indian Institute of Technology Madras*

Presenting Author: Velvili S

Abstract:

The lifecycle of *Plasmodium falciparum* is complex due to its multiple stages, each characterized by distinct gene expression profiles. This stage-specific gene expression enables the parasite to effectively evade the host's immune responses, complicating malaria treatment. Epigenetic modifications, particularly those on chromatin and histone tails, regulate this differential gene expression. These modifications are recognized by epigenetic reader proteins, whose interactions play a crucial role in managing the parasite's antigenic variation and pathogenicity. While various reader proteins exist in the parasite, such as bromodomain that reads acetylation mark and chromodomain which reads H3K9me3 mark, only a limited number have been characterized in *P. falciparum*. Characterizing these reader proteins is essential for a deeper understanding of malaria pathogenesis. In this study, we have identified an epigenetic reader protein that binds to a repressive histone mark previously unreported in *P. falciparum*. This interaction has been validated through biochemical assays and at the endogenous level as well. Our ongoing research aims to elucidate the significance of this novel interaction in the parasite's lifecycle.

Elucidating the role of unique Epigenetic modifications in *Plasmodium falciparum*

Jissmole Lukose Pallithanam[1], Arumugam Rajavelu [1]

[1]*Department of Biotechnology, Indian Institute of Technology Madras*

Presenting Author: Jissmole Lukose

Abstract:

P. falciparum uses differential gene expression mechanisms as strategies to adapt to two different host species and environments. Among them, parasites rely heavily on epigenetic-mediated mechanisms for controlling chromatin organization, gene regulation, morphological differentiation, and antigenic variation. The epigenome of *P. falciparum* is distinct from mammals as it has few transcription factors, thus tight regulation of gene expression largely depends on the nature of the chromatin structure in the parasite. Most of the research has been focused on post-translational modifications in the N-terminal tail of histones, the PTMs present within the globular domain are less well understood. How the PTMs in these domains affect chromatin-templated processes is not clear. Emerging reports have confirmed that *P. falciparum* chromatin harbors many modifications at unconventional residues. Thus, a detailed investigation of these PTMs would help in understanding the chromatin dynamics mediated gene expression changes in a human malaria parasite. The aim of our study is to identify unique epigenetic modification and characterise its functional relevance in *P. falciparum*. We have identified a demethylation mark at arginine 42 on histone H3, the enzyme introducing the modification and also the dynamic distribution of this mark during the RBC stages of *P. falciparum*.

Investigating the deregulated miRNAs and their expression in differentiated thyroid cancer of South Indian ethnicity

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Presenting Author: Dhanaraj R

Abstract:

Differentiated thyroid cancer (DTC) is the most prevalent form of thyroid malignancy, with microRNAs (miRNAs) playing a pivotal role in its pathogenesis through post-transcriptional regulation of target genes. This study aimed to identify and validate key dysregulated miRNAs associated with DTC progression, focusing on miR-4728-3p, miR-9-5p, miR-146b-5p, and miR-218-5p. Publicly available transcriptomic datasets were analyzed to profile miRNA expression patterns in DTC, revealing significant differential expression. Validation in serum samples using quantitative real-time PCR (RT-qPCR) confirmed that miR-4728-3p and miR-146b-5p were significantly altered in DTC patients compared with controls. Target gene expression analysis (BRAF, TG, NRAS, and HRAS) and in vitro functional assays in thyroid cancer cell lines are planned to elucidate the role of these miRNAs in regulating tumor cell proliferation, migration, and invasion. Collectively, these findings highlight the potential of specific miRNAs as biomarkers for DTC and may provide a basis for developing novel diagnostic and therapeutic strategies.

Identification of Novel ATP-site GSK-3β Inhibitors by Employing Molecular Modeling Approaches and Free-Energy Calculations

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Presenting Author: Nachiket Joshi

Abstract:

Alzheimer's disease (AD) is a multimodal disease with various intricate, interconnecting pathways. Aggregated tau protein is a major concern and represents a crucial mechanism that can be targeted to reveal disease-modifying therapy. The hyperphosphorylated tau protein, owing to the increased activity of Glycogen synthase kinase 3β (GSK-3β), leads to the formation of neurofibrillary tangles, thus hampering normal cognitive function. Herein, we explored the potential of novel derivatives targeting GSK-3β within the ATP binding site in comparison to the co-crystallised ligand and standard inhibitor Staurosporine. Initially, 2,5-disubstituted thiazole derivatives bearing a bis-amide moiety were designed and docked against GSK-3β (PDB ID: 5k5n). A few hits showed higher docking scores and similar amino acid interaction profiles compared to Staurosporine and the Co-crystallised ligand. The top 5 hits were subjected to MD simulations for a period of 500ns. These simulations revealed the potential of the compounds to inhibit GSK-3β. Furthermore, to determine the binding affinity, MM-PBSA calculations were performed to determine the free energy of the protein-ligand complexes. RMSD, RMSF, Radius of gyration, and Solvent-accessible surface area parameters were accessed to determine the binding potential of the hit molecules. Furthermore, PCA analysis was carried out to reveal the conformational changes involved in the binding.

N-terminal acetylation affects mitochondrial function and mitochondrial biogenesis in alcohol-induced neurotoxicity

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[1]*School of Bio Sciences and Technology, Vellore Institute of Technology, Vellore 632014, Tamil Nadu, India*

Presenting Author: Raghul Kannan S

Abstract:

N-terminal acetyltransferases 10 (Naa10) catalyze protein N-terminal (Nt) acetylation and regulate mitochondrial structure, function, and mitophagy and have implications on cancer. However, its role in the alcoholic brain is unknown. We hypothesize that alcohol could alter the Naa10 expression and could contribute to altered mitochondrial function and impaired mitochondrial biogenesis. Further, we expect methylsulfonylmethane (MSM) could restore the alcohol-induced effects in C57BL6/J mice brains. Alcohol reduced Naa10 expression and mitochondrial copy number/content and decreased mitochondrial biogenesis, along with impaired neurobehavior in alcohol-induced mice. Intriguingly, Naa10 expression is negatively correlated with mitochondrial copy number and mitochondrial biogenesis. Further, alcohol exposure altered Nt-acetylation of Pgc-1a, impacting the mitochondrial biogenesis pathway. MSM administration restored Naa10 expression, mitochondrial function and mitochondrial biogenesis. Together these results suggest that Naa10-mediated Nt-acetylation of Pgc-1a impaired the protein-protein interaction, leading to decreased mitochondrial biogenesis.

Molecular Screening of Genes Associated with Differentiated Thyroid Cancer in the South Indian Population: A Case Control Study

Anandhi Kalaiyazhagan[1], Bhuvarahamurthy Venugopal[1]

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Presenting Author: Anandhi Kalaiyazhagan

Abstract:

The global incidence of thyroid cancer has increased markedly in recent years, establishing it as the most prevalent endocrine malignancy. Papillary (PTC) and follicular thyroid carcinomas (FTC) together account for over 90% of well-differentiated thyroid carcinoma (DTC) cases. Although exposure to ionizing radiation is a well-characterized etiological factor, genetic predisposition plays a crucial role in DTC pathogenesis. The present study aimed to elucidate the association between specific single nucleotide polymorphisms (SNPs) in BRAF (rs7801086), NRAS (rs14804), HRAS (rs28933406), and TG (rs2076740) genes and the susceptibility to DTC in a South Indian cohort. A case–control study was performed involving 200 histopathologically confirmed DTC patients and age- and sex-matched healthy controls. Genotyping was conducted using polymerase chain reaction–restriction fragment length polymorphism (PCR–RFLP) followed by Sanger sequencing. Significant associations were observed for NRAS-rs14804, HRAS-rs28933406, and TG-rs2076740 with increased DTC risk. Moreover, a novel mutation was identified in TG gene, suggesting a potential role in thyroid tumorigenesis. These results indicate that genetic variations in RAS and TG pathways may predispose individuals to DTC, necessitating further studies to validate and elucidate their functional roles in thyroid carcinogenesis.

Studies on Crosslinking Efficiency of Bovine Pericardium Using *Terminalia Chebula* Extract for tissue engineering applications

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Presenting Author: Agnes Mary S

Abstract:

Decellularized thin membranes such as surface covering of internal organs and linings of other body cavities are considered as biological substitutes to improve and renew the functions of organs or tissue in regenerative tissue engineering therapies. These cell free collagen substitutes of extra cellular matrix (ECM) are derived commonly from pericardium, peritoneum, amniotic membrane, etc., have been used for tissue constructs for bone, cartilage, skin, cornea and cardiovascular applications. Crosslinking of collagen substrates carried out using chemical and physical techniques. Bovine pericardium was pericardium crosslinked with *Terminalia chebula* (*T. chebula*) extract, a natural agent known for its antioxidant, anti-inflammatory, and antibacterial properties. Phytochemical analysis confirmed the presence of flavonoids, tannins, saponins, alkaloids, and phenolic compounds, with polyphenols acting as natural crosslinkers to enhance both mechanical stability and bioactivity. Collagen membranes were treated with 1%, 5%, and 10% concentrations of *T. chebula* extract and compared with glutaraldehyde-crosslinked controls. Results demonstrated that *T. chebula*-crosslinked membranes exhibited improved structural integrity, enzymatic resistance and antimicrobial efficacy. By effectively mimicking the ECM, these membranes promoted cell adhesion, proliferation, and tissue regeneration. The findings suggest that *T. chebula*-based crosslinking offers a biocompatible, sustainable alternative for applications in cardiac regenerative medicine and advanced wound care.

RAS Effector, RASSF10, Modulates Cancer Cell Metabolism

Rajesh Das[1], Prof. Prof. Mahalingam S [2]

[1,2] *Department of Biotechnology, Indian Institute of Technology Madras*

Presenting Author: Rajesh Das

Abstract:

Ras mutations occur in over 30% of cancers and are particularly prevalent in pancreatic, lung, and colon cancers. Ras proteins function as molecular switches regulating cell proliferation and survival; however, activating mutations in the gene result in permanent signalling, which drives oncogenesis. The Ras-association domain family (RASSF) comprises non-enzymatic Ras effectors that mediate cell cycle arrest and apoptosis, yet are often silenced in various types of cancer. While the role of RASSF proteins in repressing Ras-driven tumours is recognised, their mechanisms remain unclear. Our study investigates the functional role of the newest RASSF member, RASSF10—a putative tumour suppressor, commonly silenced by promoter hypermethylation in cancer, with an unknown mode of action. Using DNA microarray and proteomic analyses, we identified GNL3L (a nucleolar GTPase) and IMPDH1 (involved in guanine nucleotide biosynthesis) as targets of RASSF10. Although RASSF10 is not a transcription factor, it downregulates GNL3L and IMPDH1, both c-Myc targets. ChIP-PCR confirmed c-Myc binding at the GNL3L promoter. Interestingly, RASSF10 modulates c-Myc activity by forming a complex with c-Myc and competing with the heterodimerising partner, Max. Collectively, these findings suggest a novel mechanism by which RASSF10 suppresses proliferation by modulating c-Myc-mediated transcription.

RASSF7: A novel transcription factor orchestrating oncogenic signalling and tumour dynamics

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Presenting Author: Jithin P. V.

Abstract:

The RAS signalling pathway is a critical determinant of cell fate decisions, including proliferation, differentiation, and apoptosis, and is frequently dysregulated in human cancers through activating mutations in RAS alleles. Despite extensive efforts, direct RAS targeting has shown limited therapeutic success, steering attention toward non-enzymatic RAS effectors such as the RAS-association domain family (RASSF) proteins. RASSF7, an N-terminal member of this family, exhibits context-dependent roles in both tumour initiation and suppression. RASSF7 possesses a basic helix-loop-helix-leucine-zipper and a transactivation domain, suggesting its potential function as a transcriptional regulator. Current study characterizes the transcriptional regulatory function of RASSF7 and its influence on cellular signalling pathways. ChIP–Seq analysis identified two potential consensus RASSF7 binding motifs and elucidated its downstream target genes, while further transcriptomic profiling by RNA–Seq will reveal global transcriptomic alterations associated with RASSF7. Functional characterization using domain-specific mutants delineated the importance of DNA-binding and transactivation domains for its regulatory function. Finally, our data indicate that RASSF7 is a nucleo-cytoplasmic shuttling protein which forms homodimer via its leucine-zipper domain to modulate transcriptional regulation governing cellular processes. These insights reveal a previously unidentified role of RASSF7.

RAS Effectors Orchestrating MYC Activity: Molecular Insights Into Tumorigenesis

Sujoy Sow Mondal[1], Prof. Mahalingam S[2]

[1,2] *Department of Biotechnology, Indian Institute of Technology Madras*

Presenting Author: Sujoy Sow Mondal

Abstract:

RASSF (Ras Association Domain Family) proteins are non-enzymatic Ras effectors that play pivotal roles in diverse cellular processes, including cell cycle regulation, apoptosis, and tumor suppression. The RASSF family comprises ten members, classified into C-terminal (RASSF1–RASSF6) and N-terminal (RASSF7–RASSF10) subgroups based on the position of the Ras association (RA) domain. The oncogenic transcription factor c-MYC is a master regulator of cell growth and proliferation, frequently deregulated in human malignancies, yet remains largely undruggable. Our previous study demonstrated that RASSF7 interacts with c-MYC, thereby suppressing its oncogenic activity (Anbarasu et al., 2018). Interestingly, RASSF1, RASSF7and RASSF10 forms complex with oncoprotein c-MYC and regulates c-MYC induced cell division. Furthermore, RT-qPCR analyses and dual-luciferase reporter assays, provided evidence that c-Myc negatively regulates RASSF1, RASSF7 and RASSF10 transcription in breast cancer cells. Surprisingly, RASSF1, RASSF7 and RASSF10 deregulates c-Myc expression despite not being a transcription factor. Collectively, these findings uncover a previously unrecognized regulatory interplay between non-enzymatic Ras effectors and c-MYC signalling. This study provides new mechanistic insights into the modulation of oncogenic c-MYC function and highlights the potential of RASSF–MYC interactions as novel therapeutic targets for MYC-driven cancers.

Computational profiling of Chromosome 1 – A comprehensive search for cancer biomarkers

Bhatt Baby Bhavana[1], Devyani Charan[2], Trupti Patel[2]

[1]Department of Biotechnology, School of Biosciences and Technology, Vellore Institute of Technology, Vellore; [2]Department of Integrative Biology, School of Biosciences and Technology, Vellore Institute of Technology, Vellore; [2]Trupti Patel Prof., & Head, Dept. of Integrative Biology, School of Bio Sciences and Technology, VIT, Vellore*

Presenting Author: Bhatt Baby Bhavana

Abstract:

Cancer is caused by a complex combination of genetic changes in both the protein-coding and non-coding regions of DNA. Although gene mutations themselves have been the subject of much research, the impact of variations outside of these regions has received less attention. Using the most recent human genome reference (T2T-CHM13v2.0), this study used sophisticated computational tools to analyze frequently disregarded DNA segments, such as specific intronic mutations and variations in gene copy numbers across human chromosome 1. We studied the potential interactions between these genetic changes and currently available medications by combining gene activity measurements with pathway analysis and mutation mapping. Nine genes were identified by our network analysis as being significantly involved in cancer-related processes, some of which are essential regulators of tumor growth. It was discovered that structural DNA alterations that cross key regulatory regions have a significant impact on the expression of some of these nine genes. Our drug repurposing analysis revealed that Odevixibat and Tazarotene were promising options for new therapeutic applications. In particular, we modeled the binding of the Notch2 protein variant that has a specific intron mutation with six naturally occurring compounds derived from black turmeric (*Curcuma caesia*) to gain a better understanding of the regulation of the Notch pathway. The modeled Notch2 protein and the ligands, viz., Compound 3,88,91 from *Curcuma caesia* Roxb (Black turmeric) and UBS109, EF31, Bisacurone from *Curcuma longa*. The anti-cancer capabilities of these medicinal plants have been demonstrated to reduce the chance of acquiring resistance when used either alone or in combination with chemotherapy treatments. These ligands exhibited stable interactions and favorable binding orientations, as demonstrated by molecular docking analysis. The work as a whole demonstrates the potent influence of hidden DNA variations in determining the behavior of cancer and creates new opportunities for focused therapeutic approaches.

Targeted Nisosomes protect the heart from doxorubicn induced injury: Evidence from Oxidative stress pyroptosis and apoptosis pathways

Singaravel Vijayapoopathi[1], Dhanaraj Ramamurthy[1], Dhyaneshwar Mani[1], Bhuvarahamurthy Venugopal[1]

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Presenting Author: Dhyaneshwar M

Abstract:

Doxorubicin (DOX) remains a cornerstone in breast cancer chemotherapy; however, its clinical application is limited by dose-related cardiotoxicity. To overcome this challenge, epidermal growth factor receptor (EGFR)-targeted, centuximab-conjugated niosomes co-loaded with DOX and vitexin (NIODVC) were developed to enhance tumor selectivity and minimize off-target toxicity. In 4T1 breast tumor-bearing mice, NIODVC demonstrated superior therapeutic efficacy and significant cardioprotection compared with non-targeted formulations (NIODV). The optimized system exhibited improved stability, controlled drug release, and enhanced cellular uptake through EGFR-mediated endocytosis. Hematological and biochemical analyses revealed that NIODVC restored cardiac enzyme balance and antioxidant competence, while histopathological examination confirmed the preservation of myocardial integrity. RT-qPCR analysis further indicated the upregulation of antioxidant genes (Nrf2, Ho-1, Nqo1, Sod1, Sod2) and suppression of pro-oxidant and pyroptotic mediators (p67phox, Nlrp3, Caspase-1, Gsdmd, Il-1β, Il-18), alongside favorable modulation of apoptotic markers by increasing Bcl2 and decreasing Bax expression. Notably, intratumoral administration of NIODVC provided greater cardioprotection than intravenous delivery. Collectively, NIODVC represents a multifunctional nanocarrier with dual potential to enhance the anticancer efficacy of DOX and alleviate its cardiotoxic effects, offering a promising strategy for safer and more effective breast cancer therapy.

Bioinformatic and Biochemical Characterization of Putative Esterase/Lipase SsoEst5 from *Sulpholobus solftaricus*

Paras Gautam[1], N Manoj[1]

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Presenting Author: Paras Gautam

Abstract:

Lipases from archaea show thermo alkali-, acido -, psychro-, and/or halophilic nature which make them suitable candidate for industries like pulp and paper, textiles, cosmetics and biodiesel production. However, in archaea, esterases and lipases are less characterized, here we characterized "SsoEst5" from *Sulpholobus solftaricus* using bioinformatic and biochemical studies. Through bioinformatic studies we found sequence and structural homologs of SsoEst5 and showed it belongs to α/β hydrolase superfamily. MSA of structure homologs revealed unique Catalytic triad in SsoEst5. By observing catalytic triad conservation and structure alignment studies using classical α/β hydrolase 5FDF, we proposed the oxyanion hole residues, Thr58 and Phe152 in SsoEst5. Structure comparative analysis of SsoEst5 the presence of cap domain (made by α4a, α4b and α4c). Using biochemical studies we have shown optimum temperature (70 ° C) and optimum pH (7.5) of SsoEst5 along specific activity with p-nitrophenol acetate and p-nitrophenol palmitate and providing evidence that SsoEst5 is an esterase and lipase. We visualized the interactions of SsoEst5 active site residues with the p-nitrophenol esters through docking studies and observed the interaction of Catalytic Ser151 and oxyanion hole Thr58 with carbonyl oxygen of ligand ester group. As an esterase and lipase SsoEst5 has high industrial applications.

Molecular and Biophysical Insights into the Structure and Function of the Single-stranded DNA-binding Protein from *Pseudomonas aeruginosa*

Sachin Mishra[1], Narayanan Manoj[1]

[1] *Department of Biotechnology, Bhupat and Jyoti Mehta School of Biosciences, Indian Institute of Technology Madras, Chennai, Tamil Nadu, India*

Presenting Author: Sachin Mishra

Abstract:

DNA replication is the process of DNA synthesis that ensures that the DNA content in a species remains constant. During DNA replication, DnaB helicase unwinds the dsDNA, and the SSB protein stabilizes the single-stranded state. In *Pseudomonas aeruginosa*, SSB (PaSSB) is a homotetrameric protein, consists of an intrinsically disordered C-terminal domain containing a linker and a conserved acidic tail, 'DDDIPF', and an N-terminal DNA-binding domain with an OB-fold that binds to ssDNA. The linker of E. coli SSB contains 3 copies of the PXXP-motif, while PaSSB contains only one PXXP-motif and two PXPXX motifs. However, the DNA-binding activity of PaSSB and the effects of the PXXP-motif, temperature, and salt concentration on DNA-binding are poorly understood in *P. aeruginosa*. In this study, we aim to structurally and functionally characterize the PaSSB. Our study has shown that the thermal denaturation of PaSSB is reversible, allowing the protein to regain most of its activity upon temperature reversal. However, PaSSB demonstrates limited chemical stability, undergoing complete denaturation in 1.9M Guanidinium Chloride. Additionally, our study has demonstrated that the DNA-binding activity of PaSSB is dependent on temperature and salt concentration. Therefore, PaSSB is a crucial protein, and a cellular environmental balance is required for its biological role.

Structure and function insights into the DnaB helicase of *Pseudomonas aeruginosa*

Sunanda Gautam[1], Narayanan Manoj[1]

[1]*Indian Institute of Technology, Madras, Chennai, Tamil Nadu, India*

Presenting Author: Sunanda Gautam

Abstract:

DNA replication is a fundamental biological process that enables a cell to duplicate its genetic material. The DNA replication process has been well studied in *E.coli* and is conserved in most bacterial species. Among most bacterial species, loading of DnaB helicase on the replication fork site required DnaC or DnaI protein. *Pseudomonas aeruginosa* lacks these loading proteins, resulting in a distinctive loading strategy of PaDnaB. The current understanding of the function of PaDnaB helicases is not clear due to a lack of biochemical and structural information about this protein. To understand the domain-specific structure and function, we report the biochemical characterization of PaDnaB, including full-length and various deletion mutants of PaDnaB. The Cryo EM structure of PaDnaB shows that the protein is in a ring-shaped homo hexamer. The apo form of PaDnaB forms a trimer of Dimers. The CTD shows C6 symmetry while the NTD shows C3 symmetry. The complete characterization of PaDnaB will help to understand the distinctive structural and functional features of this protein.

Metabolic profiling in human postmortem brain under the influence of alcohol use

Indrani Paramasivan Latha Laxmi[1], Ramasamy Tamizhselvi[1]

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Presenting Author: I P Latha Laxmi

Abstract:

Studies reveal that alcohol consumption is associated with cognitive and metabolic deficits. Human prospective studies and rodent models show notable neural and cognitive effects due to increased apoptotic cells in brain tissue, causing increased oxidative stress. Elevated pro-inflammatory markers due to alcohol exposure are associated with the prognosis of neurodegenerative disorders. Alcohol has a strong influence on the neurotransmission receptors. Alcohol exposure increases the risk of depressive actions through its interaction with the benzodiazepine/GABA receptor complex. Research shows that glutathione and neurotransmitter systems were differentially expressed in different regions of brain tissue under the influence of alcohol. The purpose of the current study is to evaluate the metabolic profiling of human postmortem brain tissue exposed to alcohol compared to non-alcohol-exposed brain tissue samples using liquid chromatography-mass spectrometry. The differentially expressed metabolites among the groups were identified, and the key pathways involved were analysed. Results show that the detected metabolites were mainly involved in the neurotransmission system. Glutamate and Choline metabolites were affected due to alcohol exposure. Our findings provide insights into differentially dysregulated metabolites in human brain tissue and thereby contribute to targeting metabolites and therapeutic strategies to minimise the neurotransmission and metabolic impacts.

Natural Killer cell immunotherapy for augmentation of immune synapse formation and degranulation against leukaemia

Anushka Banerjee[1,2], Rakesh Kumar Majhi [1,2]

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[2]*Center for Excellence in Cancer, Gangwal School of Medical Sciences and Technology, IIT Kanpur*

Presenting Author: Anushka Banerjee

Abstract:

The transient receptor potential vanilloid 2 (TRPV2) ion channel is the highest expressed ion channel in Natural Killer (NK) cells, however its role in modulating immune cell function, remains unexplored. A critical knowledge gap exists in elucidating how TRPV2 regulates NK cells activity for effective tumor cell elimination. Our findings show that TRPV2 is highly expressed in human NK cells, and its activation leads to significantly increased expression of lytic molecules and cytokines, superior immune synapse formation. High-resolution confocal microscopy, and SEM reveal that TRPV2 facilitates better NK-leukemia cell contact, strengthening the synaptic interface and boosting cytotoxic function. TRV2 knock-out NK92 cells show 50% reduction in immune synapse formation, degranulation and leukemia cell killing. TRPV2 deficient NK cells show impaired serial killing activity. TRPV2-enriched educated NK cells show better leukemic cell killing capacity than TRPV2-low immature NK cells. TRPV2 mediates synapse activity by modulating the cytoskeletal dynamics, likely by Rho-Rac pathway. The membrane ruffling and lamellipodia are differentially affected by TRPV2 modulation, implying impact on lipid metabolism in NK cells. These data provide new insight into the molecular pathways by which TRPV2 augments innate immune responses, supporting the development of TRPV2-targeted strategies to improve NK cell immunotherapy for leukemia.

Therapeutic potential of human macrophage Piezo1 ion channel modulation ameliorating atherosclerosis progression

Srayasi Majee[1,2], Tamojit Santra[1,2], Santosh Kumar Misraa[2], Rakesh Kumar Majhia[2]

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Presenting Author: Srayasi Majee

Abstract:

Cardiovascular diseases account for one-third of global deaths annually, with atherosclerosis being a major contributor. Blood flow is limited by plaque formation, formed due to fatty deposits and impaired clearance of accumulated circulating cells. Macrophages transforminto sticky foam cells after excessive uptake of oxidized LDLs, and need to be cleared by healthy macrophages. Recently, we showed that the mechanosensitive ion channel Piezo1 present in macrophages, play a key role in atherosclerosis progression. However, its effect on macrophages are poorly studied. Our study shows that pharmacological modulation of Piezo1 ion channel in human PBMC derived macrophages alter oxidized LDL uptake, lipid droplet accumulation, foam cell formation, foam cell apoptosis, cytoskeletal organization and mitochondrial fragmentation. Piezo1 modulation alters cytoskeletal dynamics in macrophages. Piezo1 modulation alters the differentiation status of macrophages to proinflammatory M1 and anti-inflammatory M2 macrophages. Piezo1 activation reduced plaque size and inflammation by 30% in atherosclerotic mice. Moreover, we have formulated a liposome loaded with Piezo1 modulators, aimed at targeting it against atherosclerosis by intra-arterial injection to atherosclerotic mice. Our study highlights a novel approach using ion channel modulators to treat atherosclerosis and delineates the mechanistic basis involved in this interventional therapy.

Melatonin Protects Against T3-Induced Cardiac Hypertrophy Through Restoration of Antioxidant Defence and Mitochondrial Integrity

Razia Khatoon[1,2], Swaimanti Sarkar[1], Arefa Jasmin[2], Souradipta Chakraborty[1], Aindrila Chattopadhyay[2], Debasish Bandyopahyay[1*]

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Presenting Author: Razia Khatoon

Abstract:

Cardiac hypertrophy (CH) and subsequent heart failure are major causes of mortality worldwide. Triiodothyronine (T3), a critical thyroid hormone, regulates cardiac growth and metabolism; however, elevated T3 levels are known to induce pathological cardiac hypertrophy, aggravating cardiac dysfunction. Melatonin, a pineal indoleamine primarily known for its role in circadian rhythm regulation, also functions as a potent antioxidant with cardioprotective potential. The present study investigates the protective effects of melatonin pretreatment against T3-induced cardiac hypertrophy in adult male Wistar rats. T3 administration significantly increased heart weight, lipid peroxidation, and total reactive oxygen species (ROS) generation, as analysed by flow cytometry. Reduced glutathione (GSH) levels, as well as the activities of key antioxidant enzymes such as superoxide dismutase (SOD) and catalase—were markedly affected following T3 treatment. Pretreatment with melatonin effectively mitigated these alterations by reducing LPO and ROS generation, restoring GSH content, and normalizing antioxidant enzyme activities. Histological (H&E) and scanning electron microscopy (SEM) analyses revealed that T3 disrupted myocardial architecture and surface topology, while melatonin preserved tissue integrity. Furthermore, T3 induced mitochondrial membrane depolarization, as determined by JC-1 staining in FACS, which was attenuated by melatonin. These findings suggest that melatonin pretreatment confers significant protection against T3-induced cardiac hypertrophy by attenuating oxidative stress, preserving mitochondrial function, and maintaining cardiac structural integrity.

Rust Meets Roots: Plant-Derived Inhibitors Against the Protein Language of Corrosion

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Presenting Author: Kokilaramani Seenivasan

Abstract:

Microbiologically influenced corrosion (MIC) is not just an industrial challenge—it is a global threat to infrastructure sustainability and a hidden driver of antimicrobial resistance. Biofilm-forming microbes orchestrate corrosion through protein-regulated electron transfer and biofilm stabilization, while conventional biocides often worsen environmental toxicity. In this work, introduce a metaproteomics-guided strategy to unravel the molecular underpinnings of MIC and identify plant-derived green inhibitors as sustainable alternatives. Using high-resolution LC-MS/MS, microbial consortia from corroded steel in industrial cooling systems were analyzed. Functional annotation via UniProt and KEGG revealed key corrosion-associated pathways, including upregulated outer membrane cytochromes, hydrogenases, and quorum-sensing proteins, which collectively drive biofilm persistence and metal reduction. To counteract these pathways, evaluated phytochemical inhibitors—polyphenols, alkaloids, and terpenoids—through electrochemical impedance spectroscopy, confocal microscopy, and antimicrobial assays. Strikingly, these green inhibitors disrupted biofilm architecture, reduced corrosion current densities, and outperformed synthetic biocides in terms of biodegradability, cytocompatibility, and dual antimicrobial–anticorrosion activity. The findings establish a new paradigm linking protein expression to corrosion dynamics and demonstrate that metaproteomics can directly guide the design of eco-friendly corrosion inhibitors. Beyond industry, this framework has translational potential in biomedical device protection, infection control, and sustainable engineering—where the fight against corrosion converges with the quest for conservation.

NovoMolGen: Rethinking Molecular Language Model Pretraining

Roshan Balaji[1], Kamran Chitsaz[2], Quentin Fournier[2], Nirav Pravinbhai Bhatt[1], Sarath Chandar[2]

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[2]*Mila-Québec AI Institute, Montréal, Québec, Canada*

Presenting Author: Roshan Balaji

Abstract:

Designing de novo molecules with desired property profiles requires efficient exploration of the vast chemical space ranging from 10^23 to 10^60 possible synthesizable candidates. While various deep generative models have been developed to design small molecules using diverse input representations, Molecular Large Language Models (Mol-LLMs) based on string representations have emerged as a scalable approach capable of exploring billions of molecules. However, there remains limited understanding regarding how standard language modeling practices such as textual representations, tokenization strategies, model size, and dataset scale impact molecular generation performance. In this work, we systematically investigate these critical aspects by introducing NovoMolGen, a family of transformer-based foundation models pretrained on 1.5 billion molecules for de novo molecule generation. Through extensive empirical analyses, we identify a weak correlation between performance metrics measured during pretraining and actual downstream performance, revealing important distinctions between molecular and general NLP training dynamics. NovoMolGen establishes new state-of-theart results, substantially outperforming prior Mol-LLMs and specialized generative models in both unconstrained and goal-directed molecular generation tasks, thus providing a robust foundation for complex predictive tasks like retrosynthesis and text instructed molecule design.

Advancing Protein-Ligand Binding Site Prediction Using Attention-Enhanced 3D Graph Neural Networks

Sukanya Tukaram Naik[1], M. S. B. Roshan[2], Nirav Pravinbhai Bhatt[1,2]

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Presenting Author: Sukanya Naik

Abstract:

Introduction: Identifying precise protein-ligand binding sites is a major challenge in structural biology, crucial for drug discovery and understanding molecular mechanisms. This study reframes docking as a graph interaction prediction problem, representing both proteins and ligands as 3D molecular graphs. Objectives: We employ Graph Neural Networks (GNNs) to analyze these graph-structured data, training on the the next generation : PINDER dataset, to predict optimal rotation and translation for accurate protein-ligand alignment. Our model architecture integrates Equivariant Message Passing Neural Networks (EquiMPNN) and a multi-head cross-attention mechanism to capture complex receptor-ligand interactions. Key Findings: Ablation studies reveal that incorporating attention layers significantly improves docking accuracy, with models using two attention layers achieving higher CAPRI scores compared to those without attention. This demonstrates that attention mechanisms are essential for capturing critical interaction regions and enhancing structural prediction. To the best of our knowledge this is the first study that interpretes proteins and ligands as 3D structural graphs and solely relying on these structures predict the binding transformations in protein-ligand interactions.

Investigating a bioconjugation-free glucometer-enabled readout for hemin DNAzyme transducer and application in a biosensor

Saba Parveen[1], Arunansu Talukar[2], Mrittika Sengupta[1], Souradyuti Ghosh[1]

[1]*Centre for Life Sciences, Mahindra University, Hyderabad*
[2]*Department of Geriatric Medicine, Medical College and Hospital Kolkata, West Bengal*

Presenting Author: Dr Souradyuti Ghosh

Abstract:

The development of affordable and accessible disease diagnostic tools is crucial for democratizing healthcare. Personal glucometers, widely available and easy to use, have emerged as promising platforms for decentralized molecular detection beyond glucose monitoring. Horseradish peroxidase (HRP) mimic hemin-G-quadruplex DNAzyme transducers is one of the most widely used molecular toolkits that has been extensively utilized in aptamer-linked immobilized sorbent assay (ALISA) for detecting pathogen, cancer, toxic metals, metabolites, and DNA/RNA. In this work, we introduce a novel method to integrate HRP-mimic DNAzyme activity with glucometer readout and investigate assay conditions such as buffer, pH, peroxide, and redox mediators to determine the suitable operational window for biosensing measurement. The assay's adaptability to different commercial glucometer brands and initial testing with native HRP indicated broad applicability, including that of enzyme-linked aptasorbent assay (ELISA). Finally, a dengue virus target sequence was detected using the DNAzyme transducer with a femtomolar limit of detection and a 9-order-of-magnitude dynamic range. These findings lay foundational groundwork toward transforming glucometers into versatile, low-cost biosensors for enzyme-based immunoassays, potentially replacing centralized ELISA/ALISA instrumentation with portable, user-friendly, and equitable alternatives towards molecular disease detection technologies worldwide.

Exploring the ribosomal protein gene expression signatures as lineage-dependent markers of gastric cancer progression

Aishwarya Murali[1,2,3], Chartsiam Tipgomut [4], Anand D. Jeyasekharan [4,5,6,7], Himanshu Sinha[1,2,3,*]

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Presenting Author: Aishwarya Murali

Abstract:

Introduction Ribosomes, composed of rRNA and ribosomal proteins (RP), exhibit tissue-specific variability, known as ribosome heterogeneity, which preferentially translates certain mRNAs to tailor cellular function. In cancer, the changes in the RP gene expression can promote preferential translation of oncogenic mRNAs. Gastric adenocarcinoma (GAC), which is the fifth most common cancer and is known for its heterogeneity, serves as an important model for studying differences in RP gene expression. Objectives To investigate the heterogeneity in RP gene expression in cell types from a tumour compared to cell types from normal tissue in GAC. To determine how RP expression patterns shift during different stages of tumour progression at the cellular level. Additionally, we seek to analyse the expression patterns of RP genes across different molecular subtypes of gastric cancer. Key Findings By analysing publicly available single-cell RNA sequencing datasets, we found that RP gene expression varies during tumorigenesis and tumour progression in a lineage-dependent manner. Furthermore, RP genes exhibit differential expression in specific molecular subtypes of gastric cancer. We observed that RP gene expression is dysregulated in cancer, and the non-linear patterns of gene expression indicate a heterogeneous requirement for different RP genes at various stages of cancer.

Unlocking fungal pharmacogenomics: A systems-level approach to mapping the genetic basis of multiple drug-drug interactions

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Presenting Author: Amrish P

Abstract:

The urgent need for antifungal therapies has driven interest in drug repurposing and combinations. While bacterial research has investigated how genetic and experimental factors influence drug combination responses, similar questions for fungi remain largely unexplored. Our pilot study screened six diverse Saccharomyces cerevisiae strains with a combination of ibuprofen with either clotrimazole or caspofungin. The initial results showed that each drug combination's effect on the yeast was unique, with varying degrees of influence from genotype, media, assay type, and drug concentration. To further investigate the genetic basis of these complex interactions, we conducted a high-throughput screen of 1,011 S. cerevisiae isolates using the same drugs in two different media. We observed a diverse mosaic phenotypic landscape unrelated to the strains' clade or ecological niche. In some cases, environmental changes dramatically altered the interaction, as seen with simvastatin-clotrimazole, where most of the interaction pattern shifted from synergy to antagonism with the change in media. Currently, we are using Genome-Wide Association Studies (GWAS) to identify the genetic polymorphisms linked to these phenotypes. The goal is to get insights to predict complex drug interaction patterns from genomic and environmental data, providing a framework for developing personalized antifungal therapies and predicting multi-drug resistance.

Traditional Medicine Meets Modern Mechanism: Amalaki Rasayana Modulates Galectin-3-C-Epitope Oligomerization in Cardiac Hypertrophy

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Abstract:

Traditional Medicine Meets Modern Mechanism: Amalaki Rasayana Modulates Galectin-3-C-Epitope Oligomerization in Cardiac Hypertrophy Abstract Introduction: Pressure overload–induced cardiac hypertrophy (PO-CH) is a major precursor to heart failure, the molecular mechanisms behind its progression is yet incompletely understood. Galectin-3 (Gal-3), a β-galactoside-binding lectin, has been implicated in cardiac remodeling, but the role of its C-terminal epitope oligomerization is unclear. Objectives: This study aimed to identify the extracellular Oligomeric state of the Gal-3 C-terminal epitope (Gal-3-C-epitope) as a mediator of PO-CH and evaluate the therapeutic potential of Amalaki Rasayana (AR), an Ayurvedic formulation rich in Gallic acid (GA), in modulating Gal-3–dependent signaling. Key Findings: In rodent models of aging and PO-CH, Gal-3-C-epitope oligomers were elevated in serum and on cardiomyocytes surface, correlating with adverse remodeling. AR/GA treatment reduced Gal-3-C oligomers and hypertrophic signaling by promoting Gal-3 Ser6 phosphorylation, enhancing intracellular retention, and preventing extracellular oligomerization and cell-surface binding. Human and animal data confirmed therapy-associated declines in Gal-3-C-epitope oligomers. Conclusion: Gal-3-C-epitope oligomers represent novel targets and drug therapy efficacy monitoring biomarker in cardiac hypertrophy, and support the potential of AR/GA as inhibitors of Gal-3 C-epitope-driven pathology.

Computational Screening And Molecular Docking Of Phytochemicals For Therapeutic Intervention In Arthritis

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Presenting Author: Namrata Britto

Abstract:

Arthritis, a chronic, inflammatory disorder affecting millions globally, demands safer and more effective therapeutic strategies beyond conventional drugs. Phytochemicals, with their structural diversity and bioactive potential, represent a promising resource for developing novel anti-arthritic agents. This study aims to screen and identify potent phytochemicals targeting key molecular regulators for arthritis – NLRP3 and CD28 (achieved through network pharmacology) using an integrated computational approach. A curated library of plant-derived compounds from over 200 anti-arthritic plants was evaluated through virtual screening, including ADMET profiling and molecular docking. The interaction analysis was checked to assess binding affinity and molecule interactions. Based on these analyses, three top-ranking phytochemicals were shortlisted for each target exhibiting strong and specific interactions within the active binding domains of NLRP3 and CD28. These candidates are being further investigated through molecular dynamics simulations to validate structural stability and biological relevance. The study provides a translational framework linking natural compound discovery to therapeutic development.

Binding-induced transitions in intrinsically disordered regions in protein-protein and protein-nucleic acid complexes

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Presenting Author: Prachi Bhargava

Abstract:

Intrinsically disordered regions (IDRs) lack stable three-dimensional folds and are frequently observed as missing residues in experimental structures. We study the conformational changes in IDRs when protein binds to other macromolecules. A non-redundant dataset of 356 protein-protein (PP) and 228 protein-nucleic acid (PNA) complexes, along with their unbound proteins, was analysed, and the transitions in IDRs were classified into three classes: Disordered-to-Ordered (D-O), Disordered-to-Partially Ordered (D-PO), and Disordered-to-Disordered (D-D). In both PP and PNA, the IDRs in D-O class predominantly adopt coils (77%), followed by helices and sheets. Helix formation is more common in PNA (20%) than in PP (15%), while sheets are higher in PP (8% vs 4% in PNA). Though a majority of IDR residues are solvent exposed, a significant number are present at interface and form polar bonds. Interface IDRs prefer more hydrogen bonds (H-bonds) formation with phosphates (50%) in DNA, while bases are preferred more in RNA (43%). Sugars contribute least, with ribose forming more H-bonds (25%) than deoxyribose (4%). Aromatic residues contribute more within PP interface IDRs, whereas charged residues predominate in PNA. We also report ordered-to-disordered transitions in IDRs upon complexation, indicating conformational changes can occur in both directions depending on the functional requirements.

Cellular and chromosomal interaction of bio-synthesized copper oxide nanoparticles - Induced nano-cytotoxicity and genotoxicity

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Presenting Author: Vijaya P.P

Abstract:

The widespread use of nanoparticles raises substantial environmental, health, and safety issues. The specific mechanisms by which they impact plants and animals, as well as the entire scope of their possible impact, are still unknown. The current work investigates the impact of varying CuO NPs concentrations on phytotoxicity, cytotoxicity, genotoxicity, and antioxidant activity. Exposure of Mung bean seeds to CuO NPs results in the uptake of these particles by the roots and their subsequent transportation to various plant components, including the root, stem, and leaf. This uptake of CuO NPs triggers the production of reactive oxygen species (ROS). The CuO NPs can induce toxicological consequences due to their heightened propensity to produce excessive amounts of ROS. The absorption of CuO NPs might cause deformation and disintegration of the erythrocyte membrane, resulting in cell rupture due to osmotic pressure. The Allium cepa cytotoxicity study aimed to examine the harmful effects of CuO NPs and assess their impact on cellular structures at a microscopic scale. This work aims to analyze the cellular interaction of CuO NPs by measuring the Mitotic Index (MI) in the root cells of Allium cepa. The CuO NPs rapidly interact with plant and human cells, as well as chromosomes, leading to nanophytotoxicity, nano-cytotoxicity, and nano-genotoxicity.

Membrane Lipids as Key Regulators of GPCR Dynamics

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Presenting Author: Punita Kumari

Abstract:

Lipid-activated GPCRs, such as FFAR1 and CB1, display unique structural features that distinguish them from traditional GPCRs. Unlike receptors activated by soluble ligands at conserved orthosteric sites, these receptors leverage flexible transmembrane (TM) helices to allow lipids to access the receptor directly from the membrane. This dynamic TM movement is central to how lipid ligands modulate receptor activity. Our studies show that ligands binding at the lipid-facing surface of FFAR1 and CB1 are druggable and can bypass the conserved orthosteric site. This enables selective activation of downstream signaling pathways, effectively decoupling orthosteric ligand binding from effector engagement. Such lipid-facing pockets provide new opportunities to design therapeutics with high specificity and reduced side effects. In summary, lipid-activated GPCRs exploit flexible TM motions to accommodate lipids from the membrane, and these lipid-exposed surfaces represent novel druggable sites. Understanding these mechanisms not only expands our knowledge of GPCR regulation but also offers a framework for developing next-generation modulators targeting membrane-facing pockets.