6th Edition

FS-BIO'26

FRONTIERS SYMPOSIUM IN BIOLOGY

IISER THIRUVANANTHAPURAM

ABSTRACT BOOK

6th Edition

IISER THIRUVANANTHAPURAM

SCHOOL OF BIOLOGY
IISER Thiruvananthapuram

FS-BIO'26

FRONTIERS SYMPOSIUM IN BIOLOGY

FEB
13th-15th
2026

ARYABHATTA
LECTURE HALL COMPLEX

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About FS BIO 2026

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The Frontiers Symposia in Biology is an annual conference organized by the School of Biology at IISER Thiruvananthapuram. The sixth edition of symposium will be held from 13th to 15th February, 2026 on our campus. The meeting brings together a broad range of cutting-edge research topics across all areas of biology. Students and researchers from across the country gather to exchange ideas and discuss science in our picturesque campus nestled at the foothills of the Western Ghats.

IISER
THIRUVANANTHAPURAM

Organizing Committee

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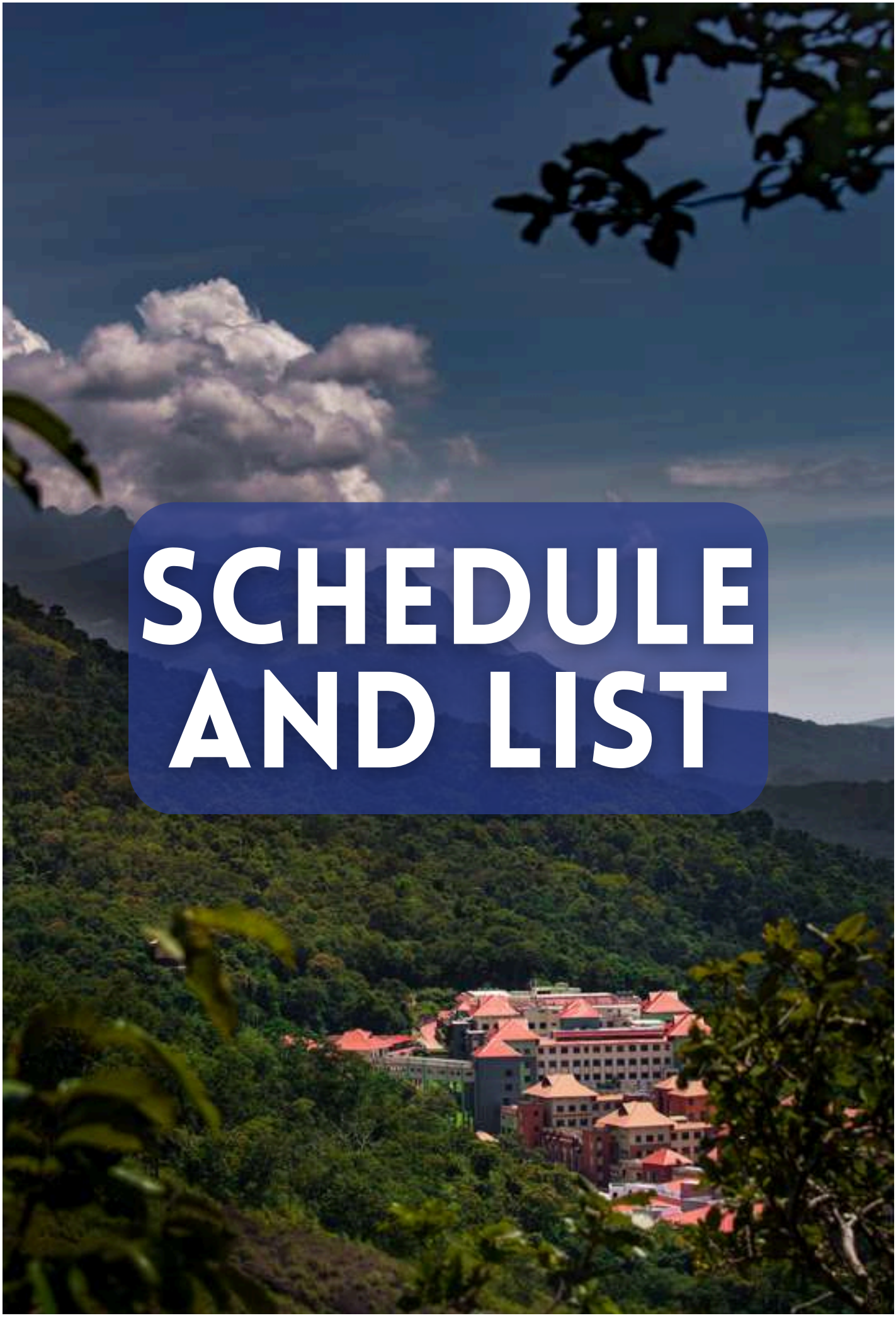

SCHEDULE AND LIST

Day 1 (Friday, 13 February 2026)

11:00 - 15:00	**REGISTRATION**
15:00 - 15:15	Inaugural Address **Prof. J. N. Moorthy, Director, IISER Thiruvananthapuram**
15:15 - 15:30	**Announcements**
	SESSION 1 **Chair:** Sanu Shameer
15:30 - 15:55	**Alok Krishna Sinha,** BRIC-National Institute of Plant Genome Research, New Delhi
15:55 - 16:20	**Chittur V Srikanth,** BRIC-Regional Centre for Biotechnology, Faridabad
16:20 - 16:45	**Tamal Das**,Tata Institute of Fundamental Research, Hyderabad
16:45 - 17:15	Announcements & Refreshments
	SESSION 2 **Chair:** Nishana Mayilaadumveettil
17:15 - 17:40	**Jayanta Mukhopadhyay,** Bose Institute, Kolkata
17:40 - 18:05	**Amrita Hazra,** Indian Institute of Science Education and Research, Pune
18:05 - 18:30	**Altaf Bhat,** University of Kashmir, Srinagar
18:30 - 20:00	**Odd numbered posters (Venue: LHC Corridor)**
20:00	Dinner

Day 2 (Saturday, 14 February 2026)

SESSION 3 **Chair:** Manish Kumar and Karthik Chandiran	
09:00 - 09:25	**Jayasri Das Sarma**, Indian Institute of Science Education and Research, Kolkata
09:25 - 09:50	**Dhiraj Kumar**, International Centre for Genetic Engineering and Biotechnology, New Delhi
09:50 - 10:15	**V Stalin Raj**, Indian Institute of Science Education and Research, Thiruvananthapuram
10:15 - 10:40	**Narottam Acharya,** Institute of Life Sciences, Bhubaneswar
10:40 - 11:10	Announcements & Refreshments
SESSION 4 **Chair:** Amrutha Swaminathan	
11:10 - 11:35	**Deepa Subramanyam,** BRIC-National Centre for Cell Science, Pune
11:35 - 12:00	**Avinash Bajaj,** BRIC-Regional Centre for Biotechnology, Faridabad
12:00 - 12:25	**Amitabha Bandyopadhyay,** Indian Institute of Technology, Kanpur
12:25 - 14:30	Group photo & Lunch
SESSION 5 **Chair:** Yashraj Chavhan	
14:30 - 14:55	**Mahesh Sankaran,** National Centre for Biological Sciences, Bengaluru
14:55 - 15:20	**Imroze Khan,** Ashoka University, Sonepat
15:20 - 15:45	**Guha Dharmarajan,** Krea University, Andhra Pradesh
15:45 - 16:10	**Hema Somanathan,** Indian Institute of Science Education and Research, Thiruvananthapuram

16:10 - 16:45	**Announcements & Refreshments**
	SESSION 6 **Chair:** Vijay Jayaraman
16:45 - 17:10	**Sanjeev Shukla,** Indian Institute of Science Education and Research, Bhopal
17:10 - 17:35	**Gautham Nadig,** Mynvax Private Limited, Bengaluru
	SESSION 7 A Panel Discussion
17:35 - 18:30	**The Leaky Pipeline : Understanding Gender Gaps in Academia** (Commemorating International Day of Women and Girls in Science) Moderator : Sandhya Sekar
18:30 - 20:00	**Even numbered posters (Venue: LHC Corridor)**
20:00	**Dinner**

Day 3 (Sunday, 15 February 2026)

	SESSION 8 **Chair:** Sandhya Ganesan and Swathi Devireddy
09:00 - 10:00	**Flash Talks Session I**
10:00 - 11:00	**Flash Talks Session II**
11:00 - 11:30	Announcements & Refreshments
	SESSION 9 **Chair:** Anirban Guha
11:30 - 11:55	**Parveen Chhuneja,** Punjab Agricultural University, Ludhiana
11:55 - 12:20	**Rohini Garg,** Shiv Nadar University, NCR Delhi
12:20 - 12:45	**Bandan Chakrabortty,** Indian Institute of Science Education and Research, Thiruvananthapuram
12:45- 01:15	**Concluding Session, Prize Distribution and Remarks**

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MPK4 Emerges as a Key Regulator of Thermosensing in Arabidopsis.

2. Chittur V Srikanth
Understanding traits of emerging non-typhoidal Salmonellae and their long-term consequences.

3. Tamal Das
Organelles as Mechanochemical Integrators: Orchestrating Collective Cell Decisions

4. Jayanta Mukhopadhyay
Fundamental mechanism of transcription in bacteria.

5. Amrita Hazra
Using vitamins to understand microbial community dynamics and patterning.

6. Altaf Bhat
Heterochromatin in 3D Genome Organisation and Genome Stability.

7. Jayasri Das Sarma
A Coordinated CD40–CD40L–Ifit2 Axis Orchestrates Antiviral Neuroinflammation and Protects Against Virus-Induced Demyelination.

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8. Dhiraj Kumar
Hijacking the messenger- host RNA splicing machinery as the target of *Mycobacterium tuberculosis.*

9. V Stalin Raj
Self-assembled nanoparticle decorated with spike protein elicits robust humoral responses against emerging coronaviruses.

10. Narottam Acharya
Antifungal vaccine: A possible reality.

11. Deepa Subramanyam
Trafficking: rules to make a healthy organism.

12. Avinash Bajaj
Targeting of Tripartite Neuron-Cancer-Immune Cell Cross-talk Activates the Tumour Microenvironment.

13. Amitabha Bandyopadhyay
Investigating the molecular mechanism of limb tendon development - discovery of a possible tendon organizing centre.

14. Mahesh Sankaran
Forest-grassland mosaics: history, dynamics and an uncertain future.

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15. Imroze Khan
Pathogen growth and virulence dynamics drive the host evolution against coinfections.

16. Guha Dharmarajan
Do elephants really address one another by name?

17. Hema Somanathan
Minimal distortion of web vibrations facilitates collective prey capture in social Spiders.

18. Sanjeev Shukla
CTCF-mediated epigenetic control of splicing fuels hypoxia-induced EMT.

19. Gautham Nadig
Biotechnology spin-off for clinical translation, manufacturing readiness, and pandemic preparedness.

20. Parveen Chhuneja
Genomics-Enabled Utilization of Wild Wheat Relatives for Accelerated Wheat Improvement.

21. Rohini Garg
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22. Bandan Chakrabortty

A Dynamic Vertex Model Reveals Coupling Between Contractility and Adhesion in Polarized Tissue Flow

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1. Aashima

Metal organic framework -polymeric hybrid Diabetic wound healing Patch for improved Transdermal Co-Delivery of Curcumin and Heparin Drugs

2. Abinash Roy

Cortical Dynamics of Disengaged Awareness: Dissociation Between Global Alpha Suppression and Frontal Asymmetry in Adolescents

3. Agrawal Ajay Hariprasad

Understanding the role of micro-RNA in regulation of circadian rhythm in *Drosophila melanogaster*

4. Akshaya Rajan

Diabetes-induced peripheral dysregulation exacerbates Parkinson's disease pathology in mice.

5. Amrita Bhattacharya

Investigating lysosomal biology and inter-organelle cross-talk during infection with lysosome-adapted pathogen, *C. burnetii*

6.Amrutha Krishnakumar

Structural and functional studies of Mycobacterial transcription regulators-the Gre factors

7. Anagha Prakash

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9. Anuraag Nallan Chakravarthi
Replication-transcription collisions influence gene expression in bacteria

10. Anuraag Srinath Avadhany
Evolution of Defense Systems

11. Ashlin Raj
Where Cells Decide to Divide: The Dynamics of the Preprophase Band

12. Dea Vincent
Replication-dependent origin of genome organization in bacteria

13. Deshmukh Abhay Laxmikant
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14. Dhanagovind P T
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16. Eeshani Abhyankar & Anushka Tyagi

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17. Fahis K.T

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18. Gayathri Binu

Investigating the adult relevance of larval hexamerins in *Drosophila melanogaster*

19. Geethika Shrimali

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20. Girija Jogwar

Understanding the role of mismatch repair in regulating meiotic recombination

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23. Gowtham K P
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24. Hana Lukman
The role of *miR-986* in the metabolism and lifespan of *Drosophila melanogaster.*

25. Haritha Hari
Structural studies of *Mycobacterium tuberculosis* LexA alone and in complex with its interacting partner: Insights into SOS Regulation

26. Janu Waghmore
SDS PAGE patterns of salivary gland proteins in Ecdysone and Hydroxyurea treated larvae

27. Janvi Jayachandran
Understanding the Roles of NASP and SGTI in CENH3 Deposition and Genome Stability in *Arabidopsis thaliana*

28. Jyotirmayi J P
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29. Killivalavan N
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30. Krishna M Nair
Can winter crops embrace warming? We report novel thermal phenotypes and key photosynthetic functional targets for temperature resilience in rapeseed-mustard

31. K S Niranjan & Samyukta Anand
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32. Dr K. Venkateshwaran
Hypocrellin B with carbon dots loaded hydrogel for the photodynamic treatment of Diabetic retinopathy

33. Malini Bhattacharyya
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34. Mayukh Mitra
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35. N. Amrissh Prakash
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37. Padmanabhan Kannan
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38. Parnika Sahoo
Toxoplasma gondii co-opts the Host ACVR1–Iron Axis to Establish a Permissive Replicative Niche

39. Pradip Bhattacharjee
Regulation of Primary Cilia Assembly by Transforming Acidic Coiled-Coil Protein 3 (TACC3) in Human Cells

40. Priyanka Jeeth
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44. R Vedaasri
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45. Sai Prasanth KRS
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46. Saswata Sahoo
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47. Shahnaz
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53. Sudhanshu Ranjan Singh
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54. Subhankar Dey
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55. Swapna Nair
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2. Anjitha K
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3. Aswathy BJ
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4. Delvin K Pauly
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5. Kushankur Bhattacharyya
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9. Ritobrato Chatterjee
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10. Santhosh Kumar S
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11. Vaisak Mohan
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12. Vismaya Prakash
When Flies Choose the Night: Understanding the Role of microRNA-9b in Circadian Rhythms


SPEAKERS

Alok Krishna Sinha

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MPK4 Emerges as a Key Regulator of Thermosensing in Arabidopsis.

Plants can perceive a slight upsurge in ambient temperature and respond by undergoing morphological changes, such as elongated hypocotyls and early flowering. The dynamic functioning of PHYTOCHROME INTERACTING FACTOR4 (PIF4) in thermomorphogenesis is well established, although the complete regulatory pathway involved in thermosensing remains elusive. We establish that an increase in temperature from 22° C to 28° C induces upregulation and activation of MITOGEN-ACTIVATED PROTEIN KINASE 4 (MPK4) in Arabidopsis thaliana, subsequently leading to the phosphorylation of PIF4. Phosphorylated PIF4 represses the expression of ACTIN-RELATED PROTEIN 6 (ARP6), required for mediating the deposition of histone variant H2A.Z at its target gene loci. Further, we demonstrate that variations in ARP6 expression in PIF4 phosphor-null and phosphor-mimetic seedlings affect hypocotyl growth at elevated temperature by modulating the regulation of ARP6-mediated H2A.Z deposition at the loci of genes involved in elongating hypocotyl cells. Interestingly, the expression of MPK4 is also controlled by H2A.Z deposition in a temperature-dependent manner. Taken together, these findings highlight the regulatory mechanism of thermosensing by which MPK4-mediated phosphorylation of PIF4 affects ARP6-mediated H2A.Z deposition at the genes involved in hypocotyl cell elongation.

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Chittur V Srikanth

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Understanding traits of emerging non-typhoidal Salmonellae and their long-term consequences.

Non-typhoidal Salmonella induced gastroenteritis is still a significant health challenge in both developing and developed world. Emergence of drug resistance strains and invasive strains have furthered the importance of these pathogens. Furthermore, chronic infections of salmonellae have shown to cause long term side effects such as gallbladder cancer. However, its causative role and the underlying mechanisms are largely unknown. Gallbladder cancers (GBCs) are known to be most aggressive and lethal forms of gastrointestinal cancers. Major contributing factors of GBC are chronic inflammation of gallbladder, presence of gallbladder stones (gallstones) and genetic aberrations. It is known that gallbladder tissue serves as a reservoir for Salmonella during chronic infections. We studied gut and gallbladder tissue microbiome through targeted metagenomics to identify pathogenic bacteria in GBC. Virulence and pathogenicity of identified SalmonellaTyphimurium from GBC tissue was studied after culture by whole genome sequencing, phylogenetic analysis, mutational profiling, and pangenome analysis. Mechanistic studies for GBC carcinogenesis were carried out in a mouse model of gallstones and chronic Salmonella infection, cellular model using GBC (NOZ) cell lines and a xenograft tumor model. Chronic S. Typhimurium infection caused chronic inflammation, pre-malignant changes, and tumor promoting mechanisms in the mouse model with gallbladder stones with activation of the epigenetic modulator Kdm6b both in the mouse model and human GBC. Inhibition of Kdm6B reduced engrafted tumor size in SCID-mice. Of the differentially regulated genes in human GBC tissue, ADAMTSL5, CX3CRI and SPSB4 were also significantly dysregulated in NOZ cells infected with Salmonella. Chronic Salmonella infection contributes to gallbladder carcinogenesis through a host epigenetic mechanism involving Kdm6b.An increased abundance of Salmonella in the gut microbiome of patients with GBC and culturable S. Typhimurium from the GBC tissue was also observed. Comparative genomics of S. Typhimurium isolated from the GBC tissue showed a high invasive index. S. Typhimurium isolates harbored horizontally acquired virulence functions in their accessory genome. Together, these attributes highlight that NTS is rapidly evolving to become invasive and to persist longer in human host. Many aspects of these traits are still unknown.

IISER THIRUVANANTHAPURAM

Tamal Das

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Organelles as Mechanochemical Integrators: Orchestrating Collective Cell Decisions.

Cells are traditionally viewed as biochemical reactors, with organelles performing well-defined classical functions. However, this view overlooks their material properties and their intimate coupling to cytoskeletal forces. We propose instead that organelles actively shape how mechanical and chemical information is processed, acting as structural elements that integrate tension, geometry, and signaling. In this talk, I will present a framework viewing organelles as mechanosensitive structural materials that bridge physical forces and biochemical signaling. Using epithelial wound healing as a model, I will show how the endoplasmic reticulum (ER) acts as a curvature-sensitive network that encodes tissue geometry into local actin dynamics, and how lysosomes function as force-responsive actuators that interpret contractile stress to control Rac1 activation and leader cell emergence. Together, these organelles form a hierarchical mechanochemical circuit that links geometry, tension, and fate decisions in collective migration.

IISER THIRUVANANTHAPURAM

Jayanta Mukhopadhyay
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Fundamental mechanism of transcription in bacteria.

I think in this talk, I will cover two aspects of transcription: RNAP assembly in B. subtilis and 's cyle' in B. subtilis and M. tuberculosis.
RNAP assembly: The bacterial RNA polymerase (RNAP) core enzyme, responsible for transcription, is composed of five conserved subunits: α_2, β, β′, and ω. In Escherichia coli, RNAP assembly follows a well-established sequential pathway: $\alpha + \alpha \rightarrow \alpha_2 \rightarrow \alpha_2\beta \rightarrow \alpha_2\beta\beta'(\omega)$. This canonical scheme has long been considered universal. Here we show that in Bacillus subtilis, RNAP assembly proceeds not only via the canonical route but also through an alternative pathway: $\alpha + \alpha \rightarrow \alpha_2 \rightarrow \alpha_2\beta'(\omega) \rightarrow \alpha_2\beta\beta'(\omega)$. We provide in vivoevidence for both $\alpha_2\beta$ and $\alpha_2\beta'$ intermediates in B. subtilis, whereas E. colisupports only the $\alpha_2\beta$ intermediate. These findings uncover a previously unrecognized plasticity in bacterial RNAP assembly, attributable to distinct α–β and α–β′ interfaces in different lineages. Our results highlight the evolutionary diversification of RNAP assembly and suggest new opportunities for developing species-specific antibiotics that target lineage-dependent assembly pathways.
σ cycle: A "σ cycle" in which the initiation factor σ associates with RNA polymerase (RNAP) core enzyme to permit transcription initiation and dissociates from RNAP core enzyme to permit transcription elongation, has been proposed to occur and to be an essential step for σ-exchange, with all principal σ factors from all bacteria. These proposals were based on studies of the principal σ factor of Escherichia coli, σ70, which, generally, albeit not obligatorily, is released from RNAP upon the transition from transcription initiation to elongation. Here, we show that, in contrast to E. coli σ70, the Bacillus subtilis principal σ factor, σA, is not released and is retained on RNAP core throughout transcriptionp elongation. We further show that a mutant E. coli σ70 derivative lacking σ region I.I (σ RI.I) is not released and is retained on RNAP core throughout transcription elongation. We also observe that B. subtilis σA and the mutant E. coli σ70 derivative lacking σ RI.I interact much more stably with RNAP than full-length E. coli σ70. We further observed that in M. tuberculosis, the principal σ factor σA and the alternative σ factor σE are immediately or stochastically released upon the transition from transcription initiation to elongation, whereas the other sigma factor, σF, is retained throughout the elongation. Incidentally, σF contains all three σ regions except region I.I. Our results indicate that the σ cycle is not a universal phenomenon in bacteria; the σ factor that includes all three σ regions except I.I is likely to be retained in the elongation complex.

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Amrita B. Hazra

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Using vitamins to understand microbial community dynamics and patterning.

Microbial communities consist of a diversity of microorganisms that collectively conduct a spectacular range of metabolism in the natural world. Within the communities, nutrients and resources are shared and the microbes compete and cooperate with one another to build a stable network of interactions.

Vitamins such as BI, B3, B6, B7 and BI2 are among the repertoire of critical nutrients that are exchanged in the human gut and marine microbial communities. The modular nature of the biosynthesis pathways of vitamin BI and B6 is well-suited for different microbes to produce intermediates, which can then be combined to synthesize the whole vitamin. In our lab, we have created synthetic microbial co-cultures that rely on the exchange of biosynthesis intermediates of vitamin BI and B6. Using these co-cultures, we show how vitamins BI and B6 can be synthesized and exchanged and the microbial co-culture dynamics that allow for stable co-cultures to form. Further, we use microscopy and modelling studies to understand why and how they localize themselves to optimize sharing of metabolites. Our molecular level studies in the context of synthetic microbial consortia provide mechanistic and functional insights into how vitamins and other metabolites might be synthesized and shared among microbes in complex communities.

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Altaf Bhat

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Heterochromatin in 3D Genome Organisation and Genome Stability.

The eukaryotic genome is spatially segregated in the nucleus into discrete structural and functional chromatin domains. The genome is organized into euchromatin and heterochromatin distinguished on the basis of their appearance, organization, localization, function, and the type of histone modifications on nucleosomes. Heterochromatin across species is tethered towards the nuclear periphery and associates with number of Inner Nuclear Membrane proteins (INM) proteins. Association with INM proteins is essential for tethering and silencing. We will discuss the role of INM proteins in heterochromatin formation, 3D genome organisation and genome stability.

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Jayasri Das Sarma

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A Coordinated CD40–CD40L–Ifit2 Axis Orchestrates Antiviral Neuroinflammation and Protects Against Virus-Induced Demyelination.

Neurotropic murine β-coronaviruses, such as MHV-A59 and its isogenic recombinant strain RSA59, provide a robust experimental platform for dissecting virus-induced neuroinflammation and demyelination, pathologies that mirror aspects of Multiple Sclerosis (MS). Our cumulative findings identify a coordinated neuromodulatory axis involving the CD40–CD40L immune checkpoint dyad and the interferon-stimulated gene Ifit2 as critical determinants of the transition from acute neuroinflammation to chronic, progressive demyelination. CD40 expressed on microglia/macrophages and endothelial cells, and CD40L on infiltrating $CD4^+$ T cells, together orchestrate antiviral immunity by promoting microglial activation, T-cell priming in cervical lymph nodes, and effective T-cell recruitment to the CNS. Genetic ablation of CD40 or CD40L results in dampened microglial/macrophage responsiveness, impaired effector T-cell entry, uncontrolled viral replication, and severe chronic demyelination marked by axonopathy and poliomyelitis. In parallel, the IFN-induced tetratricopeptide protein Ifit2 acts as a potent antiviral effector by directly interacting with the RSA59 nucleocapsid protein, thereby limiting replication and spread. Ifit2 deficiency causes extensive viral dissemination throughout the brain, spinal cord gray and white matter; reduced CX3CRI-dependent microglial activation, attenuated $CD4^+$ and $CD8^+$ T-cell infiltration, and persistent viral load. Although the blood–brain barrier remains relatively intact in Ifit2-/- mice, insufficient leukocyte recruitment and impaired lymph node T-cell activation ultimately led to profound chronic demyelination. Together, these studies reveal that CD40–CD40L signaling and Ifit2 function together promote early antiviral neuroinflammation, which is essential for viral clearance and thereby prevents the evolution of virus-driven chronic demyelination. This neuromodulatory framework highlights antiviral, rather than immunosuppressive, targets relevant to virally triggered MS-like pathology.

IISER
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Dhiraj Kumar

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Hijacking the messenger- host RNA splicing machinery as the target of *Mycobacterium tuberculosis.*

Mycobacterium tuberculosis (Mtb), the pathogen causing tuberculosis (TB) in humans, has evolved a larger repertoire of mechanisms that enable it to alter host immune responses, thereby facilitating infection and pathogenesis. Among many cellular processes regulated by Mtb, our observation on the alteration of host RNA splicing stands out. RNA splicing is fundamental to eukaryotic gene expression. Since changes in gene expression are the classical means of cellular response to any stress, including infections, the ability of the bacteria to establish a stranglehold on this process allows it to almost design the cellular responses in a manner that suits the pathogen. We have identified several bacterial virulence factors that help Mtb to execute its function of altering host RNA splicing. Our results, in addition to providing a novel mechanistic faceoff between the host and the pathogen, also provide clues on the possibility that the evolution of virulence in Mtb probably coincided with its ability to alter host RNA splicing.

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V. Stalin Raj

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Self-assembled nanoparticle decorated with spike protein elicits robust humoral responses against emerging coronaviruses.

Protein-based nanoparticles have emerged as versatile platforms for drug delivery and vaccine design. Among them, lumazine synthase (LS) is a bacterial protein that self-assembles into icosahedral nanocages (~15 nm in diameter) with sixty symmetric projections, resembling virus-like particles. This highly ordered structure makes LS an attractive scaffold for antigen presentation. Exploiting these properties, we genetically extended LS with domain B of protein A (pA), enabling binding to human IgG Fc-tagged spike S1 subunit of SARS-CoV-2, thereby forming a pA-LS-S1-hFc complex. The spike-decorated nanoparticles bound to the ACE2 receptor and displayed a characteristic speckle-like appearance. Biophysical analyses, including TEM, AFM, and DLS, revealed increases in both particle height and diameter compared with unconjugated pA-LS. Immunization of mice with the spike-decorated nanoparticles elicited robust humoral responses and neutralized pseudoviruses of SARS-CoV-2 wild type and variants, including Delta and Omicron. Following live virus challenge (Wuhan-Hu-1, Delta, or Omicron) in K18-hACE2 transgenic mice, immunized groups exhibited increased body mass, reduced lung viral loads, and significantly lower disease index scores. Furthermore, we expanded the applicability of this platform by generating mosaic nanoparticles decorated with S1-hFc proteins from two epidemic coronaviruses, SARS-CoV-1 and MERS-CoV. These mosaic nanoparticles bound their respective cellular receptors, ACE2 and DPP4, and showed receptor-specific co-localization. Immunization with the mosaic nanoparticles induced cross-reactive immune responses in mice, neutralizing both SARS-CoV-1 and MERS pseudoviruses. Together, these findings demonstrate the potential of LS nanoparticles to elicit robust immune responses and provide broad protective coverage against multiple pathogens through a single vaccine platform.

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Antifungal vaccine: A possible reality.

Systemic candidiasis is a mycosis caused by Candida species and inflicts ~1.2 million deaths annually worldwide. Despite its severity, an approved antifungal vaccine remains an unmet human need. Since every pathogens need to replicate in the host to survive and cause infections, DNA replication is a suitable target for designing drugs and vaccines. In a quest to design live whole cell vaccines, we have generated an array of DNA replication defective strains and their vaccine efficacies are being evaluated in pre-clinical models. Similar to a division of labor of the replicative DNA polymerases at the replication fork, the phenotypes and the ability to prevent reinfections upon immunization of leading and lagging strand DNA synthesizing polymerase defective strains are also diverged. Extensive repeated studies in mice revealed that Polδ defective strains induce robust protective immune responses to prevent fungal re-infections but not by the Polε defective attenuated strains. Among the virulence attenuated fungal strains evaluated so far, CNA25 is a promising candidate to fully develop as a pan-fungal vaccine. Our observations on these strains at the pre-clinical stages will be discussed.

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Trafficking: rules to make a healthy organism.

Cell fate determination in the early embryo and in embryonic stem cells are regulated by a number of mechanisms. Recent studies have shown that intracellular trafficking plays an important role in cell fate choice, with alterations in these resulting in developmental errors. It has been well established that several neurodevelopmental disorders such as intellectual disability (ID) and autism spectrum disorder (ASD) have genetic underpinnings. Recently, mutations in the CLTC gene coding for the clathrin heavy chain (CHC) protein have been associated with ID. However, there is limited information regarding the mechanism behind these mutant forms of the CHC protein in the context of ID.

We have developed an experimental model using Drosophila melanogaster to understand the molecular underpinnings of ID driven by two specific point mutations in the CLTC gene. Our results show that mutant forms of CLTC affect vesicle movement, synaptic maturation, brain morphology and behaviour in fruit flies.

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Targeting of Tripartite Neuron-Cancer-Immune Cell Cross-talk Activates the Tumour Microenvironment.

Tumour microenvironment (TME) is fundamental to cancer progression wherein dynamic interactions between cancer cells and stromal cells, including endothelial cells, fibroblasts, and immune cells, have been well-established. Recently, the presence of nerve fibres in TME has emerged as another crucial component that promotes cancer progression and metastasis. Innervating nerve fibres have been shown to engage in cross-talk with cancer and stromal cells, including immune cells. The release of neurotransmitters by nerve fibres, such as norepinephrine and acetylcholine, can directly activate the corresponding receptors, allowing cancer cell growth and acquisition of hallmarks. Conversely, cancer cells stimulate nerve growth into TME by either axonogenesis or neo-neurogenesis via the secretion of neurotrophic growth factors. This cross-talk between neurons and cancer cells directs towards the potential of anti- neurogenic agents for cancer therapy, and its local targeting can provide benefits in combination with chemotherapy, targeted therapy, and immunotherapy. In my poster/talk, I will present our recent efforts in utilising a local anaesthetic, Bupivacaine (BUP), to abrogate the engagement of cholinergic nerve fibres with TME through acetylcholine. Hydrogel-based localised delivery of BUP at the tumour site reduces the acetylcholine levels and slows the tumour growth. Further, scRNA sequencing analysis revealed that BUP-Gel remodels the immune microenvironment and primes macrophage and T-cell responses. Lastly, BUP-Gel synergises with localised chemotherapy, systemic chemotherapy, targeted therapy, and immunotherapy to inhibit tumour growth and potentiate anti-tumour immune response. Therefore, this study suggests targeting nerve-cancer- immune cell crosstalk to reprogram TME towards its anti-tumorigenic nature and ultimately inhibit the tumour progression.

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Investigating the molecular mechanism of limb tendon development - discovery of a possible tendon organizing centre.

The human musculoskeletal system is a remarkable example of biomechanical engineering, optimized for load-bearing and coordinated movement. Muscles generate force, which is transmitted to bones through tendons. The system's exceptional efficiency relies on seamless tissue transitions: muscle fibersintegrate with the collagen matrix of tendons, which anchor to the bones via the enthesis, a specialized region combining properties of cartilage and tendon. These transitions are critical for efficient load transfer. However, the cellular and molecular bases of enthesis development remains largely unexplored. The tendon development in the axial skeleton system provides a conceptual framework for the developmental basis of the organization of the musculoskeletal system. However, the fundamental difference between the appendicular and axial skeleton in terms of the tissue origins of muscle and cartilage provides a challenge.

Lately, our research group has been investigating aspects of appendicular tendon development. We have used mouse genetics to trace the lineage of limb tendons and classical chick embryology techniques to pinpoint the location of tendon organizing center in the limb bud. We have also discovered three novel genes involved in tendon biogenesis. I will present our latest data on this line of investigation highlighting aspects of appendicular tendon development.

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Forest-grassland mosaics: history, dynamics and an uncertain future.

Forest-grassland mosaics, characterized by abrupt boundaries between the two contrasting vegetation types, are an enigmatic and puzzling feature of many landscapes. Although traditionally believed to be artifacts of human activity, paleo-ecological evidence has revealed that many of these mosaics are in fact ancient ecosystems that predate human presence, often supporting unique biodiversity. They have been documented from diverse array of sites across the globe, ranging from the tropics to temperate regions. Their occurrence under these diverse climatic and biotic conditions has made it challenging to derive general theories for the mechanisms creating, structuring and maintaining these mosaics. Here, I discuss some of our ongoing work in one such forest-grassland mosaic, the iconic montane shola-grasslands of the Western Ghats: from their history, to the factors maintaining these mosaics, the conservation challenges they face, both currently and in the face of future climate change, and the implications of our results for a broader understanding of what structures these mosaics globally.

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Pathogen growth and virulence dynamics drive the host evolution against coinfections.

The occurrence of coinfections, where hosts are simultaneously infected by multiple pathogens, is widespread in nature and has significant negative impacts on global health. In humans, over one-sixth of the world's population is affected by coinfections, contributing to several diseases. However, despite the broad ecological relevance and impact on global health, most biomedical research has focused on understanding interactions between a single host and a single pathogen. The extent to which coinfections could impact host adaptation and immune system evolution, particularly in comparison to infections by single pathogens, thus remains largely unknown. Also, what roles do individual pathogen species play in this evolutionary process? To address these questions, in this study, we combined theoretical modeling and experimental validation in a model insect *Tribolium castaneum* evolving against two coinfecting bacterial pathogens with contrasting growth (e.g., fast- vs slow-growing) and virulence (fast- vs slow-killing) dynamics. Our findings show that fast-growing pathogens causing rapid mortality surges (i.e., fast-acting) can effectively limit the host's adaptive success against coinfections. While hosts rapidly evolved better survival against slow-growing bacteria causing long-lasting infections, adaptation against coinfections was significantly delayed and resembled the slow rate of adaptation against fast-acting pathogens. Finally, RNAseq analyses revealed that the observed delay in adaptation was associated with the limited scopes for suitable immune modulations against fast-acting pathogens. They might also be costly and pleiotropic (e.g., phenoloxidaseactivity), posing challenges for further immunomodulation and slowing adaptation. Our study thus highlights how individual pathogens' growth and virulence dynamics critically regulate adaptive responses against coinfections.

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Do elephants really address one another by name?

In a 2024 paper, Pardo et al. (Nature Ecology and Evolution 8: 1353-1364) tested the hypothesis that wild African elephants (Loxodonta africana) address each other using “name-like” calls – receiver-specific, non-imitative, arbitrary and learned vocal labels. The authors analyze a dataset of elephant recordings with a machine learning approach (i.e., Random Forest Models; RFMs), and show that an elephant’s call acoustics could be used to predict the targeted receiver more accurately than expected by random chance. The authors also used playback experiments to show that elephants responded more quickly and vocally to calls addressed to them vs. other elephants. Given the above results the authors conclude that elephants vocally label each other using “name-like” calls. While, being a hallmark study in terms of the novelty of the hypothesis being tested, I question the results of Pardo et al. Specifically, I demonstrate that the classification accuracy of the RFMs used to identify receivers based on acoustic data were severely overestimated. Indeed, using an extensive dataset of bird vocalizations, I show that arbitrarily assigned bird calls substantially outperform the observed elephant vocalizations in predicting the identity of the receiving elephant. This surprising result is driven by the extreme level of imbalance in the elephant dataset in conjunction with a misspecified null model that fails to control for caller identity and temporal autocorrelation. I also develop a simple theoretical model – based on an accepted fundamental driver of animal communication (i.e., acquisition of information), in conjunction with behavioralaspects (social structure and associative learning) common in elephant societies – that reveals that the use of arbitrary, receiver-specific “name-like” vocal labels by elephants are not necessarily required to explain the results of the playback experiment. Taken together my analyses reveal that there is limited evidence that elephants use arbitrary, receiver-specific “name-like” vocal labels to address each other.

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Minimal distortion of web vibrations facilitates collective prey capture in social Spiders.

The webs of social spiders are geometrically heterogeneous, asymmetric, and represent a costly extended phenotype essential for prey capture. In *Stegodyphus sarasinorum*, these collectively constructed webs comprise dense silk strands lacking obvious directionality. Here, the vibrational properties of such webs were studied using finite element analysis, followed by behavioural assays of prey capture using live prey and artificially generated, prey-mimicking vibrational signals. Image analysis revealed coordinated movement of group members towards the prey. Simulations of these collective movements reveal that prey-induced vibrations propagate through the web with minimal distortion, enabling rapid recruitment. Once the prey is surrounded, spiders exhibit near-field task partitioning, with some individuals attacking the extremities, others targeting the core body regions, while continual web repair occurs in the vicinity of the prey. Together, these results demonstrate that collective prey capture in S. sarasinorum is enabled by efficient transmission of vibrational information and near-field task partitioning which support persistence of colonies of this sit-and-wait predator.

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CTCF-mediated epigenetic control of splicing fuels hypoxia-induced EMT.

Tumor hypoxia reprograms chromatin and RNA processing to fuel EMT and metastasis in breast cancer, positioning epigenetic control of splicing as a key adaptive axis. Our study demonstrates that hypoxia upregulates CTCF expression through TET2-mediated DNA demethylation, which is crucial for its induction by HIF1α. This hypoxia-induced CTCF regulates the alternative splicing of genes involved in EMT, thereby advancing cancer progression. This talk will highlight how hypoxia elevates CTCF to induce PRMT5, establishing a histone H4R3me2s/H3R8me2s signature that links chromatin state to splice-site choice in EMT-relevant transcripts. Mechanistically, PRMT5-catalyzed dimethylation at a conserved intronic region between TCF3 exons I8a and I8b recruits DNMT3A and is read by MeCP2, enforcing RNA Pol II pausing and PTBP1 engagement to exclude exon I8a, thereby producing the pro-invasive E47 isoform under low oxygen. These findings place PRMT5 as a central chromatin–splicing integrator downstream of hypoxia-responsive CTCF, connecting epigenetic marks to isoform programs that potentiate EMT and invasion. Therapeutically, perturbing the HIF1α–CTCF–PRMT5– MeCP2–PTBP1 axis or leveraging targeted epigenome editing of upstream hypoxic circuitry offers a rational path to blunt metastatic progression in breast cancer.

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Biotechnology spin-off for clinical translation, manufacturing readiness, and pandemic preparedness.

This talk outlines the progress of a IISc-incubated biotechnology spin-off advancing novel recombinant protein subunit vaccines for influenza and respiratory syncytial virus (RSV), with emphasis on clinical translation, manufacturing readiness, and pandemic preparedness.

We describe a recombinant influenza vaccine platform manufactured using a baculovirus–Sf9 insect cell expression system. Our lead seasonal influenza vaccine has completed a Phase I first-in-human clinical study. The manufacturing process was designed to support rapid strain updates, scalable production, and reproducible product quality, with purification and formulation approaches compatible with standard cold-chain distribution and multi-site manufacturing. Preclinical evaluation demonstrated robust strain-matched functional antibody responses.

In parallel, we are advancing a next-generation influenza vaccine candidates incorporating conserved antigenic elements to support broader and more durable protection, with ongoing preclinical immunogenicity and functional characterization. In the ferret influenza model, vaccination was associated with strong serum antibody titers and reduced viral replication following a heterologous mismatched challenge, supporting translational relevance.

In addition, we are developing RSV vaccine candidates based on stabilized prefusion F protein antigens produced in CHO cells. Current efforts focus on structure-guided antigen design, expression optimization, and immunogenicity assessment to enable Phase I clinical development.

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Genomics-Enabled Utilization of Wild Wheat Relatives for Accelerated Wheat Improvement.

Wheat is one of the world's most important food crops, providing nearly 20% of the calories and protein consumed globally. Ensuring its resilience is therefore central to global food security under climate change. Bread wheat originated through two sequential interspecific hybridization events; however, domestication and modern breeding have drastically narrowed its genetic base, rendering modern cultivars increasingly vulnerable to biotic and abiotic stresses. To address this challenge, our research program has focused on systematically harnessing the untapped genetic diversity of wild wheat relatives for wheat improvement. We have assembled an extensive collection of over 1,500 accessions representing approximately 20 wild Triticum and Aegilops species and developed diverse pre-breeding resources.

These genetic resources have been comprehensively characterized using advanced genomics and high-throughput genotyping platforms. To date, more than 40 rust resistance genes have been successfully introgressed from wild relatives and mapped using SSR- and SNP-based approaches. Fine mapping of key resistance loci, such as Lr76–Yr70 from *Aegilops umbellulata*, was achieved through chromosome flow sorting and sequencing, while resistance gene enrichment sequencing (MapRenSeq) enabled precise localization of LrP–YrP and LrAp from Aegilops peregrina. Rapid gene discovery was further accelerated through bulked segregant analysis coupled with SNP arrays and whole-genome resequencing (BSA-Seq), allowing simultaneous mapping of leaf and stripe rust resistance genes from multiple wild species.

Genome-wide association studies in fully sequenced wild diversity panels have also led to the identification of novel loci governing resistance and adaptation including heat stress resilience and nitrogen-use efficiency, particularly from progenitor species. These advances provide powerful tools for the targeted introgression of beneficial alien genes with minimal linkage drag, accelerating wheat improvement. As a translational outcome, six improved wheat varieties have been developed with resistance genes and quality traits identified, transferred, and mapped from wild wheat relatives and are being cultivated in the farmers' field. These advancements will further drive the development of next-generation wheat varieties with enhanced productivity and adaptability to changing environmental conditions.

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Decoding Epigenetic and Structural DNA Signals in Plant Growth and Development.

DNA is often described as a simple string of genetic letters, but in living cells it behaves more like a dynamic and responsive material. Beyond its sequence, DNA can fold into different shapes and carry chemical marks that influence how genes are switched on or off. These structural and epigenetic features play a key role in controlling plant growth, development, and responses to the environment.

In this talk, I will introduce G-quadruplexes, special DNA shapes that can form at important regulatory regions of the genome. Proteins that recognize and resolve these structures help ensure that genes are expressed at the right place and time, particularly during plant development. I will discuss how such DNA structures contribute to root and seedling growth in the model plant Arabidopsis.

I will then broaden the discussion to crop plants, showing how changes in DNA packaging and chemical marks help plants cope with stresses such as drought and salinity. Using chickpea as an example, I will highlight how epigenetic regulation supports stress tolerance and productivity. Overall, this talk aims to show how plants use both DNA structure and epigenetic signals to adapt, survive, and grow.

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A Dynamic Vertex Model Reveals Coupling Between Contractility and Adhesion in Polarized Tissue Flow

During morphogenesis, a fertilized egg transforms into a complex multicellular structure through coordinated tissue flow and deformation driven by contractility, geometry, and adhesion. In Drosophila, Myosin II (MyoII) recruitment at curved regions initiates axis elongation via geometric coupling, but sustained polarized movement requires integration of MyoII-generated contractile forces with integrin-mediated adhesion and mechanochemical feedback. Although key molecular components have been identified experimentally, the mechanisms that couple these factors to maintain robust tissue flow remain unclear. To address this, we developed a dynamic vertex model that integrates experimental insights with theoretical mechanics, modelingadhesion and deadhesion as attachment and detachment forces. Our results reveal a novel mechanism in which MyoIIcontractility is mechanically coupled to integrin-based adhesion to drive sustained polarized tissue flow.

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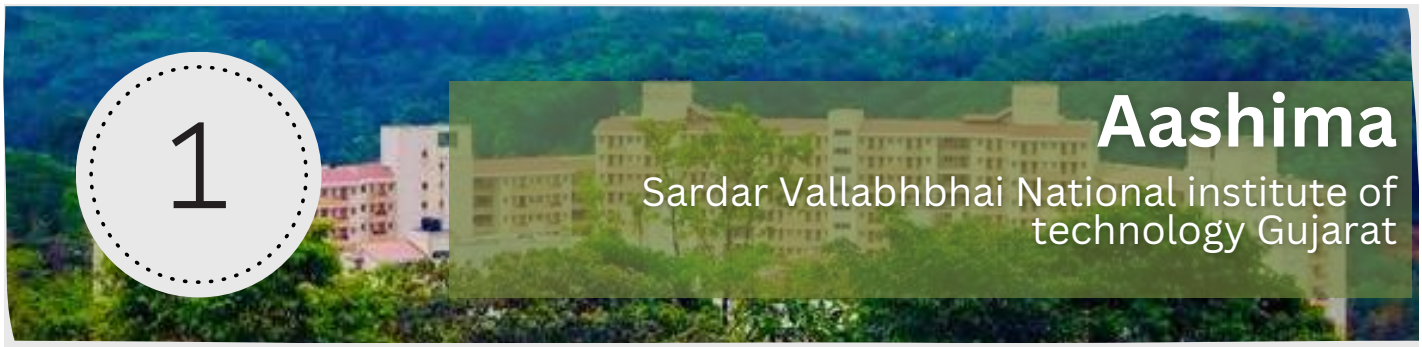

Metal organic framework -polymeric hybrid Diabetic wound healing Patch for improved Transdermal Co-Delivery of Curcumin and Heparin Drugs

In order to effectively manage infections without causing adverse effects, modern healthcare engineering requires wound dressings with strong mechanical strength and good biocompatibility that allow for prolonged codrug delivery for acute injuries. Curcumin and heparin were encapsulated within the pores of the biocompatible metal–organic framework, zinc l-glutamate (Zn-GA), a well-known bio-MOF, along with two biocompatible polymers—poly (vinyl alcohol) (PVA) and poly (ethylene glycol) (PEG)—in an 8:2 ratio to create a novel cryo patch. In comparison to current wound dressings, this cryo patch demonstrated a number of superior qualities, such as exceptional mechanical durability (13.8 N), an improved water swelling ratio (231.1%), antifreezing ability (-46 °C), adhesiveness, self-healing capabilities, and a significant drug-loading capacity (roughly 45,000 times more curcumin than water). Additionally, it showed hemocompatibility (hemolysis levels below 2% even at high concentrations of up to 40 mg/mL), prolonged drug release, and potent antioxidant, antifungal, and antibacterial properties.HaCaT cells demonstrated outstanding cell viability in in vitro experiments, and the curcumin/heparin@patch markedly accelerated wound closure in scratch tests. After 48 hours, the area occupied by migratory cells rose 1.5 and 1.6 times, respectively, in comparison to the control and drug-free patch, at a low dose of 250 μg/mL. The cryo patch also showed possible anti-inflammatory qualities. These results demonstrate how the cryo patch is a promising option for advanced wound dressing applications due to its exceptional combination of high drug-loading capacity, improved wound healing at low drug concentrations, and biocompatibility.

Keywords: Cryo Patch , transdermal drug delivery,wound healing

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2

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Cortical Dynamics of Disengaged Awareness: Dissociation Between Global Alpha Suppression and Frontal Asymmetry in Adolescents

Background: Adolescent neurodevelopment is characterized by the maturation of prefrontal inhibitory circuits alongside heightened emotional reactivity. In neural systems, alpha-band oscillations (8–13 Hz) regulate thalamocortical gating, where power suppression reflects a release of inhibition and increased cortical excitability. Simultaneously, lateralized alpha activity, specifically Frontal Alpha Asymmetry (FAA), serves as a biomarker of emotional valence, distinguishing approach- and withdrawal-related motivation. Typically, states of heightened arousal in adolescents are closely coupled with valence-driven emotional polarization.

Objective: This study investigated the neurophysiological effects of Nadanusandhana meditation, a targeted focused-attention protocol, to examine whether high cortical activation can be functionally uncoupled from the emotional lateralization shifts commonly associated with arousal.

Methods: Thirty meditation-naïve, right-handed adolescents (aged 12–18) participated in a 21-session intervention focused on sustained attention to internal perceptual stimuli. Resting-state EEG data were acquired pre- and post-intervention using a 14-channel system at a 128 Hz sampling rate. Signal processing included band-pass filtering (1–40 Hz) and artifact removal. Global Alpha Power (GAP) was computed as an index of general cortical engagement, and FAA ($\ln F4 - \ln F3$) was calculated to assess hemispheric dominance.

Results: Paired-samples t-tests revealed a significant post-intervention reduction in GAP ($p < 0.001$; Cohen's $d = 1.02$), indicating a substantial decrease in alpha-mediated cortical inhibition and increased alertness. Notably, despite this pronounced state change, FAA remained stable ($p = 0.585$), showing no significant directional shift in hemispheric dominance. Topographic mapping confirmed a uniform, bilateral reduction in alpha power rather than a lateralized redistribution.

Conclusion: These findings demonstrate a distinctive neurophysiological profile characterized by heightened cortical engagement without concurrent emotional lateralization, which we term "aroused equanimity." The protocol selectively engaged attentional networks without recruiting valence-biased neural circuits. This suggests a potential neuroplastic mechanism for training sustained attention in the developing adolescent brain while minimizing emotional reactivity.

Keywords: Alpha oscillations; Frontal alpha asymmetry; EEG

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3

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Chronobiology Lab

Understanding the role of micro-RNA in regulation of circadian rhythm in *Drosophila melanogaster*

Earth's 24-hour rotation subjects organisms to rhythmic changes in light and temperature, driving the evolution of endogenous circadian clocks. These clocks, governed by genetic and molecular oscillators, sustain ~24-hour cycles even under constant conditions, enabling organisms to anticipate daily environmental fluctuations. In *Drosophila melanogaster*, circadian rhythms are regulated by specialized pacemaker neurons, where the internal clock operates via a transcriptional-translational feedback loop. Notably, some clock protein levels peak hours after their corresponding mRNA levels, indicating possible post-transcriptional regulation. micro-RNAs (miRNAs), small non-coding RNAs, modulate gene expression by binding to target mRNAs, leading to degradation or translational inhibition and have been implicated in the regulation of circadian rhythms in *Drosophila*.

Building on previous findings from our lab showing that robustly expressed miRNAs in clock neurons impact the circadian clock, our present study focuses on a genetic screen of 15 less - abundant miRNAs expressed in circadian clock neurons, and conserved between humans and *Drosophila*. We assessed the effects of downregulating each miRNA in clock neurons, evaluating circadian properties such as phase, robustness, and free-running period (FRP).

Our initial experiments focused on characterizing the role of miR-9b by examining its expression under downregulation (miR-9b sponge; miR-9b SP) conditions. Downregulation of miR-9b using circadian cell specific GAL4 drivers resulted in a phase advance of the morning locomotor activity peak, accompanied by altered amplitude of locomotor rhythms and changes in the free-running period (FRP) under constant darkness (DD). These results highlight the regulatory role of miR-9b in modulating circadian outputs, emphasizing its contribution to the post-transcriptional control of the *Drosophila* circadian clock.

Keywords: Circadian rhythm, Drosophila, miRNA

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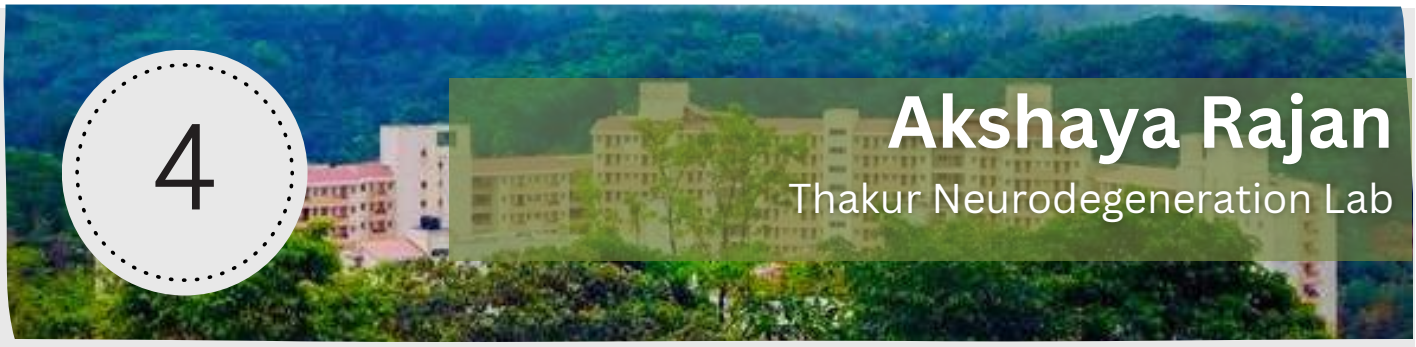

4

Akshaya Rajan

Thakur Neurodegeneration Lab

Diabetes-induced peripheral dysregulation exacerbates parkinson's disease pathology in mice

Growing evidence indicates a strong association between the metabolic disorder type 2 diabetes mellitus (T2DM) and the neurodegenerative movement disorder Parkinson's disease (PD). Despite strong epidemiological links between T2D and PD, the mechanistic pathways by which chronic metabolic dysfunction drives neurodegenerative processes in PD remain poorly understood.

In this study, we investigated whether chronic diabetic alterations predispose to or exacerbate PD-related pathology using a combined T2DM–PD mouse model. T2DM was induced in C57Bl/6 mice by a high-carbohydrate, high-fat diet, followed by intranigral injection of α-synuclein (α-Syn) preformed fibrils to establish a progressive PD phenotype. Non-diabetic groups received a control diet. Dietary intervention reliably induced a diabetic phenotype, as confirmed by glucose and insulin tolerance tests.

The diabetic group also had higher plasma cholesterol levels than the control group. PD-associated phenotypes, such as loss of grip strength and depression, were exacerbated in the T2DM-PD group. Despite similar dopaminergic neurodegeneration between PD and T2D-PD groups, neuroinflammation was markedly increased in T2DM-PD group compared to the non-diabetic control. Additionally, PD groups displayed a trend towards increased neutral lipid deposition in the substantia nigra. The detection of aggregated α-Syn in the liver of PD groups, together with enhanced lipid droplet accumulation in T2DM groups, supports a systemic contribution to PD pathogenesis.

Collectively, these findings suggest that peripheral metabolic dysfunction plays a critical role in modulating neurodegenerative processes and provide mechanistic insight into the interplay between T2D and PD. Given the rapidly rising prevalence of diabetes, particularly in India, elucidating this relationship will facilitate the identification and development of targeted interventions capable of preventing or delaying the onset of PD in the high-risk diabetic population.

Keywords: Parkinson's disease, Type2 diabetes, inflammation

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5

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Host-Pathogen Interaction Lab

Investigating lysosomal biology and inter-organelle cross-talk during infection with lysosome-adapted pathogen, *C. burnetii*

Several diseases of public health importance are caused by intracellular pathogens, including *Legionella, Mycobacterium, Salmonella, Coxiella,* and others. One such pathogen that has been our focus is *Coxiella burnetii*, the causative agent of zoonotic disease, Q fever, that replicates in a low pH, lysosome-derived compartment called the *Coxiella*-containing vacuole (CCV). *C. burnetii* vacuole is highly fusogenic and fuses with host vesicles (lysosomes, autophagosomes), thereby expanding in size and accommodating intracellular replication. *Coxiella burnetii* also expresses a Type IVB secretion system to translocate effector proteins into host cells, for biogenesis and maintenance of a persistent, prolonged infection. We hypothesize that this obligate intracellular lifestyle would dysregulate lysosomal functions and host cell vesicle fusion machinery in cells infected with *C. burnetii*, making it an intriguing model for investigating disrupted host vesicle traffic and organelle biology, with an emphasis on exocytosis.

Using fluorescence microscopy-based approaches, we observed elevated exocytosis in macrophages as indicated by increased detection of sLAMPI. Additionally, more vesicles were released by infected cells as compared to uninfected cells. Exocytosis is regulated by master transcription factor Transcription Factor EB (TFEB) and lysosomal divalent cation channel TRPMLI. TRPMLI activation results in the nuclear localization of the transcription factor EB (TFEB), one of its downstream effects being the activation of exocystosis. Using GFP-expressing strains of *C. burnetii* and fluorescence to report on the status of intracellular bacterial replication, we assessed the effect of altering exocytosis by small molecules that modulate TRPMLI activity on intracellular bacterial replication. The intricate roles of TFEB and TRPMLI in lysosomal biology are well established; however, recent studies have uncovered their functions in the cross-talk between lysosomes and other organelles. This led us to probe the cross-talk of CCV with other organelles to investigate their effect on infection.

Using live-cell confocal microscopy, we have been currently focused on cross-talk of the lysosome-derived CCV with other intracellular organelles and implications on bacterial replication. Our efforts are directed towards employing a multi-omic approach incorporating microscopy, flow cytometry, and transcriptomics/proteomics to further evaluate how infection with lysosome-adapted pathogens alters host exocytosis and exploits inter-organelle cross-talk, and to assess their relevance in host-pathogen interaction.

Keywords: Intracellular pathogen, Mitochondrial dysfunction, Organelle Biology

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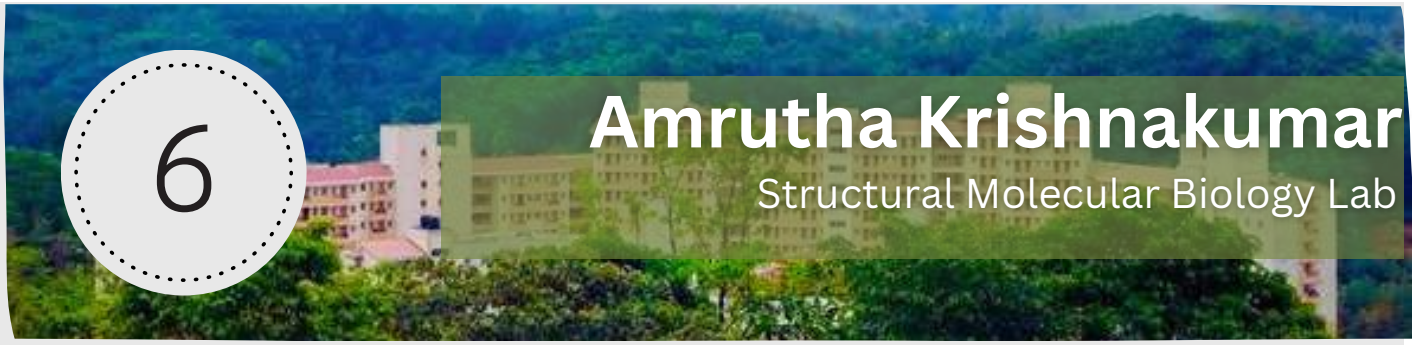

6

Amrutha Krishnakumar

Structural Molecular Biology Lab

Structural and functional studies of Mycobacterial transcription regulators-the Gre factors

Tuberculosis still remains as one of the deadliest diseases which affect people across the globe with millions of deaths annually. *Mycobacterium tuberculosis* (Mtb) is the causative agent of tuberculosis. Transcriptional pausing and backtracking by RNA polymerase (RNAP) are key regulatory mechanisms in gene expression of many bacteria including *Mycobacterium tuberculosis*. Gre factors are transcriptional regulators that rescue RNA polymerase from backtracked stalled states by enhancing its intrinsic endonucleolytic cleavage activity. It plays a crucial role for the removal of the aberrant RNA 3' transcript and resume polymerization of RNA. This study focuses on the structural and functional characterization of Gre factor from *Mycobacterium tuberculosis* (MtbGre). We have overexpressed and purified the protein and biophysically characterized it. Circular Dichroism spectroscopy studies showed proper secondary structural folding of the MtbGre. We further are exploring the possibility to target MtbGre. Virtual screening and molecular docking identified several potential candidates from the ChEMBL antibiotic library. *In vitro* validation using ITC and fluorescence assays were carried out with the top hit compounds. Intrinsic tyrosine fluorescence assays confirmed ligand-induced quenching, very notably for valganciclovir. The Mtb RNAP core complex was overexpressed and purified through affinity and gel filtration chromatography techniques. The RNAP- MtbGre complex was reconstituted in vitro using a synthetic transcription bubble, and interactions were confirmed through EMSA and ITC studies. Negative staining transmission electron microscopy (TEM) provided 2D class averages of the complex (RNAP–bubble– MtbGre). Efforts toward -ve stain 3D reconstruction and cryoEM data collection are ongoing. These findings lay the groundwork for future high-resolution structural studies and functional characterization of MtbGre. The poster presentation will describe the work progress.

Keywords: Transcription, Gre factor, Negative staining

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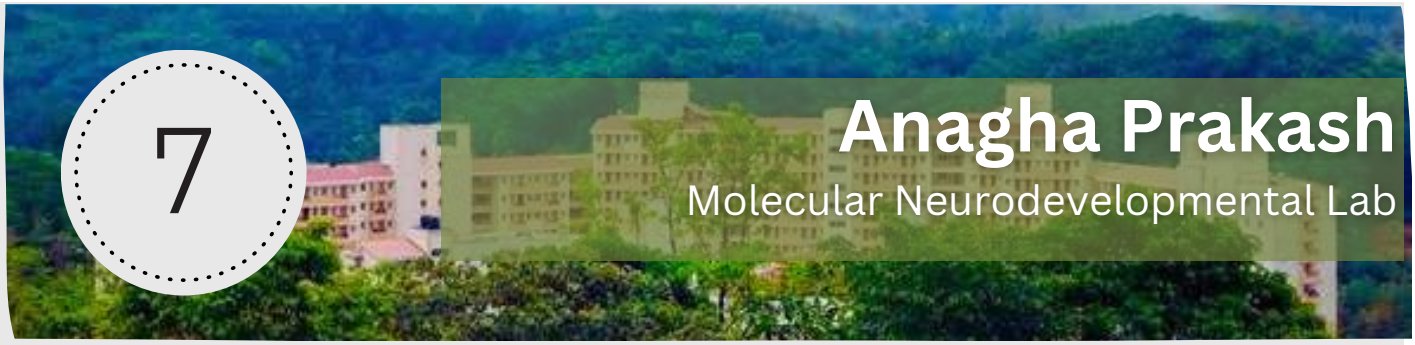


7

Anagha Prakash

Molecular Neurodevelopmental Lab

Sleep Shapes Seizures, Seizures Shape Sleep

Epidemiological studies indicate associations between epilepsy and sleep, yet the mechanisms behind this connection are unclear. Studying the effects of sleep on seizure susceptibility could further our understanding about fundamental mechanisms driving epilepsy and seizure generation. Here, we study the interaction between sleep, circadian regulation, and seizure susceptibility at the molecular, network, and systems levels using larval zebrafish. We use behavioural and molecular assays to understand how drug-induced seizures affect sleep, as well as how circadian disruption affects seizure susceptibility. Our findings show that chronic seizure induction reduces daytime sleep and disrupts clock-gene phasic expression. In turn, sleep disruption by subjecting zebrafish larvae to constant light conditions leads to paradoxically lowered seizure susceptibility. These findings demonstrate that circadian clock disruption alters seizure vulnerability and the occurence of seizures leads to alterations in sleep.

Keywords: Seizure, Sleep, Zebrafish

IISER THIRUVANANTHAPURAM

8

Anju K N
Chromatin Biology Lab

Functional Consequence Of RAD21 Mutation On Cohesin Mediated DNA Damage Repair

Chromatin architecture is the three-dimensional organization of the DNA and the accessory proteins that compactly pack the two-meter-long DNA into the microscopic nucleus. This hierarchical organization of the interphase chromatin enables the compact and efficient packing of the genome while conserving functional accessibility to different polymerases, transcription factors, and other proteins to facilitate gene regulation as well as structural stability. At the most basic level, each chromosome occupies a distinct chromosomal territory, which is a spatial domain within the nucleus that reduces the intermingling of genetic information and affects genomic function. The chromatin within the chromosome territories segregates into a transcriptionally active compartment A and an inactive B compartments. Moving to a finer scale, chromosomes are further partitioned into topologically associating domains (TADs) that are self-interacting regions with boundaries marked by the architectural proteins CTCF and cohesin. The DNA sequences inside the TADs tend to interact frequently with each other than with neighbouring sequences outside the domain. At the most fundamental level, DNA wraps around the histone proteins to form nucleosomes, which resemble a 'beads on a string' conformation. Understanding the chromatin organization at the most fundamental level is important, as it can greatly influence our understanding of the disruption of cellular homeostasis seen in cancers, neurodegenerative diseases, and genetic disorders.

Cohesin is a ring-shaped multi-subunit protein complex and plays important roles in the chromatin architecture by extruding chromatin loops in an ATP-dependent manner, thereby organizing the genome into loops and TADs, facilitating precise enhancer promoter communication, and maintaining genome stability. The core subunits of cohesin, which forms the ring, are SMC1, SMC3, RAD21 and STAG1/STAG2. Being a critical protein of the cohesin complex, mutations in RAD21 disrupts the cohesin ability to perform the different functions during various phases of the cell cycle leading to chromosomal instability. RAD21 mutations leading to various cancers and neurological disorders like Cornelia de Lange syndrome (CdLS) have been reported. In addition to this function, cohesin plays important roles in DNA damage repair.

The project focuses on studying the effect of a specific RAD21 mutation on the DNA damage break repair. Although this mutation is characterized as a pathogenic mutation the molecular implications of this mutation have not been characterized. To assess the damage repair kinetics, we aim to induce equal amounts of DNA double strand damage in both the mutant and wild-type RAD21 incorporated cell lines and study the damage repair efficiency and kinetics by using various assays.

Keywords: Cohesin, DNA damage repair, chromatin architecture

IISER THIRUVANANTHAPURAM

9

Anuraag Nallan Chakravarthi

The Mutations Lab

Replication-transcription collisions influence gene expression in bacteria

Replication-transcription collision (RTC) occurs when the replisome collides with the transcription machinery as they traverse the same DNA template in the same direction on the leading strand (co-directional) or opposite direction on the lagging strand (head-on). Most studies have focussed on the aftereffects of collisions on replication, while very few suggest the possible effects on gene expression. Some of the proposed and known effects of collisions on gene expression are RNA polymerase dislodgement (*French S, 1992*) and possible premature transcription termination (Rocha EP and Danchin A, 2003). Although collisions are detrimental, head-on collisions specifically lead to severe problems such as slowing down of transcription (*Liu B and Alberts BM, 1995*) and increased mutagenesis at the promoter (*Sankar et al, 2016*). Here, we investigated the effects of RTC on gene expression at single cell level. We developed a single-cell gfp reporter system, where the reporter is chromosomally inserted in either the co-directional or head-on orientation in *B. subtilis*. We observed an orientation-specific variation in gene expression levels at varying degrees of transcriptional and translational strengths, revealing the influence of collisions on gene expression in a DNA strand-specific manner. We propose that the nature of RTCs likely moderates gene expression in bacteria.

Keywords: Collisions, Gene expression and Single-cell

IISER THIRUVANANTHAPURAM

10

Anuraag Srinath A
CLUE Lab

Evolution of Defense Systems

Bacteria and their viruses have cohabited the Earth for billions of years. Although it is not exactly known how viruses originated, it is speculated that even the Last Universal Common Ancestor, which inhabited the Earth had an entire virome, from which other viruses have also been believed to have descended.[I], [2] This has led to several billion years of an arms race to overpower the other. But viruses being parasites, need the host replicate, and therefore have in turn incentivized their infection with metabolic genes, virulence genes and commonly superinfection immunity.[3]

Bacteria have now an extremely diverse repertoire of defense genes from the ones that independently emerged, thanks to a lot of interactions with mobile genetic elements through horizontal gene transfer. This process allowed for selection to act on such variants with a huge fitness advantage in a single generation dramatically.

Some phages may sabotage its competitors by entering a lysogenic infection phase and provide bacteria protection. This is now not a simple phage and bacteria versus, but a battle of fitness. We would like to uncover the strategies that these coevolving species would take and identify the main driver of this evolution through some experiments.

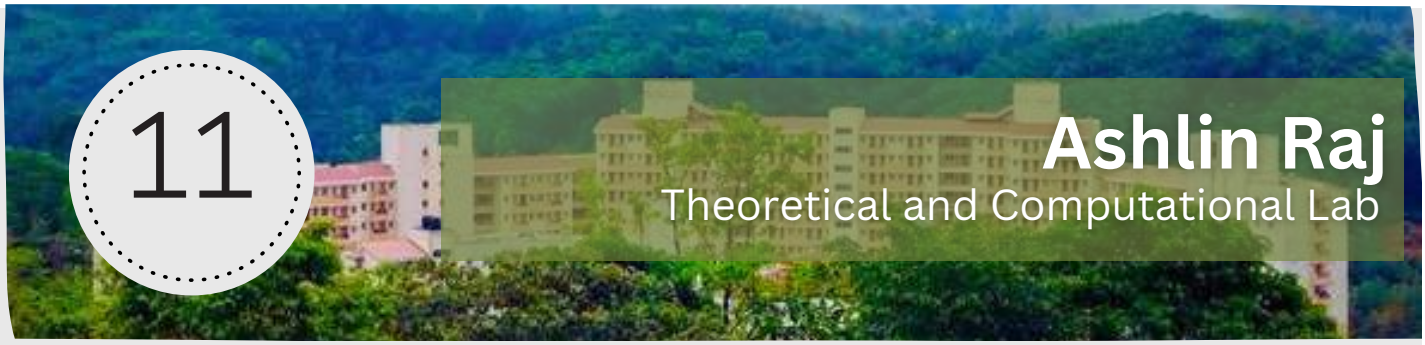
Furthermore, a concern is for sustainable therapy. Thus far, phage therapy has been depicted to hold so much promise that it could greatly reduce or even eliminate the death toll of the AMR pandemic. However, knowing that a similar hype and use of antibiotics led to such a global situation, it becomes of utmost importance to test phage therapy for its potential.

Here, we perform an experiment of coevolution of a lytic phage with 4 bacterial genotypes, where 2 are devoid of defense systems. Our objective is to understand how quickly a bacterial population can step up in the fight against their phages and to also check differences in evolvability if some were handicapped in this fight.

Additionally, we test if a genotype with a growth handicap has disadvantages against their viruses.

Keywords: Defense Systems, Evolutionary Repair, Coevolution

IISER THIRUVANANTHAPURAM



11

Ashlin Raj
Theoretical and Computational Lab

Where Cells Decide to Divide: The Dynamics of the Preprophase Band

Regeneration is the process by which organisms restore lost or damaged structures through coordinated biological mechanisms involving molecular signalling, localized cell growth, and division. In plants, regeneration critically depends on precise cell division, as new cells must be generated in specific regions to enable the regrowth of lost tissues. Successful division requires accurate specification of the cell division plane, which determines the orientation and position of the newly formed cell wall. This spatial information is transiently established by the rearrangement of cortical microtubules (CMTs) into the preprophase band (PPB), a narrow ring of microtubules that predicts the future division site. PPB formation arises from the dynamic reorganization of the CMT array driven by microtubule nucleation and catastrophe. In this work, we investigate the role of microtubule dynamics in PPB formation during plant regeneration.

IISER
THIRUVANANTHAPURAM

12

Dea Vincent
Mutations Lab

Replication-dependent origin of genome organization in bacteria

In bacteria, there is no spatiotemporal separation between replication and transcription as they both act on the same DNA template, with replication being almost 20 times faster than transcription. Hence, collisions between these machinery inevitably occur [1]. Depending on the strand of replication on which a gene is encoded, leading or lagging, collisions can be either co-directional or head-on. Several studies have shown that such collisions are detrimental to the cell as they can impede replication fork progression, interrupt transcription, and increase genomic instability, with head-on collisions being more deleterious than co-directional [2]. Consequently, bacteria have evolved to preferentially encode genes on the leading strand. This is referred to as gene-strand bias (GSB) [3]. Studies have associated the nature of the replication machinery, particularly the presence or absence of the catalytic alpha subunit PolC as in the replisome, with GSB [4]. In this study, we employed maximum likelihood and ancestral state reconstruction to investigate the coevolution of GSB with PolC. Furthermore, we also employed a hidden Markov model-based approach to analyze the various domain architectures of PolC and their correlation with GSB. Our results reveal a likely coevolution of PolC and GSB across species, and a link between the structural variability of PolC and GSB.

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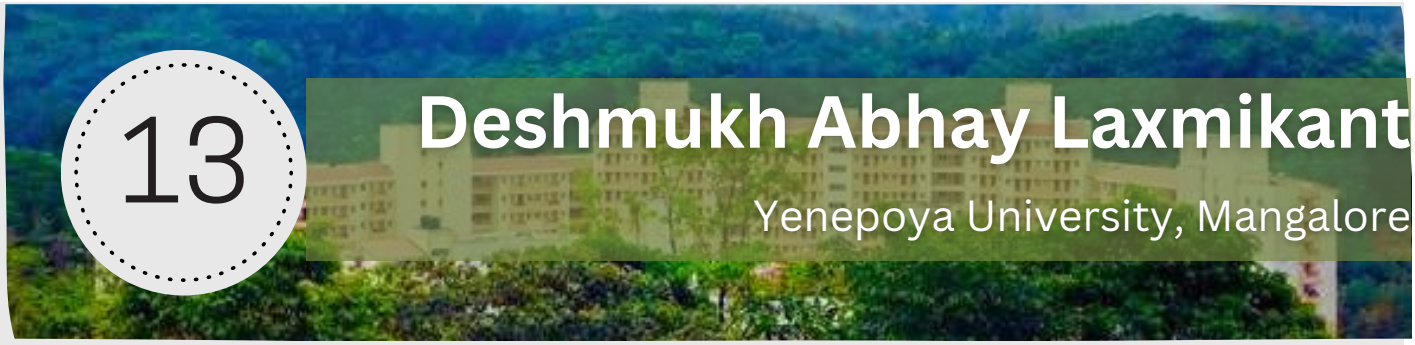
Rocha, E. P. (2002). Is there a role for replication fork asymmetry in the distribution of genes in bacterial genomes?. Trends in microbiology, 10(9), 393-395.

Keywords: Replication-transcription conflicts, Genome organization, Evolution

IISER THIRUVANANTHAPURAM



13

Deshmukh Abhay Laxmikant

Yenepoya University, Mangalore

Dual-Drug Loaded Hydrogel Scaffolds Integrated with Mesenchymal Stem Cells for Enhanced Thermal Burn Wound Regeneration

Background: Thermal burns constitute a significant global health concern. Chronic burn wounds manifest as complex clinical challenges, characterized by elevated infection risks, sustained inflammation, and hindered tissue regeneration. Conventional dressings frequently do not adequately address these multifaceted issues concurrently. This study delineates the development of a multifunctional, bioactive hydrogel system engineered to provide a supportive microenvironment for cellular therapy.

***Objective*:** The objective of this study was to develop a Pluronic FI27-based hydrogel designed for the delivery of fucoidan and ciprofloxacin, while preserving the viability and regenerative capacity of encapsulated mesenchymal stem cells, with the aim of improving burn wound healing.

***Methods*:** We synthesized a Pluronic FI27-based hydrogel via physical mixing and characterized for its viscosity, gelation time, and degradation profile. Further, we encapsulated Mesenchymal stem cells (MSC) within the matrix, studied the viability, proliferation of MSCs using trypan blue staining, and morphology using phase-contrast microscopy. Live-dead cell staining of MSC upon hydrogel encapsulation was carried out using Acridine orange and ethidium bromide double staining. We also assessed the Mitochondrial stability of the MSCs in the hydrogel microenvironment using the MitoTracker assay.

***Results*:** The optimized hydrogel system exhibited optimal viscosity and gelation time, suggesting its use for topical skin application. Further, the degradation kinetics showed significant degradation of the composites over the 5 days, confirming the natural degradation upon topical application. Encapsulated cells remained viable for 5 days, demonstrating biocompatibility of the MSCs with the hydrogel after encapsulation. Also, the MSCs exhibited cell migration inside the hydrogel microenvironment, thereby demonstrating the ability of MSCs to migrate to the wound site for effective integration.

***Conclusion*:** In conclusion, we successfully synthesized a dual-drug-loaded, cell-encapsulated hydrogel system and validated the system in vitro for a synergistic approach to burn care. By addressing both the biochemical and cellular requirements of the wound bed, this formulation represents a promising platform for advancing regenerative medicine for skin regeneration.

Keywords: Hydrogel, Cell therapy, Burn wound

IISER THIRUVANANTHAPURAM

14

Dhanagovind P T
Stem Cell Biology Lab

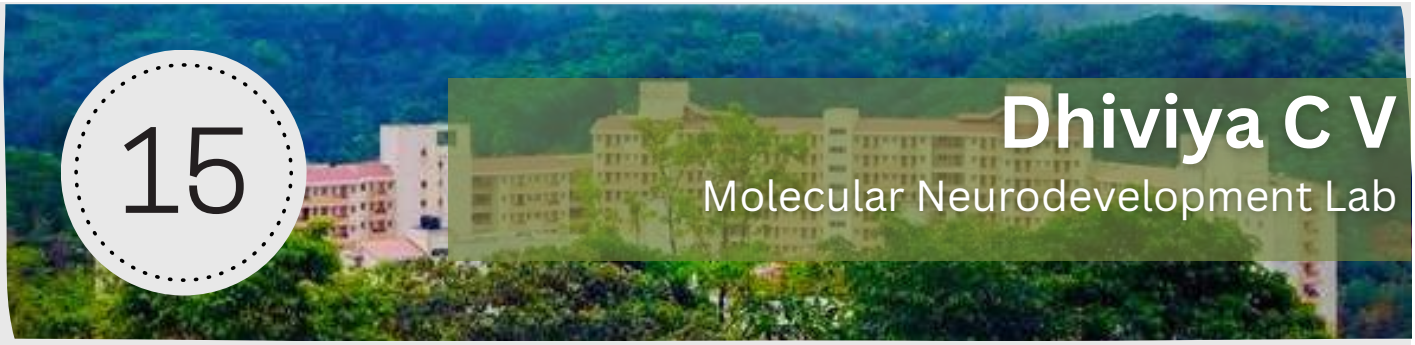
Oxygen sensing pathway in the maintenance of adult neural stem cell pool

Neurogenesis is a well-orchestrated process by which the cells of the central nervous system are generated. Postnatally, there are two major neurogenic niches in the mammalian brain – the Subgranular Zone (SGZ) and the Subventricular Zone (SVZ), both considered to be hypoxic. Disruption of the hypoxia pathway through loss-of-function studies have previously been shown to result in defective neural stem cell (NSC) maintenance and also decline in neurogenesis. HIF-Iα is one of the primary mediators of hypoxia signaling, whose activity is tightly regulated by Prolyl hydroxylase domain (PHD) enzymes, integral players in oxygen sensing pathway. We sought to examine the effect of HIF-Iα stabilization by disrupting the oxygen sensing pathway. Long-term deletion of the PHD proteins I and 3 (*Phd1*$^{-/-}$; *Phd3*$^{-/-}$, dKO) led to enhanced activation of hypoxic signaling. In contrast to what we anticipated based on prevalent notion, we observed a substantial loss of proliferative activity of NSCs in the SGZ. The NSC population displayed dramatic changes with reduced total pool size accompanied by pronounced alterations in their morphology with shortening and arborization of radially-oriented fibers. Moreover, we observed a marked decrease in proliferative neural progenitors, evidenced by the reduced number of DCX+ newborn neurons. Contrarily, there was a significant increase in the number of astrocytes, indicating a predisposition of the NSC population towards astroglial differentiation at the expense of neurogenesis.

Consistent with in vivo phenotype, pharmacological stabilizing of HIF-Iα in Embryonic day I4.5 (EI4.5) neural stem/progenitor cell (NSPC) cultures led to reduced proliferation and astrogliogenesis at the expense of neurogenesis. However, competitive inhibition of LDHA partially revoked the compromised proliferation. Subsequent scRNA-seq analysis of dentate gyrus (DG) revealed higher oxidative phosphorylation function in quiescent NSCs relative to other neural lineage populations. Concurrently, HIF-Iα stabilization was associated with increased mitochondrial dysfunction potentially leading to deterioration of NSC maintenance. Collectively, these findings delineate the critical role of oxygen-sensing pathway and metabolic regulations governing the fate decisions of the adult NSC population.

Keywords: HIF-Iα stabilization. Adult neurogenesis. Metabolic regulation of NSC fate

IISER THIRUVANANTHAPURAM



15

Dhiviya C V
Molecular Neurodevelopment Lab

Immune–Circadian Crosstalk: A Novel Role for Complement Factors in Sleep

Animal behaviour is influenced by various factors and genes, and the products they encode play a crucial role in orchestrating behaviour. Complement cascade proteins, in addition to their canonical immune function, play non-canonical roles in synaptic pruning during neurodevelopment, neural progenitor proliferation and regulation of emotional and cognitive behaviours. More recently, some complement factors have been found to play a role in determining stress resilience. However, we lack a comprehensive understanding of their expression in the developing brain and their role in neurodevelopment and the establishment of behaviours.

Using RNA sequencing data from a zebrafish mutant of a key complement factor, we observed that several genes regulating circadian rhythm and neurodevelopment are downregulated without this factor. Zebrafish show robust diurnal sleep–wake cycles and clock gene expression early in life, stabilising by about 4–5 days post-fertilisation. Light-entrainable pacemakers, the pineal gland and retina maintain circadian rhythm in zebrafish. We found the changes in the expression of circadian genes translated into increased daytime activity in homozygous mutants compared to their wild-type siblings. Concurrently, the mutants showed decreased daytime sleep, where they had similar number of sleep bouts, but each bout was of shorter duration. These observations point towards complement factors playing a previously unknown role in the maintenance of circadian rhythm and sleep. Our current work focuses on understanding the mechanisms by which deficits in the complement system affect neurodevelopment and circadian rhythms.

Keywords: Sleep, Neurodevelopment, Complement factor

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16

Eeshani Abhyankar &
Anushka Tyagi
Laboratory of Immune Cell Biology (LICB)

Investigations into CARPs-mediated modifications of their substrates

Organisms have evolved a variety of complex mechanisms to preserve cellular homeostasis. Stress conditions can lead to protein misfolding and aggregation, rendering proteins inactive and potentially toxic to cells. Heat shock proteins constitute a family of molecular chaperones that coordinate the refolding and degradation of misfolded proteins, thereby maintaining proteostasis. Dysregulation of these protein expressions has been associated with numerous pathological conditions, including neurodegenerative diseases, such as Alzheimer's and Parkinson's, as well as various cancers. In addition, CHIP (C terminus of HSC70-Interacting Protein), also known as STUBI, is a master regulator of protein quality control, and its pathogenic mutants are associated with Spinocerebellar Ataxia. Post-translational modifications play a critical role in regulating the activity and turnover of these proteins. In our lab, we investigate the roles of ubiquitination and SUMOylation in regulating the heat shock proteins HSP70 and HSP90, and STUBI. We show that CARPs (Caspase-8/10 Associated Ring Proteins), already reported to ubiquitinate STUBI, also ubiquitinate HSP70 and HSP90 in a ubiquitin ligase-dependent manner. Interestingly, we also explore HSP70, HSP90 and STUBI as potential substrates of CARPs-mediated SUMOylation. In doing so, we seek to determine whether CARPs act as mediators of the ubiquitination-SUMOylation crosstalk.

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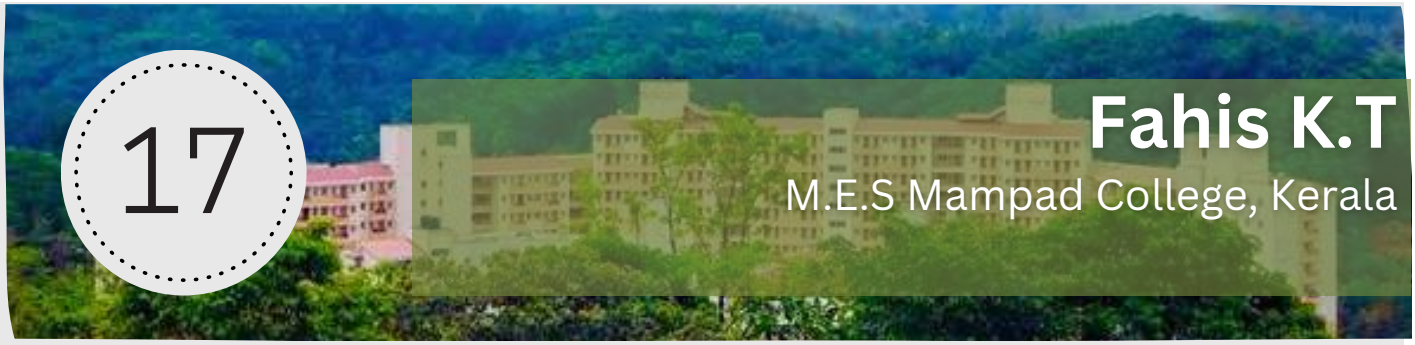

17

Fahis K.T
M.E.S Mampad College, Kerala

How do aquatic invertebrate assemblages vary in dendrotelms of the Evergreen Rainforests of the Western Ghats?

Dendrotelms are ephemeral water bodies that provide unique microhabitats that support diverse organisms, ranging from bacteria and protists to invertebrates and higher-order taxa (Kitching, 2000). Widely occurring in both temperate and tropical forests, together with other phytotelmata, they constitute a significant yet understudied component of global aquatic ecosystems (Carpenter, 1982). Despite their ubiquity, the diversity of aquatic invertebrates inhabiting water-filled tree holes in the tropical forests of Asia remains poorly documented. This study highlights the abundance of different taxa in 46 dendrotelms across four sampling sites in the Western Ghats. Water-filled tree holes were identified and sampled using a syringe, sucking tube, dip spoon, and trays, and the coordinates of the sampling sites were recorded using GPS devices in the field. The samples were then stored in separate bottles and transported to the laboratory. Sorting and preliminary identification were performed using an Olympus trinocular microscope. Data were analyzed using different diversity indices. The Shannon index revealed that Sairandhri had a comparatively higher diversity (0.695), and the Walakkad Region had low diversity measures (0.325) among the study sites. Despite the higher abundance, more dominance was observed in the Sispara region. Simultaneously, the dendrotelms of the Kakkadampoyil region have more evenly distributed fauna. Scirtidae and Culicidae were the most dominant taxa in the aquatic invertebrate assemblages of water-filled tree holes at the study sites. Further investigations are envisaged to elucidate how dendrotelmic parameters regulate spatiotemporal variations in community structure and shape predator–prey interactions within these specialized aquatic ecosystems.

Keywords: Aquatic Invertebrates, Western Ghats, tree holes

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18

Gayathri Binu
DREAM Lab

Investigating the adult relevance of larval hexamerins in *Drosophila melanogaster*

The life cycle of holometabolous insects alternates non-feeding or “closed” periods (embryonic and pupal) with feeding or “open” ones (larval and adult). Therefore, the resources required to proceed through a closed developmental phase come from the stores made during the preceding open phase.

In *Drosophila*, larval growth is supported by nutrient intake, while the non-feeding pupal stage is fueled by vast reserves of storage proteins, primarily hexamerins known as Larval Serum Proteins (LSPs). Hexamerins are the storage proteins found in insects, known to reach a peak expression level during a specific developmental period. Larval Serum Proteins(LSP) - I and 2 are the hexamerin protein complexes found in Drosophila. LSP complexes are maximally produced in the third instar larvae by the larval fat body, analogous to vertebrate liver and adipocytes. It gets released into the hemolymph, making up 70% of the total hemolymph protein. Prior to pupa formation, LSP is reuptaken from the hemolymph to the fat body for utilization throughout development. These proteins are synthesized by the fat body during feeding and sequestered to serve as a critical amino acid source for metamorphosis. The mobilization of these stores is not a simple passive process but is tightly regulated by a sophisticated interplay between developmental timing and systemic nutrient-sensing mechanisms. The fat body itself acts as a primary nutrient sensor, utilizing the Target of Rapamycin (TOR) pathway to assess amino acid availability. High nutrients activate TOR, promoting protein synthesis and suppressing autophagy.

In this study, we analyze the role of these hexamerins by tissue-specific genetic manipulations, tease apart their role in larvae versus adults, and try to elucidate the signalling mechanisms that may be involved in the regulation.

Keywords: Drosophila, hexamerins, metabolism

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19

Geetika Shrimali
Chronobiology Lab

Circadian Rhythms involvement in Sensorimotor Decision-Making

Circadian rhythms organize physiology and behavior across the day; however, how the internal timekeeping system modulates neural decision-making circuits remains poorly understood. *Drosophila* larvae as a model system was used to investigate the influence of circadian timings on sensorimotor decisions that select actions in response to mechanosensory stimuli. At different timepoints behavioural assays under light-dark and constant darkness conditions suggested differences in behaviour in a time dependent manner, suggesting circadian effect. While developmental progression contributed to behavioral variability, experiments using developmentally synchronized larvae demonstrated circadian modulation. The results obtained thus far favor the hypothesis that circadian state might shape larval behavioral choices independent of development and establish a link between clock neurons to specific nodes within the larval decision-making circuit.

Keywords: Circadian Rhythms, sensorimotor decision-making, neural circuit

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20

Girija Jogwar
Genome Stability Lab

Understanding the role of mismatch repair in regulating meiotic recombination

Meiotic recombination is crucial for faithful segregation of homologous chromosomes. During this process, programmed DNA double-strand breaks (DSBs) are repaired by homologous recombination as crossovers and non-crossovers. During the DSB repair process, strand invasion into the homologous chromosomes generates heteroduplex DNA, which is stabilized by the mismatch repair related Msh4-Msh5 complex by forming clamp-like structures. The heteroduplex DNA may also contain mismatches due to the differences in homologous chromosome sequences. The mismatch repair protein Msh2 plays an important role in recognizing and repairing mismatches generated during heteroduplex formation. The presence of excess mismatches results in heteroduplex rejection and prevents its stabilization by the Msh4-Msh5 heterodimer.

A previous study in the lab showed that Msh5 binding is reduced in genomic regions with high heterozygosity (Dash et al., 2024). To further investigate the effect of heterozygosity, we analyzed Msh5 binding in *msh2Δ* by performing calibrated ChIP-sequencing in homozygous and heterozygous *S. cerevisiae* backgrounds. Heterozygous *msh2Δ* strains showed a significant increase in Msh5 binding compared to wild-type. The SNP density in Msh5-bound regions were also enhanced in the *msh2Δ* mutant relative to wild type, suggesting enhanced Msh5 binding in heterozygous regions in the absence of Msh2. These results suggest a role for Msh2 in rejecting meiotic recombination intermediates containing excess mismatches. Interestingly, in the homozygous background lacking mismatches, the overall Msh5 signal was similar in *msh2Δ* and WT, but a higher percentage of Msh5 peaks showed localization at DNA hotspots suggesting a role for Msh2 which is independent of its MMR functions. To investigate it further, we performed calibrated Msh2 ChIP-Seq. Msh2 showed strong localization overlapping with that of Msh5. This clearly suggests that it interacts with recombination intermediates irrespective of the presence of mismatches.

To further understand the role of Msh2 in meiosis, we will analyze Msh2 localization in the heterozygous strains. We will also quantify Msh2 foci in meiotic chromosome spreads in WT and meiotic mutants to gain insight into the meiotic role of Msh2. Overall, we aim to unravel the meiosis-specific function of Msh2 independent of mismatch repair. This will help identify non-canonical functions of Msh2, if any.

Keywords: Mismatch repair, meiosis and recombination

IISER THIRUVANANTHAPURAM

21

Gopika Gopan
Pondicherry University

Characterization of Kit Signaling in Hematopoietic Cells

Kit is a type III Receptor Tyrosine Kinase that elicits the biological response via binding to Kit ligand (KL) aka stem cell factor. In addition to the activation of intrinsic signaling pathways, Kit has been shown to interact with lineage-restricted type I cytokine receptors and produce cross signals eg., EpoR, IL-7R, IL-3R. To investigate other Kit-activated receptors, we tested Kit and IL-4R cross-receptor activation in murine bone-marrow derived mast cells, which express both Kit and IL-4R at the surface level. Kit upon activation by Kit ligand (KL), activated IL-4Rα, γC, and STAT6 independent of its cognate ligand IL-4. Though KL and IL-4 are individually mitogenic, combinations of KL and IL-4 synergistically promoted mast cell proliferation. Furthermore, we investigated the mechanism behind the KL and IL-3 induced synergistic proliferation in human megakaryocytes. Lipid rafts are reported to be important for Kit mediated signaling pathway in hematopoietic stem cells. We observed loss of synergistic proliferation induced by KL and IL-4 or IL-3, upon depletion of lipid raft by methyl-β-cyclodextrin. We observed that lipid rafts are required for KL and IL-3 dependent enhanced phosphorylation of Erk and p38. We also observed that KL and IL-3 dependent synergistic proliferation are independent of Jaks. Furthermore, to identify the novel Kit signaling dependent protein expression, we performed total proteomics using 2D gel electrophoresis (2DGE). The protein spots with highly significant differential expression were excised and identified using MALDI-TOF/MS or LC-MS/MS. The pathway analysis of proteins identified from 2DGE revealed that most of the identified proteins were involved in pathways that regulate cell proliferation, metabolism, actin reorganization and platelet formation. Overall, the findings suggest IL-4R as novel Kit interacting receptor and lipid rafts are required for Kit intrinsic and cross receptor signaling.

Keywords: C-kit, Hematopoiesis, Lipid raft

IISER THIRUVANANTHAPURAM

22

Gopika K G
DREAM Lab

Interplay between hypoxia and insulin signaling in regulating growth and metabolism in *Drosophila melanogaster*

Oxygen is vital for cellular metabolism, and reduced oxygen levels (hypoxia) activate the Hypoxia-Inducible Factor (HIF) pathway to maintain homeostasis. In *Drosophila melanogaster*, the HIF-α homolog Similar (Sima) and HIF-β homolog Tango (Tgo) mediate hypoxia responses, while Fatiga (Hph) promotes Sima degradation under normoxia. Although HIF signaling is well studied in hypoxic adaptation, the non-canonical regulation of Sima based on nutrient availability and its role in growth and metabolism remain poorly understood.

Our studies show that larvae exposed to 5% oxygen display delayed pupation and reduced body size, phenotypes mimicked by ubiquitous sima overexpression. Using tissue-specific GAL4 drivers, we found that sima activation in insulin-producing cells (IPCs) suppresses growth and delays development, while sima knockdown enhances growth. Reporter analyses (HRE-LacZ) revealed sima activity in IPCs even under normoxia, which increases under hypoxia. sima overexpression in IPCs causes DILP2 retention and reduced insulin signaling, indicating that Sima directly regulates insulin output in response to oxygen and nutrient cues.

Ongoing work aims to dissect tissue-specific Sima functions in IPCs and fat body using molecular reporters and physiological assays. This study will uncover how HIF signaling integrates oxygen and nutrient status to coordinate growth and metabolic balance in Drosophila.

Keywords: Hypoxia, Hypoxia inducible factor, sima,Insulin producing cells,DILPs

IISER THIRUVANANTHAPURAM

23

Gowtham K P
EvoSysBio Lab

Prokaryotic Distribution Of Predominant Eukaryotic Enzyme; Structural And Evolutionary Analysis

PFKFB is a bifunctional enzyme that regulates fructose-2,6-bisphosphate (F-2,6-BP), a key metabolite that shifts cells between glycolysis and gluconeogenesis. While the enzyme is well-studied in eukaryotes, its presence and behavior in bacteria remain incompletely understood. Earlier reports suggested that only Desulfovibrio desulfuricans carried a PFKFB homologue. In this work, the evolutionary spread and structural features of PFKFB were examined across both bacteria and eukaryotes. Domain architecture analysis revealed ten possible arrangements, with the canonical Kinase and phosphatase domain layout being the most common and abundant. Unexpectedly, several bacterial groups, including Thermodesulfobacteria and Myxococota, also carried clear PFKFB homologs. Phylogenetic analysis revealed two separate bacterial lineages: Desulfovibrio formed a highly diverged branch, while Myxococota sequences grouped with algal proteins, suggesting horizontal gene transfer or shared ancestry.

Sequence and structural checks showed that the F-2,6-BP binding region in PFKI is missing in both bacterial proteins, whereas the FBPI binding residues are fully retained. Overall, the study shows that the bifunctional framework of PFKFB is present in bacteria, but the regulatory features differ considerably between lineages. These findings extend the known distribution of PFKFB and provide a basis for exploring whether F-2,6-BP has any regulatory function in bacterial metabolism

Keywords: Biochemistry, Evolution and Phylogenetics

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24

Hana Lukman
DREAM Lab

The role of *miR-986* in the metabolism and lifespan of *Drosophila melanogaster*.

MicroRNAs (miRNAs) are small, non-coding RNAs that guide post-transcriptional gene regulation by targeting messenger RNAs. They are essential for maintaining metabolic homeostasis by modulating genes that control nutrient storage, energy use, and response to metabolic stress. In line with the growing awareness of miRNAs in metabolic regulation, we are investigating the specific contributions of *miR-986* in controlling lifespan and metabolic adaptation in *Drosophila melanogaster*.

Functional characterisation of the microRNA revealed that brain-specific knockdown and overexpression of *miR-986* have significant roles in starvation resistance, accompanied by marked changes in metabolic profiles and feeding behaviours, thereby reinforcing its role in sustaining homeostasis. Intriguingly, knockdown and overexpression of *miR-986* in the fly brain yielded similar results, rather than the expected opposite phenotypes. Simultaneously combining overexpression and downregulation of the miRNA in the same fly rescues the phenotype, suggesting unexpected regulatory complexity. This also suggests the possible existence of compensatory pathways that activate only when both extremes (knockdown and overexpression) are present, allowing a “rescue” of the phenotype and warranting further investigation, such as target identification and pathway analysis, to clarify the underlying molecular mechanisms.

Ongoing efforts to identify downstream target genes of miR-986 aim to clarify the molecular circuitry responsible for organismal responses to nutrient stress and to investigate their potential role in neurodegeneration and ageing. By manipulating miRNA levels, we identify a novel role for *miR-986* in orchestrating both metabolic homeostasis and lifespan. Understanding miRNA-mediated regulatory circuits in flies provides a robust framework for elucidating the molecular basis of ageing and metabolic diseases in higher organisms.

IISER
THIRUVANANTHAPURAM

Structural studies of *Mycobacterium tuberculosis* LexA alone and in complex with its interacting partner: Insights into SOS Regulation

Tuberculosis still remains as one of the deadliest diseases which affect people of various age category including children. 1.23 million died from the disease in the year 2024 out of nearly 10 million TB cases around the globe, as reported by WHO in its Global Tuberculosis Report 2025. Tuberculosis remained a central cause for morbidity and mortality for centuries. SOS response is a fundamental DNA repair mechanism found across prokaryotes. It is regarded as an evolutionarily conserved pathway among bacterial species, even though the functional outcomes vary between species. LexA and RecA are the two key regulators of this response. LexA functions as a dimer and bind to SOS boxes, which are typically located downstream of RNA polymerase binding sites, there by suppresses the SOS genes. However, upon DNA damage, RecA forms a filamentous structure known as activated RecA (RecA*). This activated form of RecA facilitates LexA autocleavage and subsequent de-repression of the SOS genes. In *Escherichia coli*, where the system is best characterized, LexA regulates the expression of over 50 genes involved in DNA repair, cell cycle control, and mutagenesis. While much of our understanding comes from studies in *E. coli*, increasing attention is now being given to *Mycobacterium tuberculosis*. This study will summarize the optimization, overexpression, and purification of wild-type *Mycobacterium tuberculosis* LexA (MtbLexA) and its non-cleavable mutant variants. Biophysical and functional characterization of both the wild-type and mutant forms are carried out to understand their stability and activity profiles. Additionally, crystallization studies have yielded crystals of full-length MtbLexA mutant, both alone and in complex with a small molecule inhibitor. Diffraction quality crystals for both (~ 5.8 to 6.3 Å) are being optimized for better resolution diffraction data, thus showing potential for structure-based drug design as a lead candidate.

Keywords: *Mycobacterium tuberculosis*, LexA, Tuberculosis, Autocleavage, SOS Pathway

IISER THIRUVANANTHAPURAM

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SDS PAGE patterns of salivary gland proteins in Ecdysone and Hydroxyurea treated larvae

The present study investigates the effects of chemically induced modulation of protein synthesis in the salivary glands of *Drosophila melanogaster* and *Drosophila nasuta nasuta*. Larvae were exposed to hydroxyurea and ecdysone, compounds known to influence protein synthesis. SDS–PAGE analysis of salivary gland proteins in D. melanogaster revealed nine prominently visible protein bands (87, 71, 65, 56, 41, 37, 28, 22, and 19 kDa) in both control and ecdysone-treated groups, whereas hydroxyurea treatment resulted in a reduction in the number and intensity of protein bands, with only 87, 65, and 56 kDa bands remaining prominent. Similar results were observed in D. nasuta nasuta, where protein fractions of 94, 46, 34, 17, and 12 kDa were detected in control and ecdysone-treated larvae, while hydroxyurea-treated samples showed only 94, 46, and 34 kDa bands. These findings indicate that the chemicals ecdysone and hydroxyurea have modulated protein synthesis of the salivary gland in Drosophila species.

Keywords: SDS-PAGE, PROTEIN, DROSOPHILA

IISER THIRUVANANTHAPURAM

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HapGen Lab

Understanding the Roles of NASP and SGT1 in CENH3 Deposition and Genome Stability in *Arabidopsis thaliana*

Centromeres are essential regions of eukaryotic chromosomes responsible for the formation of kinetochore complexes that connect to spindle microtubules during cell division. This ensures faithful chromosome segregation, which is fundamental for genome stability. In plants, the centromere is defined by the histone H3 variant CENH3, whose precise deposition onto centromeric chromatin is essential for maintaining centromere identity and genome integrity. The histone chaperone NASP (Nuclear Autoantigenic Sperm Protein) has been established as a key factor mediating CENH3 loading in *Arabidopsis thaliana*; however, the precise molecular mechanisms by which NASP facilitates this process remain poorly understood. In parallel, SGTI (Suppressor of G2 allele of SKPI), a conserved regulator of kinetochore stabilisation and CENP-A activation in yeast, flies, and mammalian systems, has not yet been functionally characterised in the context of plant centromere biology.

This study aims to explore the mechanistic roles of NASP and SGTI in CENH3 deposition and genome stability in Arabidopsis thaliana.

Keywords: CENH3 loading; Centromeric chromatin; NASP and SGTI; Genome stability; Haploid induction; *Arabidopsis thaliana*

IISER THIRUVANANTHAPURAM

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Meiotic Cohesins in Cancer: Oncogenic Reactivation and Chromatin Dysregulation

The eukaryotic genome contains all the information required for cellular function, but spans several meters and must be compactly organized within the nucleus. This is achieved through its association with histone and non-histone proteins to form chromatin, a dynamic structure that balances compaction with accessibility. Higher-order chromatin organization is a key regulator of gene expression and strongly influences cell identity, genome stability, and developmental outcomes.

A central regulator of genome architecture is the cohesin complex, a ring-shaped, ATP-dependent protein assembly that holds DNA strands together and organizes the genome in 3D. Working together with the architectural protein CTCF, it drives loop extrusion to form topologically associating domains, enabling long-range regulatory interactions between enhancers and promoters. Beyond interphase genome organization, cohesin plays essential roles throughout the cell cycle, including sister chromatid cohesion, DNA replication and repair, and accurate chromosome segregation.

Cohesin exists in distinct mitotic and meiotic forms. Somatic cells predominantly express mitotic subunits such as RAD2I, SMCIA, and STAGI/2, whereas germ cells utilize meiosis-specific subunits including REC8, SMCIB, and STAG3 to remodel chromosomes for homolog pairing and recombination. These meiotic cohesins are normally silenced in somatic tissues, but their aberrant reactivation or dysfunction can disrupt genome stability and alter cell fate.

Reactivation of meiotic cohesin subunits has been reported in several cancers and may disrupt chromatin organization and genome stability. To study this, we are generating a somatic cell line system to replace a mitotic cohesin subunit with its meiotic counterparts and assess the consequences on cohesin complex assembly, cohesin loading, cell viability, chromatin reorganization, and oncogene expression.

The project aims to elucidate how cohesin-mediated loop extrusion and 3D genome organization regulate chromatin architecture and gene expression, and how alterations in cohesin function reshape cellular identity and developmental outcomes.

Keywords: Meiotic cohesins, Chromatin organisation, Cancer

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29

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Annamalai University

AI-Driven Synthetic Biology: Charting the Next Frontier in Biological Engineering

Synthetic biology aims to design and construct novel biological systems for applications in medicine, agriculture, and environmental sustainability. However, traditional trial-and-error approaches are time-consuming, costly, and limited in handling biological complexity. The integration of artificial intelligence (AI) is transforming synthetic biology into a data-driven, predictive engineering discipline. This abstract highlights how AI is redefining synthetic biology by accelerating biological design, improving precision, and enabling scalable, reliable biological engineering. Advances in machine learning, deep learning, and generative models are being applied to multi-omics datasets, protein structures, metabolic networks, and gene regulatory circuits. AI-assisted tools enable rational design of DNA sequences, optimization of metabolic pathways, prediction of protein structure–function relationships, and automated design–build–test–learn (DBTL) cycles. These approaches significantly reduce experimental burden while enhancing accuracy and reproducibility. AI-driven synthetic biology has enabled rapid enzyme engineering, intelligent genome editing, programmable gene circuits, and the development of engineered microbes for drug synthesis, bioremediation, and climate-resilient agriculture. The convergence of AI with high-throughput automation and synthetic genomics is fostering autonomous biological laboratories and digital twins of living systems. Despite its promise, challenges remain, including data bias, model interpretability, biosecurity, and ethical governance. Addressing these issues through standardized datasets, explainable AI, and robust regulatory frameworks will be critical. AI-driven synthetic biology represents a paradigm shift from empirical biology to predictive bioengineering, positioning it as a central frontier for next-generation innovations in health, sustainability, and industrial biotechnology.

Keywords: Synthetic biology, Artificial Intelligence and Predictive Bioengineering

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EcoPhys Lab

Can winter crops embrace warming? We report novel thermal phenotypes and key photosynthetic functional targets for temperature resilience in rapeseed-mustard

Our current understanding of how the winter crops would respond to the mean temperature rise and terminal heat stress is limited. Also, the vulnerability of the winter crops to elevated temperatures is a key limitation for promoting them in the non-traditional warmer regions. In this study, we selected rapeseed-mustard as a model winter crop system to understand the effects of high temperature environment on winter crops. Precisely, we used the OJIP chlorophyll a fluorescence (ChlF) approach to analyse various Photosystem II (PSII) dynamic processes, potential limits to PSII efficiency, and relationships between PSII parameters and yield traits under high temperature environment. Seventeen rapeseed-mustard genotypes were grown under elevated temperature conditions in a greenhouse, with daytime temperatures peaking at ~37°C. ChlF was measured at the flowering stage during morning, afternoon, and evening time points to assess PSII efficiency (FV/FM), performance indices (PITOT and PIABS), and OJIP fast kinetics. We found significant genotypic variation in the afternoon loss and evening recovery of PSII bioenergetic properties, including FV/FM, FM, PIABS, and PITOT, although no permanent PSII damage was evident. We identified a strong increase in photoprotective regulation under elevated temperature. The IP phase of the OJIP kinetics was strongly associated with PIs and enabled classification of genotypes into two thermal phenotypes based on their sensitivity to diurnal warming. Among all parameters, afternoon PITOT showed the strongest relationship with silique yield, highlighting its potential as an emerging functional bioenergetic trait for identifying heat-resilient, high-yielding rapeseed-mustard genotypes under high temperature environment.

Keywords: Chlorophyll a fluorescence, high temperature, OJIP fast kinetics

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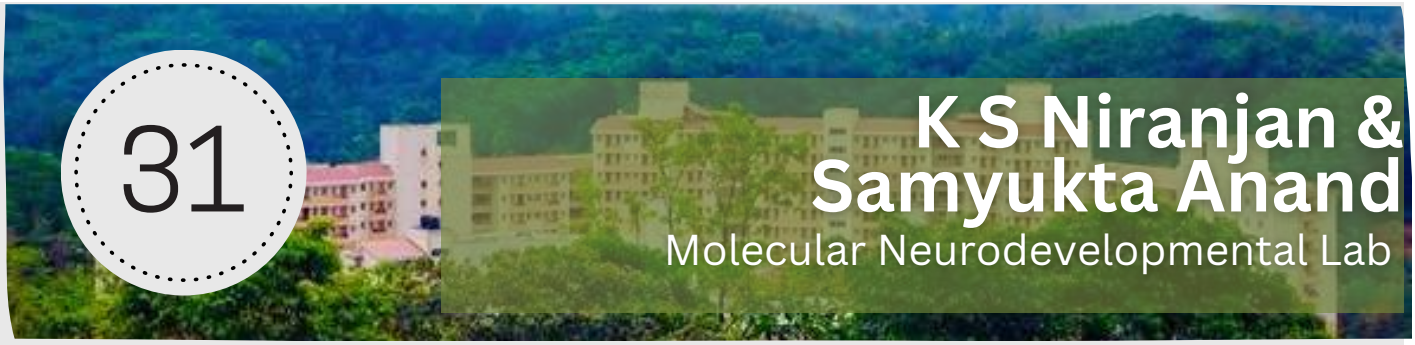

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Molecular Neurodevelopmental Lab

Uncovering the Genetic Basis of Hot-Water Reflex Epilepsy Using Zebrafish

Hot water epilepsy is a type of reflex epilepsy in which seizures are caused by the stimulus of hot water poured over the head. In humans, it has been documented that bathing with hot water can trigger seizures, and these seizures usually begin in a specific area of the brain, but in some cases, they can spread and affect the entire brain. Previous investigations have largely been limited to case reports, and the genetic basis for hot-water reflex epilepsy (HWE) is only beginning to be understood. In this study, using the zebrafish (*Danio rerio*) model, we sought to elucidate the cellular and molecular underpinnings of hot-water reflex epilepsy (HWE) resulting from mutations in two candidate genes: *slc1a1* and *r3hcc1*. A heat treatment paradigm was standardized in larval wild-type zebrafish to characterize the seizure phenotypes elicited due to elevated temperature. The heat exposure reliably induced not just simple behavioural changes such as hyperactivity, but also whirlpooling and convulsions. At the molecular level, the heat exposure led to an upregulation of the thermosensitive ion channel genes *trpv1* and *trpv4*, and the immediate early gene *c-fos*, a marker for neuronal activity. Temporal and spatial expression analysis of *slc1a1* and *r3hcc1* genes was performed to determine their developmental and regional expression patterns. Mutant zebrafish lines were generated using CRISPR-Cas9 genome editing and current behavioural experiments on homozygous and heterozygous mutants using the aforementioned heat treatment paradigm will establish whether these mutations result in enhanced seizure susceptibility. In parallel, slc1a1 was pharmacologically inhibited using DL-TBOA in order to mimic the behavioral phenotypes observed in the absence of functional *slc1a1*. The mutant lines will be used to further characterize the cellular and molecular basis of hot water epilepsy in patients with mutations in *slc1a1* and *r3hcc1*. We plan to conduct further heat exposure treatments with the mutant lines generated.

Keywords: Zebrafish, Reflex epilepsy, Genetic disorder

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Hypocrellin B with carbon dots loaded hydrogel for the photodynamic treatment of Diabetic retinopathy

A nanoparticulate photodynamic therapy approach was employed to enhance the singlet oxygen production using carbon dots associated with the attempt to improve the solubility of photosensitizer Hypocrellin B, for the management of posterior segment eye diseases Diabetic retinopathy.

Keywords: Hypocrellin B, retinopathy, photodynamic

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33

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Central University of Kerala

Leaf Ecophysiological Responses of *Kandelia candel (L.) Druce* to Salinity and Implications for Species-Level Ecological Adaptation

Kandelia candel (L.) Druce, an important mangrove species in coastal ecosystems, faces significant ecological challenges in the stressful environment. This study explores the physio-biochemical responses of *K. candel*, providing insights into its adaptive strategies under salinity stress. Two-month-old seedlings were grown in pots under varying salinity conditions (control, 10, and 20 Parts Per Thousand [PPT]), and analysed for leaf-level ecophysiological trait responses at 15 and 24 days after treatment (DAT). Analysis of key adaptive attributes - such as oxygen evolution, water potential, antioxidant activity, and biochemical stress markers revealed significant variation in leaf traits across salinity levels. Oxygen evolution and consumption rate and total phenolic content declined at high salinity, indicating stress-induced alterations. At 20 PPT, elevated malondialdehyde (MDA) levels, decreased chlorophyll stability index, and increased membrane injury served as key stress markers of salt-shock-induced leaf-tissue damage. These physiological disruptions underscore the sensitivity of *K. candel* leaves to high salinity. This investigation also highlights the beneficial roles of short-term salinity exposure on the leaves. *K. candel* can tolerate salinity stress (below 20 PPT), which could be attributed to increased leaf nitrogen, water potential (Ψ), and proline. These adaptations likely enable *K. candel* to withstand moderate salinity levels, underscoring its physiological resilience for survival in saline environments. Overall, leaf salt-sensitivity played a notable role in determining leaf scale susceptibility. These leaf growth and physio-biochemical traits are reliable screening parameters for stress biology research as they remarkably affect stress responses.

Keywords: Antioxidants, Ecophysiology, Leaf water potential, Mangrove, Photosynthetic apparatus, Salinity

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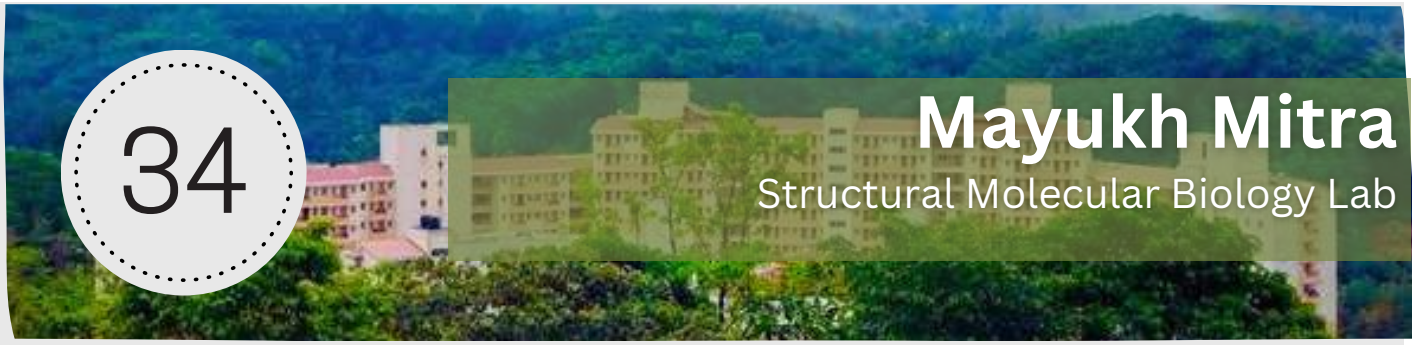

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Mayukh Mitra

Structural Molecular Biology Lab

Identification of molecular determinants of MtbMfd oligomerisation

The transcription-coupled repair factor (TCRF), known as Mfd (Mutation frequency decline) in *Mycobacterium Tuberculosis* is expected to transition between monomeric and oligomeric states *in vitro* and *in vivo*. Our *in vitro* studies gave an indication that the MtbMfd oligomeric species could be a dodecamer. While the non-pathogenic EcMfd is a monomer in solution. We had no knowledge of the conformation or the intermonomer interactions before the structure of monomeric MtbMfd at 3.6 Å was solved in our lab. In this study, we examine all possible oligomeric states of MtbMfd that can exist based on the crystal contacts of molecules present in the unit cell from the solved MtbMfd crystal structure. We analyse the possible inter-chain interactions in the oligomer by comparing the hydrophobic and hydrophilic regions in the monomer and oligomeric form by tracking the changes in accessible surface. This analysis is further supplemented with contact maps of the oligomeric forms and inter-chain hydrogen bonds. Here we report the presence of a highly hydrophobic patch on the C-terminal D7 domain of MtbMfd, which, in the case of *E. coli*, is hydrophilic. We also propose various interactions between the monomers in the probable Dodecameric forms, and experimentally pursue two such interactions.

Keywords: ASA, MtbMfd, Oligomarisation

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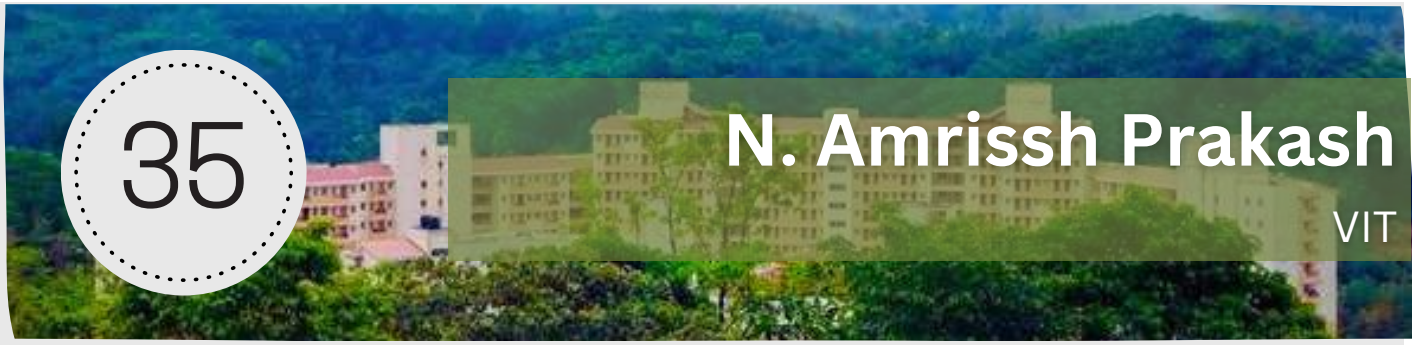

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N. Amrissh Prakash

VIT

E. coli lon protease in the substrate MarA in the conversion of 2,4 dnp to anp a novel inducer antibiotic resistance

Role of e coli encoded lon protease substrate novel inducer phenotypic antibiotic resistance

Keywords: Lon protease, dnp to anp, MarA conversion

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DREAM Lab

Neuromodulatory Control OF Metabolic Homeostasis by miR-100 in *Drosophila melanogaster*

MicroRNAs (miRNAs) are short, single-stranded, non-coding fragments of RNA that function as post-transcriptional gene expression regulators. They play a pivotal role in modulating essential cellular processes such as cell division, development, differentiation, apoptosis, inflammation, and response to viral infections. In particular, miRNA-mediated metabolic fine-tuning is important to maintain energy homeostasis and overall health of the organism. In this study, we investigate miR-100, a microRNA conserved across bilaterians, with a focus on its role in the neuromodulation of stress-dependent metabolic changes. In *Drosophila melanogaster*, miR-100 is encoded within the polycistronic let-7 complex and has been implicated in cancer and immune regulation in mammalian systems. Through tissue-specific screening, we observed that pan-neuronal downregulation of miR-100 enhances stress resistance. Further experiments revealed that reduced miR-100 expression leads to altered metabolic profiles. Our findings reveal a previously uncharacterized role for miR-100 in neuromodulatory regulation of metabolism under stress, providing new insights into the post-transcriptional control of stress-related metabolic adaptations.

Keywords: microRNA, stress resistance and response, metabolic homeostasis

IISER THIRUVANANTHAPURAM

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EpiOmics Lab

Genome-wide Analysis of Antimicrobial Resistance–Associated Genetic Variation in *Mycobacterium tuberculosis* Lineage 4

Drug-resistant tuberculosis (TB) remains a major global public health challenge, accounting for over one million deaths annually. The emergence and global dissemination of multidrug-resistant (MDR) *Mycobacterium tuberculosis* strains has substantially undermined current control strategies. A critical requirement for effective surveillance and intervention is a detailed understanding of how antimicrobial resistance (AMR) mutations arise, spread, and accumulate within specific pathogen lineages. However, distinguishing true causal resistance determinants from spurious associations driven by clonal population structure and shared ancestry remains a major analytical challenge in bacterial genome-wide association studies (GWAS).In this study, we investigated genetic variants associated with AMR in *M. tuberculosis* Lineage 4 (L4) using a GWAS framework that accounted for population structure. We performed a k-mer–based GWAS on 3,080 whole-genome-sequenced L4 isolates from multiple regions, phenotyped for resistance to 13 antibiotics. Our bioinformatics pipeline integrated genome assembly and annotation, pan-genome construction, phylogenetic inference, and association testing using linear mixed models with kinship correction. Draft genome assemblies were annotated using Prokka, pan-genomes were constructed with Panaroo, maximum-likelihood phylogenies were inferred using IQ-TREE, and k-mer–based association analyses were performed using Pyseer. Preliminary results demonstrate the robustness of this analytical framework, recovering well-established resistance determinants such as gyrA mutations associated with fluoroquinolone resistance with strong statistical support. We also observed several variants exhibiting apparent associations across multiple, mechanistically unrelated drug classes. Consequently, downstream analyses will focus on disentangling lineage-specific effects and linkage disequilibrium from resistance signals, enabling more reliable identification of causal AMR determinants and improving the interpretability of GWAS results in *M. tuberculosis*

Keywords:GWAS,Mycobacterium tuberculosis,Antimicrobial resistance

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Toxoplasma gondii co-opts the Host ACVR1–Iron Axis to Establish a Permissive Replicative Niche

Intracellular pathogens rely on host signalling networks to establish a permissive niche; however, the host kinases co-opted during infection remain poorly defined. To systematically uncover such dependencies, we exploited the polypharmacology of kinase inhibitors and combined their pre-existing activity profiles with parasite burden phenotypes to computationally infer host kinases that regulate *Toxoplasma gondii* replication. This approach, applied across ~300 kinases using 32 broad-spectrum inhibitors, identified ACVRI/ALK2 as a previously unidentified host determinant of *Toxoplasma* development in the host.

We find that *Toxoplasma* induces a temporal activation of the ACVRI-SMADI/5/9 axis between 6-I2 hours post infection, corresponding to the onset of rapid parasite expansion within the parasitophorous vacuole. Pharmacological inhibition or genetic silencing of ACVRI suppresses SMAD phosphorylation, disrupts vacuolar architecture, and markedly reduces parasite replication, establishing ACVRI as a key regulator of intracellular development. Importantly, *in vivo* administration of a selective ACVRI inhibitor in C57BL/6 mice markedly reduced parasite burden, confirming the requirement of ACVRI signalling for successful parasite establishment under physiological conditions

Integration of multiple existing datasets of infected host cells consistently revealed early activation of iron-regulatory genes, a downstream branch of the ACVRI-SMAD pathway. Functionally, iron supplementation rescues parasite growth in ACVRI-inhibited or knockdown cells, demonstrating that ACVRI supports parasite development by maintaining a bioavailable intracellular iron pool essential during the 6-I2 h early expansion window.

Toxoplasma secretory factors were sufficient to activate the ACVRI-SMAD axis, and interrogation of an existing pooled CRISPR screen with dual host-pathogen single-cell transcriptomic readouts enabled us to shortlist parasite effectors likely responsible for this activation. Ongoing work is focused on identifying the specific effector(s) that regulate this pathway

Together, these findings identify ACVRI as a mechanistically validated signalling hub hijacked by *Toxoplasma gondii* to remodel host iron homeostasis, establishing the ACVRI-iron axis as a key host vulnerability that can be targeted to disrupt intracellular parasite development.

Keywords: Polypharmacology, Host Gene Remodelling, Co-option of Host Iron Axis

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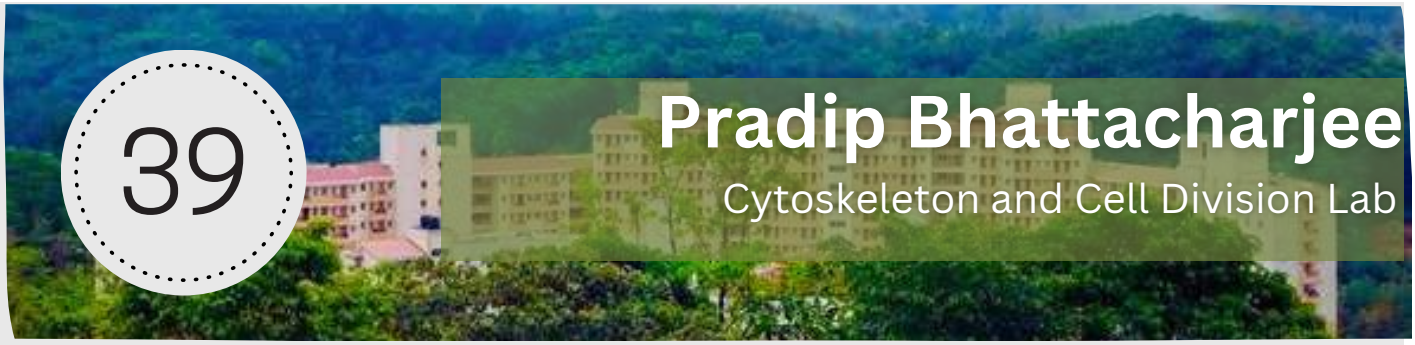

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Pradip Bhattacharjee
Cytoskeleton and Cell Division Lab

Regulation of Primary Cilia Assembly by Transforming Acidic Coiled-Coil Protein 3 (TACC3) in Human Cells

The primary cilium is a microtubule-based, centrosome-derived organelle present in most human cells and plays essential roles in mechanosensation, development, differentiation, and tissue homeostasis. Its assembly and disassembly are tightly coordinated with cell cycle progression, forming during G0/GI and disassembling prior to mitosis. Dysregulation of ciliogenesis is associated with cancer and a spectrum of disorders collectively termed ciliopathies, yet the molecular mechanisms governing ciliary biogenesis and resorption remain incompletely understood. A recent study implicated transforming acidic coiled-coil protein 3 (TACC3) as a suppressor of ciliogenesis in prostate cancer cells, although its mechanism of action remains unclear. Here, we show that TACC3 localizes to the basal body and is downregulated under induction of cilia assembly in cultured human cells. Depletion of TACC3 results in increased ciliated cells and elongation of primary cilia. Conversely, overexpression of exogenous TACC3 leads to a reduction of ciliary length and also decreases the overall population of ciliated cells, indicating its inhibitory role in ciliogenesis. Additional data support that TACC3 depletion deregulates the integrity of the transition zone and the dynamics of the intraflagellar transport proteins.

Keywords: Primary cilia, ciliogenesis, TACC3

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Functional annotation and structural characterization of a putative OsmC-like Protein in *Deinococcus radiodurans*

Deinococcus radiodurans is a gram-positive polyextremophile bacterium known for its extreme survivability under radiation environments. It can repair massive DNA damage including double-stranded breaks generated during radiation, desiccation, and oxidative stress in a time-efficient way during the post-irradiation recovery phase. Proteins involved in DNA repair pathways, and antioxidant and redox homeostasis pathways, play a crucial role in this restoration mechanism. Functional annotation, followed by protein-protein interaction (PPI) analysis of one of those pathways in *D. radiodurans*, reveals a previously uncharacterized protein as a putative OsmC-like protein.

Osmotically inducible protein C (OsmC) is a family of proteins involved in the detoxification of organic peroxides. They are part of a highly conserved antioxidant protein family with functional similarity to the hydroperoxide resistance protein. Proteins of the OsmC family share a conserved fold containing two cysteine residues at the active site, which are involved in the reduction of peroxides through the 'thiol-based redox' reactions. Here, we discuss the structural and functional properties of the putative OsmC-like protein in *D. radiodurans*; its sequence and structural properties, and its structural dynamics compared to radiation sensitive *E. coli* OsmC. The detailed results on the sequence and structural features of *D. radiodurans* OsmC, in comparison with known OsmCs, and their implications for understanding extremophile survivability will be presented.

Keywords: Uncharacterized proteins, protein function annotation, stress response, extremophile

IISER THIRUVANANTHAPURAM

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Reshma TT
EcoPhys Lab

Mangroves in context of global change: a study in urban areas and small islands of Kochi

Mangroves are unique coastal ecosystems of tropical and subtropical regions. With rising global temperatures and heatwaves, many species are approaching their physiological limits that threaten photosynthesis and overall fitness. Species-rich urban mangrove sites exist in and around Kochi, a major port city of Kerala, India. Many sites have deteriorated, fragile mangrove stands due to stress complexities. We selected six sites in Kochi with different hydrological characteristics, various degrees of industrial pollution, and urbanization pressure. As evergreen systems, mangroves retain their canopy year-round and are exposed to prolonged stress. The resilience of the Kochi mangroves towards urban heat, weather fluctuations, air pollution, and stress complexities may depend on their capacity to acclimate to stress exposure, particularly at the leaf level. Although there is some evidence that mangroves possess a very high photosynthetic thermotolerance (PT), our understanding of the acclimation of PT in mangroves is limited. PT is a key predictor of plant responses to heat, with metrics such as T50 (the temperature causing 50% damage to photosystem II) providing insights into species-level stress tolerance and potential tipping points. We investigated PT in II mangrove species across six sites representing three habitat types, namely riverine (Aroor, Valanthakad), estuarine (Queens Walkway, Vallarpadam), and marine (Puduvypin, Malippuram) during the peak dry summer (March–April 2025). Our study revealed strong interspecific variation in PT among mangrove species. The T50 ranged from ~49.7 °C in *Excoecaria agallocha* to ~53.6 °C in *Rhizophora mucronata,* and generally higher T50 was recorded at the marine sites. Based on ΔT_{50} (difference between the highest and lowest T50 values recorded for a species), we grouped the mangrove species into highly (ΔT_{50} > 2 °C) and moderately (ΔT_{50} = I–2 °C) acclimating categories, with no species exhibiting ΔT_{50} < I °C. Overall, elevated T_{50} values and the absence of low-acclimating species indicate substantial thermal resilience and acclimation capacity of photosystem II in mangroves existing in Kochi.

Keywords: Acclimation; Heat, Mangroves, Photosynthetic Thermotolerance, Urban Pressure

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Cytoskeleton and Cell Division Lab

Highly expressed in cancer 1 (Hec1) regulates kinetochore fibrous corona organization in human cells

Faithful chromosomal segregation relies on error-free end-on attachment of microtubules to the centromere associated supramolecular protein complex, kinetochore. Failure of this process leads to aneuploidy and associated diseases. Kinetochore outer plate protein complex NDC80 consisting of Nuf2, HecI (NDC80), Spc24 and Spc25, plays a key role in stabilizing the microtubule attachment mainly via interaction of HecI. Previous studies have shown that mitotic checkpoint kinase, Monopolar spindle-I (MpsI) interferes with HecI-microtubule binding through its interaction with HecI. Interestingly, kinetochore organizes an expanded crescent shape fibrous structure, called fibrous corona, covering its outer plate in the microtubule unattached condition and the formation of the fibrous corona requires MpsI kinase activity. However, the molecular link between HecI-microtubule interaction and MpsI kinase mediated fibrous corona organization with respect to mitotic progression remains unclear. Here, we show that HecI plays an essential role in stabilizing the expanded organization of fibrous corona in human cells. Binding analyses show that HecI mediates interaction to the core fibrous corona proteins. Mutations of the MpsI binding sites of HecI results in loss of fibrous corona expanded structures of kinetochores in the microtubule unattached condition and the same also inhibits HecI interaction with the fibrous corona proteins. Additionally, Fibrous corona stabilization was also observed in cells expressed with microtubule binding inhibitory phosphomimicking mutant of HecI specific to Aurora B phosphorylating sites in its N- terminal tail, whereas phosphodeficient mutation of the same cause loss of the fibrous corona. Results so far indicate an opposing molecular mechanism in controlling mitotic progression via stabilization of fibrous corona.

Keywords: HecI, Kinetochore fibrous corona, MpsI kinase

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Beyond Consensus: Investigating Sequence-Specific Recognition of Near-Palindromic CTCF Binding sites

At the chromosome level, chromosomes occupy separate regions within the nucleus termed chromosome territories. At the megabase scale, chromatin is organised into topologically associating domains (TADs), which are self-interacting genomic regions that engage in frequent internal DNA contacts while maintaining reduced interactions with other genomic regions. CTCF, the CCCTC-binding factor, is an II-zinc-finger DNA-binding protein that acts as the principal insulator and marks TAD boundaries. This study investigates a subset of the canonical DNA-binding sites of CTCF called near-palindromic CTCF binding sites (CBSs). The core sequences of a CBS are often palindromic in nature and identified by CTCF in a unidirectional manner. This gives rise to an interesting scenario with respect to the determinant of CTCF binding directionality since a convergent orientation of adjacent CTCF is crucial to the formation of TADs. We mapped palindromic CBSs across the genome and dissected the roles of the central base pair (A24) and adjacent CBS modules in mediating CTCF directionality and in forming topologically associating domains (TADs) at boundaries. Utilising WT Q4I8 and mutant Q4I8R datasets, statistical and motif analyses revealed a dramatic shift in binding preferences when the A24-Q4I8 interaction was disrupted, with implications for altered 3D chromatin architecture. These multidisciplinary approaches demonstrate how DNA sequence can dictate chromatin organisation and gene expression, highlighting their applications in genome engineering and in the discovery of disease-associated regulatory SNPs.

Keywords: CTCF, Palindromic DNA, Cancer

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Laboratory of Immune Cell Biology (LICB)

Influence of the MJD family DUB, ATXN3L, in Mitochondrial-Associated Degradation

Mitochondria are energy-generating organelles that govern crucial metabolic processes in eukaryotic cells, including energy production via oxidative phosphorylation, lipid metabolism, calcium signalling, and the maintenance of redox homeostasis. Mitochondria, due to their dynamic nature, are susceptible to cellular environmental changes such as hypoxia, nutrient stress, and infection. To combat this, the cell has developed various mechanisms of mitochondrial quality control that preserve mitochondrial integrity, ranging from targeted degradation of individual mitochondrial proteins to clearance of damaged mitochondria via mitophagy. The mitochondrial-associated degradation (MAD) pathway is a quality-control pathway that targets misfolded proteins or proteins associated with damaged mitochondria for ubiquitination, followed by proteasome-mediated degradation. Multiple mitochondrial and non-resident mitochondrial E3 ligases are known to ubiquitinate outer mitochondrial membrane (OMM) proteins for ubiquitination and degradation. VCP (valosin-containing protein), a AAA ATPase, assists in the extraction of the misfolded and ubiquitinated OMM protein from the mitochondrial membrane for targeting to the proteasome. In multiple neurodegenerative diseases, aggregation and reduced clearance of misfolded mitochondrial proteins have been reported. One such pathology is SCA3 (spinocerebellar ataxia 3), which is clinically manifested by circular mitochondria in fibroblasts, decreased OXPHOS and aggregates in cerebellar tissue sections. These patients exhibit accumulation of the MJD (Machado-Josephin Disease) family DUB (deubiquitinase), Ataxin-3, in Purkinje cells of the cerebellum. These cells have also been shown to have dysfunctional mitochondria, indicated by fragmented mitochondria in mouse models of SCA3 and increased ROS in these cells. Recently, ATXN3L, a paralog of ATXN3 and a more potent DUB of the MJD family, has been reported to be associated with Alzheimer's. However, very little is known about the protein or its role in mitochondrial homeostasis. In our study, we have established that ATXN3L targets the endosomal E3 ligase, RFFL, which targets MFN2 for degradation and primes damaged mitochondria for Parkin recruitment and further elimination. ATXN3L can interact with RFFL and deubiquitinate it, thus affecting RFFL-mediated degradation of two OMM proteins, PINKI and MFN2. Further, studies in our lab are trying to establish that RFFL facilitates MAD through its interaction with VCP, and ATXN3L can inhibit this process. This evidence thus establishes a novel role for ATXN3L in mitochondrial protein biology.

Keywords: Mitochondria-associated degradation (MAD), ATXN3L, Ubiquitin proteasomal system (UPS)

IISER THIRUVANANTHAPURAM

Deciphering the implications of Non-B DNA structures in the spatial organization of the genome

Genomic DNA canonically adopts a standard B-DNA conformation. However, non-canonical DNA structures such as flaps, overhangs, hairpins, holiday junctions, cruciform, gapped structures, triplexes, bubbles, R-loops, G-quadruplex and heterologous loops are formed in the genome transiently during physiological processes. Some of the structures like G-quadruplex are relatively more stable. G quadruplexes (G4s) are non-canonical structures that are exclusively formed by G-rich sequences, the conformation is such that it is folded into a four-stranded configuration. These are monovalent cation-dependent structures stabilized by hydrogen bonding. Studies have shown that these structures can form *in-vivo* and can be sequence-specific as well. They can have important regulatory roles in processes like replication, transcription and translation. G-quadruplexes are enriched in the transcription start site (TSS) of many genes compared to that in gene-rich regions. Further, at boundaries of topologically associated domains (TADs), which are functional and structural unit of genome organization, large amounts of single-stranded DNA could be generated owing to the frequent transcription of genes. It has been recently pointed out that the boundaries of TADs are enriched with G-quadruplex. They also showed that architectural proteins like CCCTC binding factor (CTCF), and cohesin are enriched in the TAD boundaries. Independent studies from zinc fingers, other than that from CTCF show that zinc fingers can bind to non-B DNA structures. In addition, TAD boundaries are sites of active transcription and are associated with the enrichment of R-loops. In this project we hypothesize that binding of the zinc fingers of CTCF to non-B DNA structures could rewire the genome organization locally.

Keywords: Chromatin, Genome organization and non-B DNA strutures

IISER THIRUVANANTHAPURAM

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Mutations Lab

Sub-lethal concentrations of antibiotics show a hormesis-like behavior in bacteria

Misuse and overexploitation of antibiotics have contributed to their dissemination in nature, resulting in an antibiotic concentration gradient in the environment and the human body as well. Consequently, bacteria are often exposed to sub-lethal levels of antibiotics, which are known to promote mutagenesis, biofilm formation, and antibiotic resistance. Despite the antibiotic's potency at sub-lethal levels, the general growth response of bacteria and the underlying mechanisms are still poorly understood. Here, we found a surprising behaviour of increased plating efficiency of *Bacillus subtilis* cells upon sub-lethal antibiotic treatments. We examined the effect under a range of sub-lethal concentrations across classes of bacteriostatic and bactericidal antibiotics, each targeting a different cellular process. We also observed this behavior when cells were exposed to a concentration gradient of antibiotics, mimicking the natural conditions. Overall, our results suggest likely activation of bacterial adaptive response in response to sub-lethal level of antibiotic treatments.

Keywords: sub-lethal, bacteriostatic, bactericidal

IISER THIRUVANANTHAPURAM

Understanding nectar robbery dynamics in a perennial shrub *Asystasia gangetica*

Nectar robbery is a well-documented form of floral damage, where foragers damage flowers while obtaining nectar without contributing to pollen transport. *Asystasia gangetica* is a perennial shrub exhibiting two colour morphs: yellow and white, and orientation morphs: horizontal and vertical, providing an active choice for nectar robbers (*Xylocopa* spp). We hypothesised that I) yellow floral morphs will receive greater robbing visits, 2) flowers with horizontal morphs will be robbed more, 3) robbed flowers will attract more primary robbers than pollinators, and lastly 4) robbed flowers would produce more nectar to compensate for the robbery. Through observations on flower-robber interactions and by quantifying the nectar compensation of robbed flowers in the field, we find that the nectar robbers show no significant differences in their choices while visiting yellow or white floral morphs. Horizontally oriented flowers were found to have significantly higher robbing visits than vertically oriented flowers. Assessing visitor choices revealed that robbing visits outnumbered pollinator visits in robbed flowers. Finally, we also found no significant difference in nectar volume or concentration of robbed flowers compared to the control. These results suggest that nectar robbery is trait-mediated and may influence pollinator behaviour, potentially affecting plant fitness.

Keywords: Plant-pollinator interactions, Insect behavior, Floral larceny

IISER THIRUVANANTHAPURAM

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Shivam kumar
HapGen Lab

What makes her SUPER? Understanding the Molecular Mechanism Behind the *superwoman* Phenotype in *Arabidopsis thaliana*

The number and position of floral organs in angiosperms are morphologically and genetically defined by complex regulatory networks. *Arabidopsis thaliana* has been widely used as a model for studying angiosperm floral development. Although much research has been done on the genetic and molecular networks that determine the identity of floral organs, less is known about the partitioning of the flower into four developmentally different whorls. The *SUPERMAN* (*SUP*) gene is a cadastral gene important for establishing boundaries between stamens in whorl three and carpels in whorl four. Epimutations in the *SUP* gene manifest as *superman*, *superwoman*, and *supersex* phenotypes. The superwoman phenotype is a consequence of hypermethylation of the *SUP* locus in the Wa-I ecotype, leading to a characteristic increase in carpel number and seed set compared to the wild-type. Here, we aim to understand the molecular mechanism underlying the *superwoman* phenotype conferred by one of the natural epialleles of the *SUP* gene. Our preliminary investigation, combining gene expression analysis with spatio-temporal expression patterns of candidate gene reporters, reveals an expanded domain of meristem maintenance factor in early floral buds, along with perturbed auxin metabolism and signalling. To test this further, we are employing auxin modulation treatments, targeted gene knockdown of candidate genes, and spatio-temporal expression analysis of additional meristem maintenance genes.

Keywords: Flower development, Plant development, Epigenetics

IISER THIRUVANANTHAPURAM

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Shreya Mishra
Vanasiri Lab

Incipient host race formation through host divergence in a butterfly

Insects interact with host plants at different stages of their life cycles. The herbivorous lifestyle compels insects to maintain close interactions with their host plants. Plants employ several defensive strategies to protect against herbivory, chief among which are secondary metabolites. Feeding on plants requires herbivorous insects to evolve physiological or behavioural adaptations that neutralize or tolerate the defensive chemicals produced by plants. The diversification and speciation of host plants over evolutionary time are often accompanied by corresponding evolutionary changes and speciation in herbivorous insects.

Butterflies can diverge in host races based on two major traits: Oviposition preference and Larval performance. We expect congruence between adult females' oviposition preferences and larval performance, as adult females should choose plants that maximize larval fitness. The difference in oviposition preference and larval performance between populations leads to host race formation over a broad evolutionary time course.

Here, we investigate the divergence of two populations: a humid zone population and an arid zone population of *Ariadne merione* based on host use. We have tested oviposition preference of adult and larval performance, and the results show differences in these two traits between populations of this butterfly. In the humid zone of Kerala, India, the population of the same species performs better and prefers one host plant, and in the arid zone of Tamil Nadu, India, another population performs better and prefers another plant. This phenomenon can lead to host race formation and eventual speciation through reproductive isolation. We also investigated which sensory cues are used in the host choice of adult females. Our results suggest that they rely on both vision and odour when locating suitable host plants.

Keywords: Oviposition preference, Larval performance, larval fitness, speciation

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Shyamalima B
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Regulation of GLUT1 trafficking by endosome-associated E3 ligase CARP2

Glucose uptake is largely facilitated by glucose transporter proteins (GLUTs). Among these, GLUTI is responsible for basal glucose uptake in many cell types. While the structure and transport function of GLUTI have been extensively studied, much less is known about the cellular mechanisms that regulate its trafficking between the plasma membrane intracellular vesicles. Emerging evidence suggests that ubiquitination plays a key role in directing GLUTI through endosomal and lysosomal pathways in response to changes in metabolic requirements. CARP2 (Caspase-8 and -I0 Associated RING Protein 2) is a RING-type E3 ubiquitin ligase that contains a FYVE-like domain, enabling its association with phosphoinositide-enriched membranes such as the plasma membrane and endosomes. Preliminary data indicate that CARP2 expression responds to extracellular glucose levels, pointing to a potential role for CARP2 as a glucose-sensitive regulator. Given the shared membrane localization of CARP2 and GLUTI, together with the involvement of ubiquitination in GLUTI trafficking, we propose that CARP2 regulates GLUTI stability and trafficking between the plasma membrane, endosomes, and lysosomes in a glucose-dependent manner. The goal of this study is to investigate the role of CARP2 in controlling GLUTI trafficking and stability, and to determine how glucose availability influences this CARP2-dependent regulation. Understanding this pathway will offer new insights into how glucose sensing is coupled to transporter regulation at the molecular level and how cells adapt their metabolism under both normal and disease conditions.

Keywords: Glucose, GLUTI, CARP2

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Somangee Chakrabarti

Chromatin Biology Lab

Investigating the Effect of Breast Cancer Gene 1 (BRCA1) DNA-Binding Domain (DBD) Point Mutations on its interaction with G-Quadruplexes (G4) Structures

The structural complexity of the human genome extends beyond the classical B-DNAhelix, encompassing non-canonical forms such as G-quadruplexes (G4s) that play important roles in gene regulation and genome stability. The tumour suppressor BRCAI, a key player in homologous recombination mediated DNA repair, has been reported to associate with G4 structures, helping resolve these secondary DNA formations during replication and transcription. This study aimed to explore the impact of BRCAI mutations on its ability to interact with G4 DNA. Mutations (EI038V, E5I5K, R866C, E597K, S889C and E9I4Q) were selected based on in silico predictions using SIFT, PolyPhen, and CADD scores to represent a range of functional consequences from tolerated to deleterious. We are trying to generate mutant constructs by site-directed mutagenesis by PCR overlap extension, followed by Sequence and Ligation Independent Cloning (SLIC) and sequence verification.

Preliminary protein expression and purification were optimized using *E. coli* BL2I cells. To analyze G4 formation, circular dichroism (CD) spectroscopy was performed, confirming the characteristic parallel G4 topology.

Integrating this with prior ChIP-seq analyses, which showed ~52% overlap between BRCAI and G4 peaks, the findings support a functional association between BRCAI and G4 structures. This work lays the groundwork for future biochemical and cellular assays to experimentally validate how specific BRCAI mutations influence its G4-binding capacity and contribute to genomic instability in cancer.

Keywords: G-Quadruplexes, BRCAI, ChIP-seq

IISER THIRUVANANTHAPURAM

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EcoPhys Lab

Mangrove thermoregulation in a tidal ecosystem: a focus on organismal thermal biology and functional ecology

Leaf thermoregulation (TRL), defined as the deviation of leaf temperature (TL) from ambient air temperature (Tair), is a key determinant of plant performance under high irradiance and thermal stress and is strongly influenced by leaf structural and water-related traits. Mangroves, largely restricted to tropical and subtropical regions, are generally classified as megatherms (TL > Tair), yet whether this behaviour is fixed or dynamically regulated across species and conditions remains unclear. We examined species-specific variation in TRL among nine coexisting mangrove species from a post-tsunami reforested habitat at Aayiramthengu, Kollam, using a combination of field measurements and controlled laboratory thermal kinetics assays. Field measurements captured natural diurnal variation in irradiance and leaf gas exchange , whereas laboratory assays exposed detached, upper canopy mature leaves to high irradiance (>1000 µmol m^{-2} s^{-1} PAR), isolating passive, trait-based thermal responses in the absence of stomatal conductance. Under field conditions, mangrove species exhibited both megathermic and poikilothermic (TL < Tair) behaviour, with several species showing transitory thermoregulatory shifts over the course of the day, often transitioning from poikilothermy in the morning to megathermy around midday with increasing solar exposure. Laboratory assays resolved species into two passive thermal categories, and comparison between field and laboratory responses revealed four thermoregulatory types, reflecting differing reliance on stomatal cooling and intrinsic leaf thermal traits. Across species, smaller leaf area combined with greater thickness and higher equivalent water thickness (EWT) promoted poikilothermic behaviour, while heating rate (T rate) showed significant positive correlations with leaf thickness and EWT, and maximum leaf temperature (Tmax) was associated with effective leaf width (ELW). Together, these findings demonstrate that mangrove leaf thermoregulation is a dynamic, trait-mediated process shaped by both passive structural properties and active physiological controls, rather than a fixed thermal classification.

Keywords: Mangroves, leaf thermoregulation, poikilothermy, megatherms, thermal traits, stomatal conductance, diurnal dynamics

IISER THIRUVANANTHAPURAM

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Decellularized Plant Leaf Scaffold

Decellularization of plant leaves has recently emerged as a promising and sustainable strategy for developing novel biomaterial scaffolds for tissue engineering and regenerative medicine. Plant leaves possess an intrinsic vascular architecture, hierarchical porosity, and anisotropic cellulose-based frameworks that closely resemble native extracellular matrix (ECM) topologies found in mammalian tissues. Through optimized decellularization protocols, cellular components are efficiently removed while preserving the structural integrity, microchannels, and mechanical stability of the leaf scaffold. These decellularized plant scaffolds provide inherent topographical cues that promote contact guidance–mediated cell migration, alignment, and organization, which are critical for tissue regeneration. Importantly, the natural venation network facilitates nutrient diffusion and potential perfusion, addressing a major limitation in conventional scaffold design. The cross-kingdom approach of integrating plant-derived scaffolds with mammalian cells represents a paradigm shift in biomaterials research, offering advantages such as low immunogenicity, cost-effectiveness, scalability, and environmental sustainability. Recent studies demonstrate successful adhesion, proliferation, and guided migration of mammalian cells on decellularized leaf scaffolds, highlighting their translational potential across diverse applications including vascular, cardiac, neural, and musculoskeletal tissue engineering. This abstract underscores the transformative potential of decellularized plant leaves as next-generation bioinspired scaffolds that bridge plant biology and regenerative medicine.

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Rab5B-positive endosomes are affected by the Golgi–CARP2 axis

Endocytic pathways play critical roles in organelle homeostasis, regulating internalization of receptors, intracellular trafficking, membrane recycling, and signaling. Early endosomes serve as central sorting hubs in these pathways, and their formation and maintenance are primarily governed by the small GTPase molecules, like Rab5. Though it is long believed that the plasma membrane is the main source for early endosome formation, recent studies in yeast show that alternative sources like the trans-Golgi network (TGN) can contribute to Rab5-positive endosome formation. However, many details, including molecular links between Golgi Rab5 endosomes, remain poorly understood.

CARP2/RFFL is a phospholipid-binding domain containing ubiquitin E3 ligase implicated in diverse cellular signalling pathways, including receptor recycling, trafficking, and cell migration. Since CARP2 localizes to both Golgi and endosomal compartments, we explored the existence of the Golgi-CARP2 axis and its potential role in early endosome formation. In this study, we investigated the role of CARP2 in endosome formation and distribution using CARP2 knockout cells and cells expressing different functionally defective CARP2 variants. We demonstrate that CARP2 is required for Rab5B-positive endosome formation and distribution. Our findings provide novel insights into non-canonical mechanisms of early endosome biogenesis and advance our understanding of organelle homeostasis. Importantly, when the hitherto unknown link between CARP2 and Rab5B is explored in renal carcinoma tissues using publicly available data, we found a clear correlation between increased expression of CARP2 and Rab5B with poor prognosis for human patients. Our findings provide novel insights into non-canonical mechanisms of early endosome biogenesis and advance our understanding of organelle homeostasis.

Keywords: Rab5B, CARP2, Endosomes

IISER THIRUVANANTHAPURAM

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Time-dependent ERα Agonist–Antagonist Switching by Cyanidin-3-O-Rutinoside via Sin3a Reprograms Early/Late Gene Expression and Cyclophosphamide Response in ERα-positive Breast Cancer

Background: Selective estrogen receptor modulators and phytoestrogens can paradoxically protect or sensitize tumors to chemotherapy, but the impact of exposure timing on cyclophosphamide response in ERα-positive breast cancer is unclear. This study examined how the dietary anthocyanin cyanidin-3-O-rutinoside (C3R) modulates ER signaling and determines cyclophosphamide chemoresistance versus chemosensitisation.

Method: ER+ cell lines: Breast Cancer- MCF-7, T-47D, osteosarcoma -MG63 and Cervical-HeLa were used. expression of ERα, ERβ, αa, DNA-PKcs, c-Myc, SLUG, and SNAIL were evaluated by using western blotting and/or Immunofluorescence. Cytotoxicity, apoptosis, mitochondrial membrane depolarization, were assessed by MTT, ANNEXIN V-FITC, JC-10 analysis respectively. Gene Silencing was carried out using SiRNA.

Result: C3R exhibits time-dependent modulation on MCF-7 and T-47D, transitioning from early agonism to late antagonism effecting the cyclophosphamide treatment outcome. In MG63, ERα agonism persists, demonstrating cell-specificity. C3R showed biphasic ERα modulation marked by enhanced early ERα, with concomitant ERβ suppression followed by late-phase ERα repression mediated through Sin3a recruitment. The duration of C3R-pre-treatment critically determined cellular response to cyclophosphamide. When administered after 3 hours of C3R exposure, cyclophosphamide treatment induced resistance, promoting proliferation through upregulation of estrogen-responsive survival factors, including NHEJ protein DNA-PKcs and proliferative driver c-Myc, which collectively attenuated cyclophosphamide-induced mitochondrial membrane depolarization and apoptosis. In contrast, cyclophosphamide treatment 24 hours post C3R exposure reversed this protective effect, restoring chemosensitivity, marked by the downregulation of ERα, c-Myc and EMT factors- SLUG and SNAIL.

Conclusion: C3R's time-dependent ERα agonist-to-antagonist transition determines chemoresistance/sensitization, revealing a paradigm where phytoestrogen exposure timing, not just concentration, governs treatment outcome.

Keywords: Cyanidin-3-O-rutinoside; Estrogen receptors; Sin3a

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Functional characterisation of the role of sterol acyltransferase in lipid droplet biogenesis

Introduction: Lipid droplets are neutral lipid–filled organelles that bud from the endoplasmic reticulum and are enclosed by a single phospholipid monolayer studded with structural and metabolic proteins. Their hydrophobic core is primarily composed of triacylglycerols and steryl esters but can accommodate other hydrophobic molecules; beyond serving as static lipid stores, lipid droplets function dynamically in stress responses, protein handling, inter-organelle communication, and cellular defence, and their formation and turnover are tightly regulated by contacts with other organelles.
Sterol acyltransferases are ER-localized, polytopic membrane enzymes that catalyze formation of steryl esters from sterols and fatty acyl-CoA, with different isoenzymes predominating under varying physiological conditions. Despite their central role in initiating steryl-ester–rich lipid droplet biogenesis, high-resolution structural and mechanistic information is lacking. In this study we aim to characterize the yeast sterol acyltransferases AREI and ARE2—defining their subcellular localization, probing ER cholesterol distribution during lipid droplet formation with a designed sensor, visualizing sensor enrichment at biogenesis sites, and investigating AREI oligomerization—to better understand the molecular events driving lipid droplet biogenesis.

Materials and methods: To investigate cellular localisation of Arel & Are2 to Fldl sites we expressed plasmid borne copy of GFP-AREI or GFP-ARE2 driven by ADH promoter in 4ΔKO mutants co-expressing Seil-mCherry genomically. The LDs were stained with neutral lipid dyes (BODIPY or MDH) and scored for the number of LDs per cell and LD-Fldl association quantitatively. Following this, Protein were isolated from the 3ΔGal-HA-Arel strain using HA pulldown (immunoprecipitation), and their oligomerisation state was analysed by western blotting and silver staining.

Results: Both pGFP-AREI and pGFP-ARE2 showed clear ER localization along with punctate distribution that colocalized with Seil foci. Interestingly, some of these Arel/Are2 puncta at Seil sites also colocalized with MDH stained LDs. For oligomerisation, preliminary results shows that Arel form oligomers.

Conclusion: Outcome of this study suggest that Arel & Are2 are ER localised colocalising with Fldl-mCherry and Are2 appears to form higher degree oligomer.

Keywords: lipid droplets, Arel, Are2, Fldl, endoplasmic reticulum (ER)

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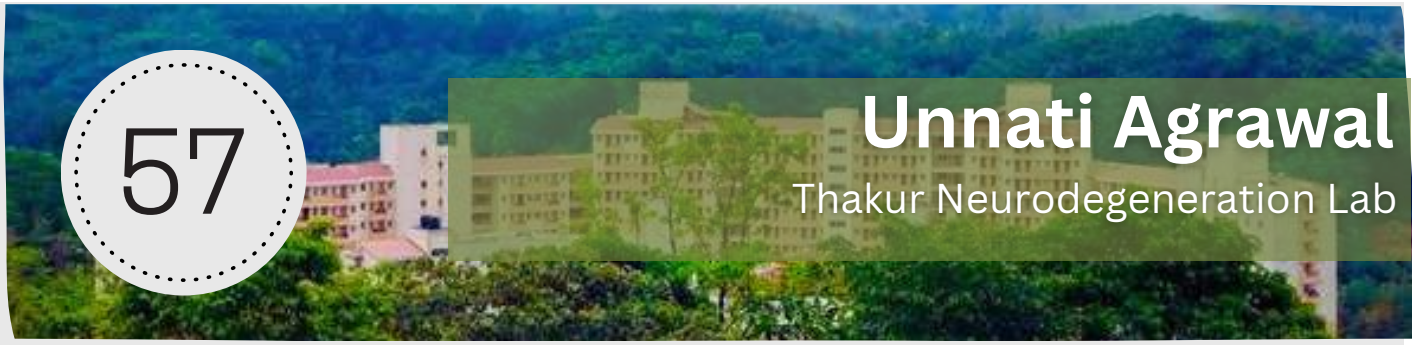

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Unnati Agrawal
Thakur Neurodegeneration Lab

Evaluating the Neuroprotective role of PROBUCOL in Parkinson's Disease

Despite advances in understanding the etiology of Parkinson's disease (PD), the absence of a definitive cure has intensified the focus on interventions targeting risk factors associated with PD, including lipid dysregulation. Several studies suggest that impaired cholesterol homeostasis accelerates PD progression. In this context, probucol, a lipid-lowering drug with established efficacy in cardiovascular and neurological diseases, represents a promising therapeutic candidate. We therefore aimed to evaluate its efficacy in a mouse model of PD that recapitulates key features of human pathology, strengthening its preclinical validation.

To address this, AAV-mediated α-synuclein overexpression in combination with preformed fibrils (PFFs) was injected stereotaxically in the substantia nigra (SN) region of the mouse brain. Probucol was administered intraperitoneally at a concentration of 15 mg/kg daily. To examine disease progression, behavioral tests were performed at different time points (4, 8, 12, and 16 weeks). Mice were sacrificed at a 16-week timepoint, and dissection of the brain was performed, followed by immunohistochemistry.
Despite the lack of severe motor deficits, the AAV+PFF model displayed a 30-35% decline in dopaminergic cells, corroborated by accumulation of phosphorylated α-synuclein (p-syn) and enhanced microglial and astrocytic activation, reflecting key hallmarks of PD. Probucol administration alleviated neurodegeneration, accompanied by decreased neuroinflammation. Additionally, probucol displayed sex-specific effects, with females demonstrating greater neuroprotection of dopaminergic cells. This effect was also evident in the attenuation of p-syn accumulation in females.

Our study revealed a sex-specific neuroprotective role of probucol in PD, prominently in females, by attenuating key pathological hallmarks of the disease. Thus, it implies that sex-specific molecular mechanisms indeed significantly influence therapeutic responses during PD progression. Collectively, our findings underscore the potential of probucol as a therapeutic candidate for PD, and further pathomechanisms will be evaluated underlying its sex-specific neuroprotective effects.

Keywords: Parkinson's disease, Neuroprotection, Probucol

IISER THIRUVANANTHAPURAM

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An Integrated In Vitro and In Silico Study on Targeted Cytotoxic and Molecular Mechanisms of Curcumin-Loaded Chitosan–$AgNO_3$ Nanoparticles in Triple-Negative Breast Cancer Cells

Triple-negative breast cancer (TNBC) represents a major global health challenge due to its aggressive behavior, limited treatment options, and high mortality rate. Conventional chemotherapeutic agents used for TNBC often cause severe side effects, as they lack of tumor specificity and damage both cancerous and normal cells. Curcumin a natural polyphenolic compound has attracted significant attention for its potent anticancer, anti-inflammatory, and pro-apoptotic properties. However, its clinical application is restricted by poor aqueous solubility, low bioavailability, and rapid systemic elimination. To overcome these limitations, the present study investigated the therapeutic potential of curcumin encapsulated within chitosan–silver nitrate nanoparticles (Ch–$AgNO_3$ NPs) for TNBC treatment. Chitosan was selected for its biocompatibility, enhanced cellular uptake, and favorable drug-loading capacity, while silver nanoparticles contribute synergistic cytotoxic and antimicrobial properties and improve nanoparticle stability. The synthesized Ch–$AgNO_3$–Cur nanoparticles were characterized using UV–Vis, FTIR, TEM, and XRD. The maximum drug-loading capacity and encapsulation efficiency were found to be 62.5 ± 0.9% and 82.5 ± 0.50%, respectively. In vitro cytotoxicity analysis revealed an IC_{50} value of 63.17 µg/mL for Ch–$AgNO_3$–Cur against MDA-MB-436 cells, demonstrating enhanced anticancer efficacy. The nanoparticles induced apoptosis in a dose-dependent manner, indicating effective cellular internalization and controlled release of curcumin. Furthermore, molecular docking studies of bioactive compound from Ch–$AgNO_3$–Cur against the human tumor suppressor protein Bcl-2 showed lower binding energy compared to cisplatin, suggesting stronger molecular interactions and potential apoptotic pathway modulation. Collectively, these findings highlight the promise of Ch–$AgNO_3$–Cur nanoparticles as a targeted and effective therapeutic strategy for TNBC. Nevertheless, further preclinical and clinical investigations are essential to validate their safety, pharmacokinetics, and therapeutic efficacy in human applications.

Keywords: TNBC; Chitosan–$AgNO_3$ nanocarriers; Apoptosis Targeted therapy

IISER THIRUVANANTHAPURAM


FLASH
TALKS

1

Amal K. Vyas
BEE Lab

Understanding the drivers of population differentiation in the Eastern honey bee (*Apis cerana*) in India

Aim: Honeybees are vital pollinators across diverse global habitats. However, Asian honeybees remain understudied, with significant gaps in our knowledge regarding their population structure and biogeography. In the Indian subcontinent, *Apis cerana* (the eastern honeybee) is widely distributed, yet its population status is poorly documented. While *A. cerana* is thought to exist as two distinct morphs—plains and hill—there has been no systematic, geographically widespread study of its population structure across the subcontinent. This study aims to address the drivers of the population structure of *A. cerana* in India.

Methods: To identify the drivers of population structure, we tested the effects of geographic distance and environmental variables on morphometric and wing-shape variation. We then compared these structures against the first and second principal components derived from 23 environmental variables.

Results: Our findings reveal three morphologically distinct population clusters corresponding to thermal and altitudinal gradients. The first population is adapted to high elevations, while the second is adapted to lower elevations and harsh continental habitats. Differences in wing shape among the lower-elevation samples indicate a third lineage adapted to lower seasonality; this group is restricted to the resource-rich, less seasonal coastal areas of the country. Environmental factors, combined with spatial position, play a major role in explaining geographically structured morphological variation. The presence of these lineages suggests that the resulting population structure stems from monopolisation followed by secondary contact.
Conclusion: Our results provide insights into the evolutionary processes shaping *A. cerana* populations in India. These findings have significant implications for the conservation and sustainable management of this essential pollinator.

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Anjitha K
Structural Molecular Biology Lab

Targeting the *Mycobacterium tuberculosis* evolvability factor Mfd

Mycobacterium tuberculosis (Mtb) is one of the most successful pathogens that can adapt and survive within host intracellular microenvironments in spite of continuous exposure to many DNA damaging agents. Mutation frequency decline (Mfd), the bacterial transcription-coupled repair factor, plays a critical role in nucleotide excision repair. In addition to its role in DNA damage repair, there are recent reports of the role of Mfd in prokaryotic virulence, and survival by promoting mutations. This has led to Mfd being called as an “evolvability factor”. Bacterial evolution drives AntiMicrobial Resistance (AMR) through chromosomal mutations. This necessitates the development of novel drugs that can effectively diminish the crisis. In this context, targeting the evolvability factor Mfd represents a promising path for the development of innovative antimicrobial drugs.

We have solved the crystal structures of MtbMfd and *Mycobacterium smegmatis* Mfd (MsMfd) (PDB: 6ACA, 6AC6) and nucleotide bound MsMfd (PDB 6ACX). Using Structure-Based Drug Discovery tools we aim to target MtbMfd. Molecular docking studies using a library of small molecules targeting MtbMfd, showed several promising hits. Out these hits, Compound 5 emerged as the positive candidate in *in vitro* screening. *In vitro* binding studies, including Isothermal Titration Calorimetry and Intrinsic Tryptophan Fluorescence confirmed the interaction between MtbMfd and Compound 5, providing key thermodynamic parameters like dissociation constant. We have crystals of MtbMfd in complex with Compound 5.

More recently, we studied Compound 6, an Mfd inhibitor in ESKAPE pathogens. *In vitro* studies showed that Compound 6 is inhibiting the ATPase activity of Mfd. Co-crystallization and cryoEM studies with the Mfd-Compound 6 complex yielded crystals and 2D class averages. Currently, we have two promising compounds that can be optimized to enhance specificity and potency against MtbMfd, providing a solid foundation for future inhibitor development.

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Aswathy BJ
DREAM Lab

Micro-managing the master regulator: Decoding the role of *miR-996* in the fly brain

MicroRNAs (miRNAs) are small, non-coding RNA molecules that post-transcriptionally regulate gene expression. They play a significant role in buffering fluctuations in gene activity, thereby preserving physiological stability. Their ability to simultaneously regulate multiple targets makes them key modulators of diverse biological processes, including development, metabolism, and lifespan. While the involvement of miRNAs in metabolic regulation is increasingly recognized, the specific roles of brain-derived endogenous miRNAs in regulating lifespan and metabolic homeostasis remain largely unexplored.

To fill this lacuna, we performed an unbiased screen in adult *Drosophila melanogaster*, in which 30 miRNAs were selectively downregulated in neurons. From this screen, *miR-996* emerged as a candidate of interest due to its physiological and metabolic relevance. Subsequent analyses investigated the effect of *miR-996* on metabolic parameters, nutrient-responsive behavior, locomotion, and aging.

Our results demonstrate that the absence of *miR-996* enhances starvation resistance and is associated with significant alterations in energy metabolism and feeding behavior, indicating a central role in maintaining metabolic equilibrium. Conversely, neuronal overexpression of *miR-996* results in increased starvation sensitivity and accelerated aging. These findings underscore the pivotal role of *miR-996* in orchestrating metabolic and longevity-related processes in the adult brain and show that it is thus a neuronal miRNA that links nutrient availability, metabolic homeostasis, and lifespan regulation.

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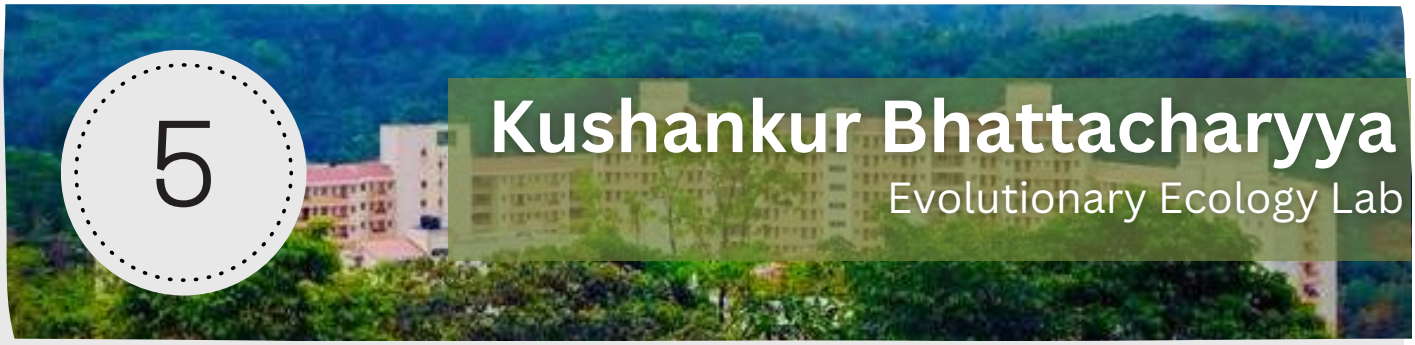
Delvin K Pauly

Cytoskeletal and cell division laboratory

Actions of a motor and a non-motor kinesin in controlling chromosomal errors

Maintenance of stable yet dynamic microtubule attachment with the kinetochores of chromosomes is fundamental to error free chromosome segregation and genetic stability during mitosis. While, the initial lateral attachment of the kinetochores to the microtubule wall *via* the molecular motor, Centromere Protein E (CENP-E) enables chromosome movement from the spindle-pole area to the spindle midzone, the final end-on attachment additionally requires action of another centromere associated non-motor kinesin Kif2C, which depolymerizes the erroneously attached microtubules to ensure fidelity of the attachment. Mechanism of how these two actions are molecularly coupled remains unclear. Our findings reveal that depletion of CENP-E contributes to erroneous attachments of kinetochores with concomitant aggravation of microtubule density near the kinetochores. Interestingly, CENP-E depletion also leads to significant loss of Kif2C from the inner centromere region and a moderate loss of Aurora B kinase, which stabilizes kinetochore localization of both Kif2C and CENP-E *via* phosphorylation. A similar Kif2C loss appears evident in cells expressed with a CENP-E mutant that is deficient of phosphorylation by Aurora B kinase. Possible link of these findings with microtubule stabilization will also be presented.

IISER THIRUVANANTHAPURAM



5

Kushankur Bhattacharyya
Evolutionary Ecology Lab

Unveiling the facets of egg cannibalism in a tropical butterfly

Though there are a large array of insects known to cannibalise under different biotic and abiotic conditions, cannibalism is rather rare among herbivorous insects. We have explored egg and pupal cannibalism in a widespread tropical butterfly species *Danaus chrysippus*. The initial larval stages were observed to cannibalise on eggs, and advanced larval stages on freshly formed pupae under density-dependent or independent conditions. We hypothesised that egg cannibalism is higher when food is scarcer. In this experiment, we offered Ist instar larvae, eggs along with three food levels: very low, low and *ad libitum*. We predicted that cannibalism rate will be highest in the very low food group and lowest in the *ad libitum* group. We found that the rate of cannibalisation was significantly lower in the *ad libitum* group than in the two other groups, although there was no difference between the very low and low groups. In another experiment, we provided eggs and leaves to each larvae, with one group of larvae provided tender (optimal), and another, mature leaves (suboptimal). As predicted, the rate of cannibalism was significantly higher in the mature leaf group. In the third experiment we provided eggs and *ad libitum* food under low, medium and high larval density. The rate of cannibalism was higher in high and medium larval density as predicted. This study shows that egg cannibalism is indeed higher when food quality or quantity is suboptimal, or there is increased competition due to higher larval density. Our results suggest that cannibalism is a facultative strategy during periods of extreme stress or higher competition. Cannibalistic individuals might gain edge by having access to higher food quantity, supplementing their nutritional demands leading to increased survival. It also opens up scopes for studying detailed behavioural repertoire of cannibalistic individuals and other conditions leading to cannibalism like kin recognition or alkaloid sequestration.

Keywords: *first instar, food scarcity, suboptimal food, larval density*

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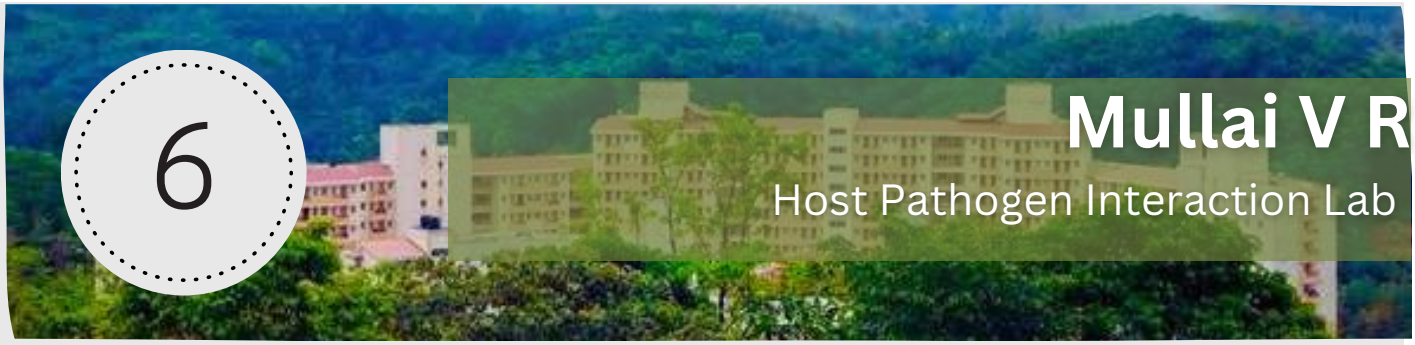

6

Mullai V R

Host Pathogen Interaction Lab

Regulation of cytokine expression by intracellular pathogens

Many pathogens have evolved to evade immune responses and establish a secure replicative niche inside host cells. Yet, at a systemic level, immunocompetent hosts often restrict pathogens through cell-mediated and cell-autonomous immunity. One such stealth pathogen is the obligate intracellular bacterium *Coxiella burnetii*, the causative agent of Q-fever, which translocates an array of bacterial proteins ('effectors') into the host cell through a type IVB secretion system (T4BSS). However, chemotactic cytokines, CXC-ligands, are considered biomarkers of Q-fever (Jansen et al., 2017), raising the question how chemokines get induced and regulated during intracellular bacterial infection. Using *C. burnetii* as a model system, we observed minimal to no CXCL10 mRNA transcript levels during Coxiella infection. However, Coxiella-infected cells robustly augmented IFNγ-activated CXCL10 expression to several fold, both at mRNA and protein level. Although CXCL10 was used as representative cytokine, this phenomenon was observed for few other cytokines and CXCL-chemokines and was dependent on Coxiella viability and functional T4BSS, since neither T4BSS-deficient nor heat-killed/ PFA-fixed Coxiella could augment CXCL10 levels in IFNγ-activated cells. Coxiella and IFNγ-induced synergistic expression of CXCL10 is transcriptionally regulated by its promoter. The specificity of this phenotype, molecular mechanisms, innate immune signaling pathways involved are currently being investigated along with its functional implications on T cell recruitment and cell-intrinsic pathogen load in infected cells through transwell migration assays. Overall, this study aims to uncover mechanisms by which pathogens modulate cytokine synthesis, secretion and downstream effects and its relevance on pathophysiology.

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7

Pravin B
SIM.BIO.SYS Lab

Synthetic Lethality Prediction via Statistical and Network Modelling

Synthetic lethality (SL) arises when the simultaneous perturbation of two genes results in cell death, whereas perturbing either gene alone has little to no effect. A well-known and clinically validated example occurs in breast cancer cells harboring BRCAI/2 mutations, where inhibition of PARPI/2 leads to the selective elimination of tumor cells. Despite the conceptual foundation of SL being nearly 80 years old, this remains the only widely successful therapeutic implementation, introduced almost two decades ago.

Traditional strategies for discovering SL interactions, such as double-knockout assays or genome-wide perturbation screens, are exhaustive, expensive, and experimentally demanding. Consequently, there has been a major shift toward computational approaches capable of predicting these complex and context-dependent interactions. Several modern algorithms, including SLMGAE, GCATSL, and KG4SL, integrate graph-based knowledge with gene annotation data to infer SL pairs. However, these models are constrained to predicting interactions only within the boundaries of existing networks. Furthermore, most are optimized primarily for accuracy rather than precision, an important limitation, as false positives are unacceptable in clinical applications.

To address these challenges, we propose SLxGO, a neural-network–driven framework that infers network-topology features directly from Gene Ontology (GO) annotations using a reference PPI network. By leveraging BioBERT to embed GO terms, the neural network effectively learns synthetic topology features. These GO-derived embeddings, combined with synthetic features for a gene pair, are provided to a gradient boosting (GB) model that performs the final SL prediction.

We benchmarked SLxGO against 8 well-established algorithms. While classification performance showed consistent improvement, the model exhibited tremendous gains in ranking metrics, highlighting its ability to prioritize biologically meaningful SL candidates. Importantly, SLxGO generalizes well to unseen gene combinations, demonstrating strong robustness. We are currently working to incorporate cell-line–specific information, which remains challenging due to limited data availability. Thus far, predictions across six cell lines (HeLa, Jurkat, 293T, K562, A375, and A549) have been generated, and we aim to release these through a publicly accessible database(SLiGO). Our ongoing efforts focus on expanding cell line coverage as well as extending the framework into a cross-species SL prediction pipeline, with particular emphasis on pathogens such as *Plasmodium*.

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8

Riniya Najeeb
HapGen Lab

Understanding the Role of NASP in CENH3 Deposition and Genome Stability in *Arabidopsis thaliana*

Centromeres are essential regions of eukaryotic chromosomes responsible for the formation of kinetochore complexes that connect to spindle microtubules during cell division. This ensures faithful chromosome segregation, which is fundamental for genome stability. In plants, the centromere is defined by the histone H3 variant CENH3, whose precise deposition onto centromeric chromatin is essential for maintaining centromere identity and genome integrity. The histone chaperone NASP (Nuclear Autoantigenic Sperm Protein) has been established as a key factor mediating CENH3 loading in *Arabidopsis thaliana*; however, the precise molecular mechanisms by which NASP facilitates this process remain poorly understood. Understanding the role of NASP as regulators of centromere function and genome stability, providing new insights into the plant-specific machinery that governs CENH3 loading

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9

Ritobrato Chatterjee

Laboratory of Immune Cell Biology (LICB)

The endosomal ubiquitin ligase CARP2 is a novel regulator of primary cilia assembly

Organelles are membrane-bound compartments that dynamically interact with one another and respond to the environment within and outside the cell, which is crucial for maintaining homeostasis. The primary cilia is one such antenna-like sensory organelle that acts as a signalling hub in mammalian cells. The Cilia formation plays important roles in diverse physiological processes, including cell division, migration, tissue differentiation and innate immunity. Impaired cilia assembly or biogenesis leads to dysregulated signalling, resulting in various ciliopathy-linked pathologies such as Bardet-Biedl syndrome, Joubert syndrome, Meckel-Gruber syndrome, Nephronophthisis, polycystic kidney disease, etc. The ciliary assembly entails post-translational modifications, such as ubiquitination, and the trafficking of modified proteins through the Golgi complex. The architecture of the Golgi, another dynamic organelle, is reported to be important for the primary cilia assembly. Knocking down Golgi resident proteins like the Giantins suppresses ciliation in zebra fish. We established CARP2/RFFL, an ubiquitin ligase, as a novel regulator of cilia assembly. Cells without CARP2 (knockout) expression showed reduced ciliation. Reconstitution of CARP2 KO cells with wild-type, but not E3-inactive or endosomal association-defective, variants of CARP2 restored the phenotype, indicating the requirement of ubiquitin ligase activity and endosomal association for these processes. Importantly, we also found that CARP2 is an interacting partner of TRIM32 (Tripartite motif containing 32), a mutation in which has been reported in some BBS (Bardet-Biedl syndrome) patients with ciliopathy. We demonstrated that the pathogenic variant (P130S) of TRIM32, but not wild-type TRIM32, increases cilia length. In summary, CARP2 positively regulates cilia assembly, and its interaction with TRIM32 is critical for cilia homeostasis.

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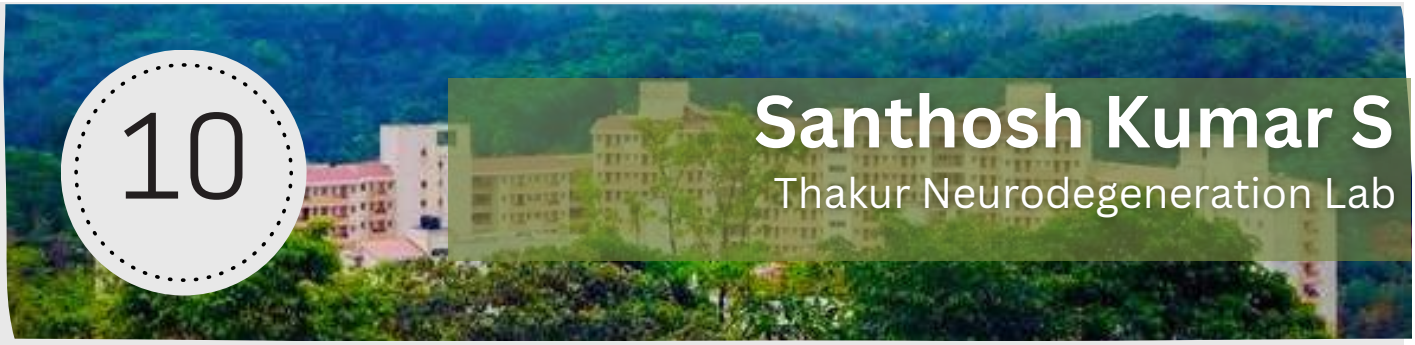

10

Santhosh Kumar S
Thakur Neurodegeneration Lab

α-Synuclein-based mouse model of Parkinsons disease displays neuroprotection upon PD180970 Administration

Parkinson's disease (PD) is a progressive neurodegenerative disorder characterized by the loss of dopaminergic neurons (DA) of substantia nigra (SN) and accumulation of α-synuclein aggregates in lewy bodies. A model that closely mimics the relevant pathological signatures is necessary to understand the disease progression and underlying mechanisms. We developed a model by combinatorial injection of AAV6-overexpressing α-synuclein and pre-formed fibrils (PFF) called SynFib model.

The model exhibited significant neurodegeneration accompanied by striatal (STR) projection loss and p-syn positive α-synuclein accumulation at SN and STR compared to relevant control groups (GFP, PFF and Syn). Further, the model exhibited a progressive gait abnormality depicting motor behavioral deficits at 24W. A time-dependent degeneration of SN-DA neurons and fibres of STR was displayed. The model shows progressive worsening of p-syn pathology at SN. A spread beyond the dopaminergic system was also evident in the medium spiny neurons (STR) and cortical regions. We observed lewy-like features of aggregates which were proteinase K-resistant, highly ubiquitinated and thioflavin-S positive. Further the model showed an elevation of neuroinflammation that reduced with time with loss of neurons. Interestingly, the aggregates spread contralaterally, which elevated ubiquitin puncta structures and were colocalizing with microglia. The model was used to evaluate the efficacy of a c-Abl inhibitor, PDI80970 (5mg/kg i.p. per day) whose administration showed neuroprotection, reduced aggregate accumulation and neuroinflammation.

The SynFib model captures various pathological signatures of PD that may be useful in studying mechanisms driving disease over time and the model displays its usefulness in pre-clinical therapy.

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11

Vaisak Mohan

Laboratory of Parasitic Diseases(LaPD)

Apicomplexan Parasites Remodel Host ER Membrane Contact Sites to Create Ceramide Acquisition Platforms

Apicomplexan parasites including *Plasmodium spp.* and *Toxoplasma gondii* depend on host-derived lipids for rapid replication within the parasitophorous vacuole (PV). We discovered that these parasites actively remodel and tether to host ER membrane contact site (MCS) machinery to acquire ceramide, a critical sphingolipid precursor. In host cells, ER-synthesized ceramide is transported to the Golgi via MCS formed between ER-resident VAP-A/B proteins and the ceramide transport protein CERT. We hypothesized parasites hijack this spatially organized pathway.

We utilized click-chemistry to tag sphingosine (dI8:I) - precursor in ceramide pathway and fluorescent tagged BODIPY-ceramide, for exogenous addition *in vitro*., Using these, we visualized robust ceramide accumulation at the PV membrane (PVM) and parasite plasma membrane. This uptake was reduced upon CERT knockdown and HPA-I2 inhibition, establishing CERT-dependent trafficking. We demonstrate that parasites dynamically reorganize host MCS proteins around the PVM. VAP-B shows strong sustained enrichment at the PVM throughout infection. VAP-B knockout alone reduced *T. gondii* development as confirmed by flow cytometry/imaging, while VAP-A knockdown produced similar defects—both without affecting invasion rates. CERT knockdown also significantly impaired development.

Proximity labeling using miniTurbo:VAP-B identified parasite proteins with VAP-binding domains and PI(3,4)-kinases, suggesting parasites instruct PI4P production at the PVM, mimicking Golgi membrane lipids for CERT recruitment. Fluorescently-tagged CERT localizes to the PVM in a PH domain-dependent manner. Strikingly, the PH domain alone retains PVM association, indicating parasites present a phosphoinositide-rich platform actively redirecting CERT from its Golgi destination. Cross-species validation confirming CERT inhibition impairing *Plasmodium yoelii* liver-stage development demonstrates a conserved apicomplexan strategy.

We are targeting identified *Toxoplasma* interactome members using CRISPR-based inducible degradation to functionally target ceramide acquisition mechanisms. This work reveals how parasites spatially remodel host membrane contact sites to create sphingolipid acquisition sites—exposing conserved druggable targets across apicomplexan pathogens.

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Vismaya Prakash
Chronobiology Laboratory

When Flies Choose the Night: Understanding the Role of microRNA-9b in Circadian Rhythms

Due to Earth's 24-hour rotation, organisms experience predictable changes in environmental temperature and light, leading to the evolution of endogenous circadian clocks. These biological clocks, driven by genetic mechanisms and molecular oscillators, maintain a ~24-hour oscillation even in constant conditions, allowing organisms to anticipate daily environmental changes. In *Drosophila melanogaster*, circadian rhythms are primarily controlled by specialized circadian pacemaker neurons, where the internal clock is regulated by a transcriptional-translational feedback loop. Interestingly, certain clock gene product proteins peak several hours after their mRNA levels, suggesting post-transcriptional regulation. MicroRNAs (miRNAs), small non-coding RNA molecules, regulate gene expression by binding to target mRNAs, leading to their degradation or inhibiting translation. miRNAs have been shown to play a key role in modulating circadian rhythms in *Drosophila*.

In this study, we focused on less abundantly expressed miRNAs conserved between humans and *Drosophila* and identified miR-9b as a strong modulator of locomotor activity rhythms in *Drosophila melanogaster*. Pan-neuronal overexpression of miR-9b using *elav*-GAL4 induced a pronounced nocturnal phenotype, with suppressed activity during the light phase and increased activity in darkness. Overexpression in clock neurons using *clk*856-GAL4 resulted in elevated nocturnal activity without a complete behavioral phase reversal. Under constant darkness (DD), miR-9b overexpression with both *elav*-GAL4 and *clk*856-GAL4 caused severe circadian disruption, with over 50% of flies becoming arrhythmic and the remaining flies exhibiting weak rhythmicity. Bioinformatic analysis indicates that miR-9b targets genes involved in neural development, cytoskeletal organization, axon guidance, and neurotransmitter release, suggesting that miR-9b influences circadian rhythms by modulating neuronal structure or synaptic function. Future directions will focus on determining the mechanisms underlying the observed activity phase shift and assessing how light influences circadian behavior in miR-9b manipulated flies.

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CAMPUS MAP

Credits: Ravikiran Thalikal, Smriri Mahesh, Aarul Jain, Ecological Society of IISER TVM

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A LINUS JERUSHA

ANUGRAHA M

ANUSHKA TYAGI

APOORVA SINGH

EESHANI ABHYANKAR

HANA LUKMAN

JANVI JAYACHANDRAN

JYOTI SINGH

JYOTIRMAYI

KAVITHA M S

MALAVIKA M

MARYAM THOMAS

MEENAKSHY A S

MEGHA V V

MONAMI SARKAR

MUHAMMED HADI

NANDANA RAJEEV NAIR

PREMA MONDAL

PAARTH MANOJ

PRASHANSHA KORI

RENUKA MANOJ

RINIYA NAJEEB

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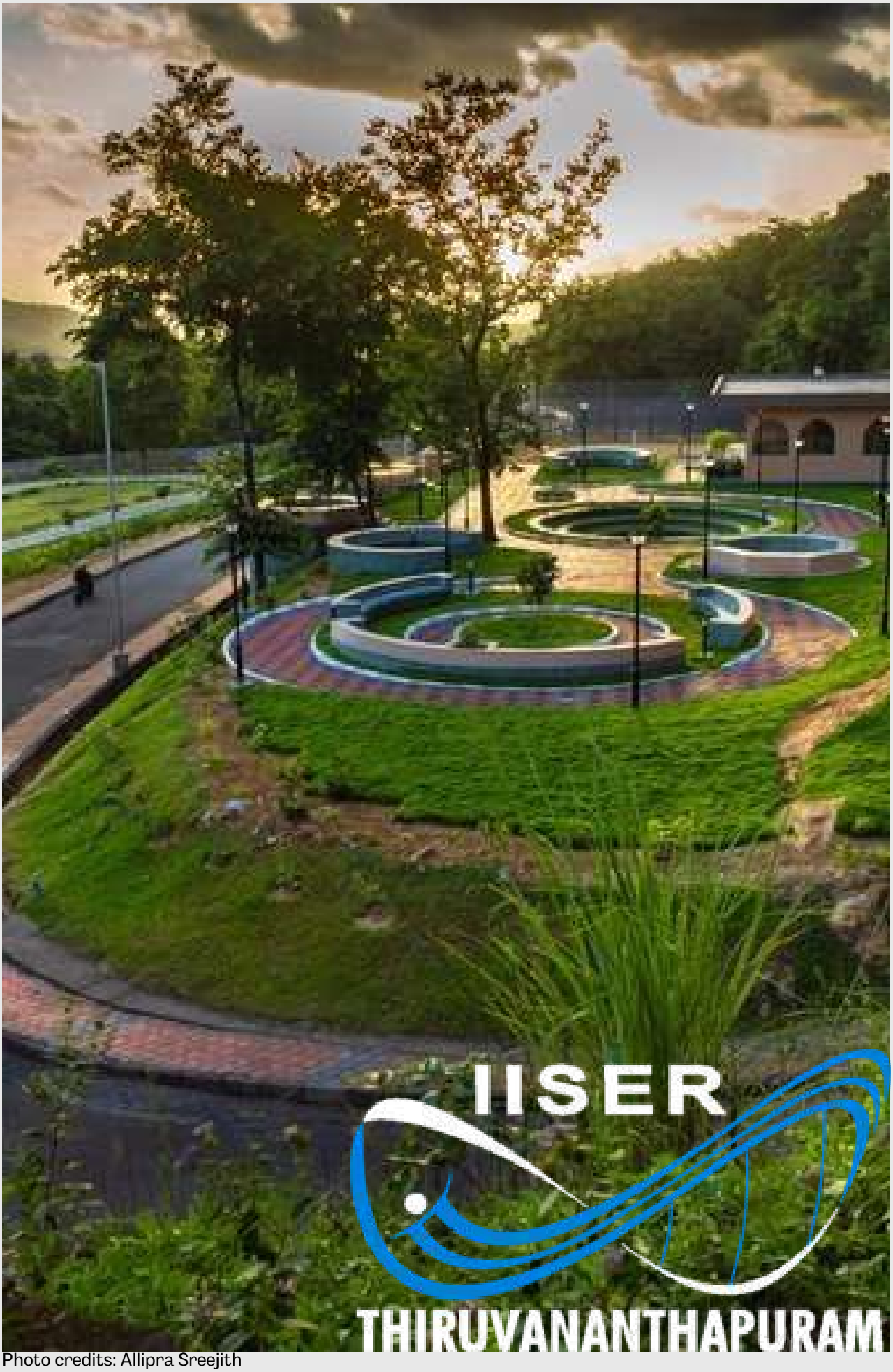

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