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NCCS
National Centre for
Cell Science

सावित्रीबाई फुले पुणे विद्यापीठ
Savitribai Phule Pune
University
(Formerly University of Pune)

6th NCCS STUDENT SYMPOSIUM 2026

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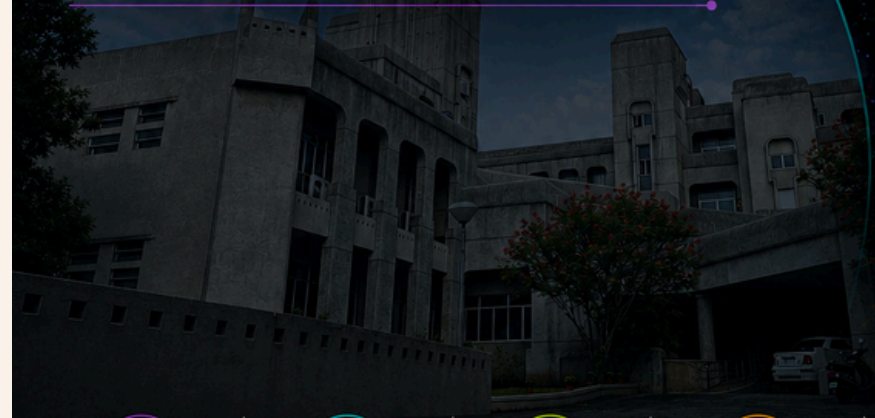
BOOK OF ABSTRACTS



ORAL PRESENTATIONS

POSTER PRESENTATIONS

INVITED TALKS

NETWORKING SESSIONS

SCIENCE COMMUNICATION

"Of the Students, By the Students, For the Students"

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About the Student Symposium

Of the Students, By the Students, For the Students

The NCCS Student Symposium is a scientific event conceived, organized, and conducted by the graduate students of the National Centre for Cell Science (NCCS), Pune. Now in its sixth edition, the symposium will be held on 23rd–24th July 2026 at the NCCS campus.

The symposium serves as a vibrant platform for UG and PG trainees, PhD scholars, postdoctoral researchers, and researchers supported through extramural projects working in diverse areas of cell biology and biotechnology. It aims to foster scientific exchange by providing young researchers with an opportunity to present their work, engage in meaningful discussions, and build collaborations with peers and experts from across the country.

The two-day event will feature invited lectures by distinguished scientists, student oral presentations, and poster sessions, showcasing cutting-edge research spanning multiple disciplines of modern biology. In addition, this year's symposium includes a special interactive session on effective science communication, designed to equip young researchers with skills to communicate their science with clarity and impact.

Beyond scientific presentations, the symposium offers participants a unique opportunity to interact with leading researchers, exchange ideas, and gain valuable insights into current advances in the life sciences. Through this initiative, we strive to cultivate scientific curiosity, encourage interdisciplinary learning, and inspire the next generation of researchers.

We warmly welcome all participants to the 6th NCCS Student Symposium 2026 and look forward to two days of insightful discussions, collaborative learning, and scientific excellence.

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NCCS
NATIONAL CENTRE FOR CELL SCIENCE

6th NCCS SYMPOSIUM 2026

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EVENT RUNDOWN

23rd JULY 2026

Venue: GARGI auditorium

08:00-08:50 AM

Registration
Attendee check-in, Welcome kit distribution .

Session - 1

09:00-09:20 AM

Inauguration
Welcome address by Director, NCCS-Pune

09:20-09:55 AM

Plenary Talk 1- Dr.Saikat Chowdhury (CCMB)
Molecular switches that regulate actin cytoskeleton nucleation.

09:55-10:10 AM

Student Talk 1- Aswathy L B
Phase separation of a structured scaffold protein: insights from Nup188.

10:10-10:25 AM

Student Talk 2- Meghadeepa Sarkar
Regulated membrane fission by Dynamin2 and EHDs dictates retrograde transport fidelity.

10:25-10:40 AM

Student Talk 3- Pooja Gulhane
Engineering regulatory networks of miR-520c-3p in non-small cell lung cancer.

23rd JULY 2026

10:40 – 10:55AM	**Tea Break**
10:55-11:30 AM	**Plenary Talk 2- Dr. Pratik kumar (NCBS)** Genetically targeted dyes for inducing and imaging protein proximity.
11:30-11:40 AM	**Sponsor Talk 1- Hycosmid**
11:40-11:55 AM	**Student Talk 4- Akshay S. Lonare** Regulation of Calcium Homeostasis and Autophagy by Nup358.
11:55-12:10 PM	**Talk 5- Ambuja Navalkar** Order- disorder transitions of protein assemblies in Cancer pathogenesis.
12:10 – 12:25 PM	**Student Talk 6- Asmita Dogra** Tollo/Toll8 signalling in peptidergic neurons drives lipid accumulation in response to mild oxidative stress in Drosophila.
12:25-1:55 PM	**Lunch Day -1**
Session - 2	
1:55-3:55 PM	**Poster session - Day 1**
3:55-4:30 PM	**Plenary Talk 3- Dr. Rashna Bhandari (CDFD)** Polyphosphate - an ancient polymer in modern cell biology.
4:30-4:40 PM	**Sponsor Talk 2- Merck**
04:40-5:00 PM	**Photo Session day 1**
5:00 PM onwards	**High tea -Day 1**

24th JULY 2026

Session - 1

08:00-09:00 AM
Breakfast
Venue- NCCS canteen

09:00-09:15 AM
Student Talk 7- Spriha Ghosh
B cell response to Nipah viral vaccine candidate.

09:15- 09:30AM
Student Talk 8- Surabhi Sharma
Understanding the role of Clathrin light chains in regulating mitochondrial function during early development.

09:30-10:05 AM
Plenary Talk 4- Dr. Amrutha Swaminathan (IISER Tvm)
How tiny fish help us understand complex behaviours

10:05-10:20 AM
Student Talk 9- Shubham Yadav
Understanding the Gαo-mediated signalling pathway using the *C.elegans* model system.

10:20 – 10:35 AM
Tea Break

10:35-11:10 AM
Plenary talk 5- Dr. Anirban Banerjee (IIT-B)
Detect-Dismantle-Dispose: An emerging framework for cell autonomous immunity.

11:10-12:40 PM
Poster session - Day 2

12:40-2:00 PM
Lunch - Day 2

Session - 2

2:00 PM-2:15 PM
Student Talk 10- Karan Selarka
Gbb is essential for maintaining Germline Stem Cells during development.

02:15-02:25 PM
Sponsor Talk 3- Leica

24th JULY 2026

02:25-02:40 PM	**Student Talk 11- Praneet Wahi** CCR9+ dendritic cells control the central tolerance and gut inflammation.
02:40- 4:40 PM	**Science Communication Session** **Dr. Sherline Pimenta (Flame University)**
04:40-04:50 PM	**Concluding remarks and Prize distribution**
04:50-5:00 PM	**Photo Session Day 2**
05:00PM onwards	**High-tea Day 2 (open to all)**

Invited Speakers

Molecular switches that regulate actin cytoskeleton nucleation

Saikat Chowdhury

Principal Scientist, CSIR-Centre for Cellular and Molecular Biology, Hyderabad, Telangana, India
Academy of Scientific and Innovative Research (AcSIR), Ghaziabad, Uttar Pradesh, India

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Abstract

The actin cytoskeleton is a dynamic filamentous network essential for processes such as cell migration, environmental sensing, shape maintenance, and vesicular trafficking. Actin filament assembly begins with a rate-limiting nucleation step, which is strongly accelerated by Arp2/3 complex. While Arp2/3 complex is indispensable for branched actin network formation, it remains inactive without stimulation by nucleation promoting factors (NPFs). Precise spatiotemporal control of actin nucleation is therefore achieved through regulation of these NPFs.

The Wiskott–Aldrich Syndrome Protein and SCAR Homolog (WASH) is the principal endosomal NPF, driving the recycling of internalized proteins back to the cell surface to maintain membrane protein homeostasis and regulate signaling. WASH functions as part of the large, multi-subunit WASH Regulatory Complex (SHRC). Despite its central role, the mechanistic regulation of WASH within SHRC has remained poorly understood due to the lack of high-resolution structural information on the complex or its subunits.

To address this, we performed high-resolution electron cryo-microscopy (cryo-EM) structural studies to resolve the architecture and conformational dynamics of SHRC. We obtained structures of SHRC in multiple states, revealing that WASH intrinsically toggles between nucleation-competent and inactive forms. This intrinsic basal activity distinguishes WASH from canonical NPFs with complex regulatory control. Biochemical assays validated these structural findings, while molecular dynamics simulations provided mechanistic and thermodynamic insights into SHRC inhibition and activation. Our results establish a structural and mechanistic framework for SHRC regulation, setting the stage for future studies of disease associated mutations and additional cellular factors that modulate WASH activity.

Polyphosphate – an ancient polymer in modern cell biology

Rashna Bhandari

Scientist, BRIC-Centre for DNA Fingerprinting and Diagnostics (CDFD), Hyderabad

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Abstract

Inorganic polyphosphate (polyP), a linear chain of phosphate residues linked by high-energy phosphoanhydride bonds, abounds in prokaryotes and unicellular eukaryotes as an energy store, phosphate reservoir, and stress regulator. Mammals have lower amounts of polyP, which is especially enriched in mitochondria and lysosome related organelles. Mammalian polyP supports coagulation, inflammation, mitochondrial homeostasis, yet its metabolism remains enigmatic. In budding yeast, polyP synthesis is allosterically activated by the inositol pyrophosphate 5-InsP7, an ATP-sensitive energy sensor made by IP6K1. Mice lacking IP6K1 show depleted platelet polyP and impaired blood clotting. Our work reveals that the synthesis of polyP in mammalian mitochondria requires active FoF1 ATP synthase, an intact proton gradient, and orthophosphate (Pi). IP6K1 regulates mitochondrial polyP synthesis via 5-InsP7 and catalytic-independent mechanisms. IP6K1-deficient cells and mice show depleted mitochondrial polyP and respiration defects. Expression of active, but not inactive IP6K1, restores mitochondrial polyP levels, while mitochondrial respiration is rescued by IP6K1 expression independent of its catalytic activity. In RBL-2H3 mast cells, isolated granules synthesize ATP-dependent polyP, requiring an intact membrane potential; IP6K1 loss impair granule acidification and polyP levels. These findings link IP6K1 and its product 5-InsP7 to polyP homeostasis in mitochondria and vesicles, unveiling multiple roles for this ancient polymer in mammalian cell biology.

Genetically targeted dyes for inducing and imaging protein proximity

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Pratik Kumar

Abstract

Controlling the permeability of small molecules across cellular membranes while ensuring they reach their intended protein target remains a major challenge. We developed a simple platform that addresses this challenge by using silicon rhodamine dyes as linkers. These dyes maintain good cell permeability while connecting genetic tags to functional small-molecule cargos. By pairing these dye ligands with enzyme-based self-labelling tags, we can target specific intracellular proteins inside living cells. These reagents serve two purposes: they enable high-resolution fluorescence imaging and allow us to manipulate the target protein biochemically. We demonstrate two applications of this induced proximity strategy. First, a biotin-conjugated dye that recruits streptavidin beads to pull down specific intracellular proteins and organelles. Second, a JQ1-conjugated dye that drives and tracks protein translocation to endogenous targets in real time. Ultimately, this design strategy provides a practical framework for developing next-generation chemical tools to explore complex
biological systems.

How tiny fish help us understand complex behaviours

Amrutha Swaminathan

Assistant Professor
Indian Institute of Science Education and Research
Thiruvananthapuram

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Abstract

Zebrafish have been used over the past several decades to further our understanding of how molecular pathways and cellular movements contribute towards early vertebrate development owing to the advantage of external fertilization and development. It is only more recently that early zebrafish embryos and larvae are beginning to be employed to understand how different behaviours are established. Zebrafish larvae begin to show complex behaviours early in life since they grow in the wild in the absence of parental care unlike young mammals. Further, the transparent nature of zebrafish larvae also facilitates the study brain activity in live animals while they demonstrate these complex behaviours. In my talk, I will discuss how we have used zebrafish to understand the establishment of complex innate behaviours such as stress and sleep. Using a novel behavioural paradigm, we established that some individuals are more resilient to stress than others from early on in life, and this ability is regulated by the innate immune system. In the last part of my talk, I will describe how transient alterations in brain activity cause sleep fragmentation in developing zebrafish larvae.

Detect-Dismantle-Dispose: An Emerging Framework for Cell Autonomous Immunity

Anirban Banerjee

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Abstract

A central challenge in immunity is how host cells detect intracellular bacterial pathogens, eliminate them efficiently, and prevent collateral damage arising from the infection itself. Using Streptococcus pneumoniae as a model, our work has uncovered a ubiquitin-driven host defense pathway that enables cells to recognize, dismantle, and safely clear intracellular bacteria. We discovered that host cells exploit degron-mediated ubiquitination of bacterial surface proteins to selectively tag invading bacteria for destruction. These ubiquitin marks recruit the AAA+ ATPase VCP/p97 to the bacterial surface, where inflammatory signaling promotes its assembly into bacteriolytic clusters that destabilize the bacterial envelope and trigger pathogen rupture. This reveals a previously unappreciated role for mechanical force as an effector arm of cell-autonomous immunity. Bacterial destruction, however, creates a second challenge: the persistence of microbial remnants that can provoke damaging inflammatory responses. We show that rupture of the bacterial membrane exposes cryptic signals on the bacterial corpse that drive its selective capture by autophagosomes. Failure of this clearance pathway results in the accumulation of bacterial remnants, inflammasome activation, and pyroptotic cell death. Together, these findings support an emerging framework of Mechanical Immunity, in which ubiquitin-dependent pathogen recognition is coupled to force-mediated bacterial destruction and autophagic corpse clearance. This work highlights how host cells integrate microbial sensing, physical antimicrobial action, and post-killing homeostasis to mount effective defense against intracellular infection.

Speak So They Understand: Practical Communication Skills for Scientists

Professor, Flame University, Pune

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Sherline Pimenta

Session overview

Effective communication is essential for researchers seeking to share ideas, present findings, and build professional networks. This highly interactive workshop equips participants with practical tools to communicate their research clearly and confidently. Through hands-on activities and peer feedback, participants will learn to explain complex concepts to diverse audiences, structure compelling research narratives, and engage meaningfully in academic and professional conversations. Designed as a communication studio rather than a lecture, the workshop emphasises practice, reflection, and immediate application, helping researchers transform technical expertise into impactful and memorable communication.

Student talks

1 Aswathy L B

National Centre for Cell Science, Pune

Phase separation of a structured scaffold protein:insights from Nup188

Nup188 is a core scaffold nucleoporin essential for nuclear pore complex (NPC), architecture, yet its biophysical behaviour beyond this structural role remains poorly understood. Although the protein consists of only 5.4% intrinsically disordered residues, bioinformatic analysis revealed a localized phase-separation propensity within its C terminal domain, suggesting a basis for condensate formation independent of classical disordered regions. In line with this prediction, purified Nup188 spontaneously underwent liquid-liquid phase separation in vitro, while live-cell imaging showed that overexpression produced distinct intracellular puncta. These puncta displayed hallmark liquid-like behaviour, confirmed by FRAP, sensitivity to 1,6-hexanediol, and fusion-fission dynamics. Using the flickerprint approach, we further showed that the interfacial tension and bending rigidity of these condensates fall within the range reported for established biomolecular condensates. Additionally, these condensates were morphologically distinct from annulate lamellae, pointing to a potential function independent of the NPC. Strikingly, introducing the native binding partner Nup93 caused the condensates to dissolve in cells, suggesting that normal complex assembly helps regulate condensate stability. Together, these results show that a large, structured, predominantly α-helical nucleoporin can phase-separate through a distinct mechanism, expanding the known functional range of nucleoporins beyond their canonical role at the nuclear pore complex.

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2 Meghadeepa Sarkar

Indian Institute of Science Education and Research, Pune

Regulated membrane fission by Dynamin2 and EHDs dictates retrograde transport fidelity

Cellular fitness relies on retrograde trafficking to return vital cargo from endosomes to the Trans-Golgi Network (TGN). While the initiation of this pathway via retromer-mediated tubulation is well documented, the downstream fission machinery has long eluded detection. This work identifies the large GTPase Dynamin2 (Dyn2) as the definitive driver of this membrane fission step. We show that Dyn2 does not act alone; its activity is tightly calibrated by EHD family ATPases. By balancing each other, Dyn2 and EHDs enforce an optimal rate of tubule cleavage, guaranteeing that cargo reaches its destination accurately and efficiently.

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3 Pooja Gulhane

National Centre for Cell Science, Pune

Engineering Regulatory Networks of miR-520c-3p in Non-small cell lung cancer: A Therapeutical Approach

Lung cancer is the foremost cause of cancer-related mortality worldwide, claiming nearly 1.79 million lives annually, with non-small cell lung cancer (NSCLC) accounting for 85% of cases and showing poor survival outcomes under current therapies. Mounting evidence indicates that microRNAs (miRNAs) are dysregulated in NSCLC, acting as either tumor suppressors or oncogenes by regulating cancer hallmarks and modulating immune cells within the tumor microenvironment (TME). In this study, we constructed a miRNA–target inter-regulatory network and identified enriched pathways, highlighting hsa-miR-520c-3p as a key regulator involved in EGFR signaling and immune signaling axes. Structural and functional assays demonstrated that miR-520c-3p directly targets AKT1 and EGFR, suppressing proliferation, migration, and colony formation in NSCLC cells, thereby underscoring its tumor-suppressive role. To further investigate its immunomodulatory potential, we developed a computational model using MATLAB simbiology(2020b) and validated predictions through statistical analysis, western blotting, and wound healing assays. These findings emphasized the pivotal role of the PI3K–AKT pathway in shaping the NSCLC microenvironment, particularly in influencing tumor-associated macrophage polarization. Employing a synthetic biology approach, we designed a miRNA construct to precisely modulate miR-520c-3p expression, enabling deeper exploration of its role in tumor–immune crosstalk. Collectively, our results suggest that modulating miR-520c-3p expression represents a promising therapeutic strategy in NSCLC, with the potential to reprogram both cancer cell signaling and immune interactions within the TME, ultimately offering new avenues for improving patient outcomes

4 Akshay S. Lonare

National Centre for Cell Science, Pune

Regulation of Calcium Homeostasis and Autophagy by Nup358

Nucleocytoplasmic transport (NCT) is mediated by nuclear transport receptors and the Ran GTPase through interactions with nucleoporins of the nuclear pore complex (NPC). Although the mechanisms governing transport across the nuclear envelope are well characterized, how NCT coordinates with cytoplasmic signaling pathways remains poorly understood. Interestingly, a subset of nucleoporins, including the RanGTP-binding nucleoporin Nup358/RanBP2, localizes to cytoplasmic annulate lamellae (AL), specialized ER subdomains whose physiological functions are largely unexplored.

Here, we show that AL are frequently positioned at ER–mitochondria contact sites (ERMCSs), key hubs for calcium signaling, mitochondrial metabolism, and autophagy. We identify a previously unrecognized role for AL-resident Nup358 in regulating ERMCS-mediated calcium homeostasis. Depletion of Nup358 increases ER–mitochondria connectivity but paradoxically impairs functional calcium transfer, leading to reduced mitochondrial calcium levels and elevated cytosolic calcium. This disruption compromises mitochondrial respiration, and elevated cytosolic calcium activates autophagy through the CaMKK2–AMPK signalling pathway.

Mechanistically, Nup358 promotes the interaction of GRP75 with IP3R and VDAC, facilitating assembly of the functional IP3R–GRP75–VDAC calcium transfer complex at the ERMCSs. Loss of Nup358 disrupts this complex, leading to defective mitochondrial calcium uptake and activation of a compensatory stress response. Ongoing studies focus on understanding how RanGTP and nuclear transport receptors regulate Nup358's function in calcium homeostasis.

Together, our findings uncover a novel cytoplasmic function of Nup358 and reveal an unexpected mechanistic link between NCT and ER–mitochondria communication-pathways that are critical for cellular homeostasis and are frequently dysregulated in neurodegenerative diseases and cancer.

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5 Ambuja Navalkar

National Centre for Cell Science, Pune

Order- disorder transitions of protein assemblies in Cancer pathogenesis.

Biomolecular condensates and protein aggregates are emerging as important regulators of cancer-related signaling, transcription, and proteostasis. In tumor biology, oncogenes can exploit condensate formation to assemble transcriptional and signaling hubs that enhance proliferative programs and metabolic adaptation. In contrast, tumor suppressors may show loss-of-function through misfolding, aberrant compartmentalization, or aggregation, thereby weakening genome surveillance and anti-proliferative responses. Our work highlights the balance in these states to be a key determinant of whether these molecular structures support normal regulation or malignant transformation. We examine how altered protein assembly states of cancer-associated proteins influence gene expression, cell fate control, and therapy resistance. We also research the potential of targeting condensate dynamics and aggregation processes as a strategy to restore tumor-suppressive function and inhibit oncogenic signaling. Understanding these mechanisms may provide a broader framework for linking phase behaviour, protein homeostasis, and cancer progression.

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6 Asmita Dogra

National Centre for Cell Science, Pune

Tollo/Toll8 signalling in peptidergic neurons drives lipid accumulation in response to mild oxidative stress in Drosophila

While it is clear that severe stress is highly detrimental, how organisms adapt to mild chronic stressors that are more common is not well understood. We found that chronic exposure to mild oxidative stress induces systemic lipid accumulation in adult Drosophila, which renders them more resistant to starvation. Toll signalling pathways are known to be activated by oxidative stress and are expressed in multiple tissues, including the nervous system. Through a genetic screen, we identified that neuronal Tollo (Toll-8)modulates starvation resistance by affecting lipid accumulation. We narrowed down Tollo activity to a specific small cohort of neuropeptidergic neurons expressing the neuropeptide Leucokinin (LK) amongst others. Upstream Spätzle2 was the ligand responsible for activating Tollo under oxidative stress. Downstream of Tollo, the JNK pathway but not the NFKB pathway, was necessary for lipid accumulation and starvation resistance. Our evidence also suggests that IPCs act downstream of Leucokinin to promote lipid accumulation and starvation resistance. Our results implicate neuronal Toll signaling in adapting to mild oxidative stress by modulating systemic lipid metabolism, in the process lending the flies a greater ability to withstand starvation stress.

7 Spriha Ghosh

National Centre for Cell Science, Pune

B cell response to Nipah viral vaccine candidate

Nipah virus (NiV) is a highly pathogenic virus endemic to Southeast Asia, with outbreaks occurring almost every other year. Although the number of cases remains relatively low, NiV is associated with a high mortality rate and currently lacks vaccines or antiviral treatments. Given its significant pandemic potential, understanding the host immune response to NiV is crucial for developing effective diagnostics and prophylactic strategies. This study investigates B cell responses targeting the NiV fusion (F) and attachment (G) surface glycoproteins, which mediate viral entry into host cells. We engineered a recombinant vesicular stomatitis virus (VSV)-based pseudovirus expressing both NiV-F and NiV-G proteins. This NiV pseudovirus (NiV-PV) serves as both a vaccine candidate and a tool for evaluating humoral immune responses. We aim to characterize the magnitude, durability, and quality of F- and G-specific B cell responses induced by NiV-PV using a range of immunological techniques. These include assessments of germinal center formation, quantification of antibody-secreting cells, and memory B cell generation. The NiV-PV platform is also employed to evaluate neutralizing antibody titers. Furthermore, we examine the role of adjuvants in enhancing the immunogenicity and longevity of the antibody response. Overall, this study provides mechanistic insights into the ability of the NiV-PV platform either alone or in combination with adjuvants to elicit robust, long-lasting, and high-affinity antibody responses against the NiV F and G glycoproteins.

8 Surabhi Sharma

National Centre for Cell Science, Pune

Understanding the role of Clathrin light chains in regulating mitochondrial function during early development.

Clathrin mediated endocytosis is a key endocytic pathway involved in regulating cell fate decisions during early development. The basic unit of the clathrin coat is the triskelion structure made up of three heavy and three light chains. While the clathrin heavy chain is known to maintain pluripotency in embryonic stem cells, the role of the Clathrin light chains in development is underexplored. Previously our lab has made and characterized Clathrin light chain knockout mouse embryonic stem cells (mESCs), which show an altered organization of the actin cytoskeleton along with aberrant expression of differentiation markers (Tiwari et al, 2025). During differentiation, pluripotent stem cells undergo a drastic change in mitochondrial morphology, transitioning from small and fragmented to elongated, tubular networks, which is accompanied by a significant increase in the number of mitochondria in mouse and human embryonic stem cells. We therefore characterized whether mitochondrial function was altered in these knockout mESCs. We observed changes in cellular proliferation rates and mitochondrial cristae organization in mESCs upon loss of specific clathrin light chains. Further, we observed that loss of Clta resulted in more tubular mitochondria, while loss of both Clta and Cltb led to a change in the overall shape of the mitochondria from tubular to donut. We also observed an increase in the number of PDH enriched mitochondria-derived vesicles (MDVs) in Clta-/- mESCs. Golgi-derived vesicles mark mitochondrial fission sites. The presence of clathrin light chain A (CLCa) in the Golgi indicate towards potential novel functions for CLCa in initiating mitochondrial fission. Our results suggest a possible role for Clathrin light chains in regulating mitochondrial morphology during early development.

9 Karan Selarka

MACS-Agharkar Research Institute, Pune

Gbb is essential for maintaining Germline Stem Cells during development.

Germline stem cells (GSCs) reside within a somatic niche and give rise to the gametes essential for species survival. Niche-derived signals, including BMPs and Insulin, tightly regulate GSC division and differentiation, so niche integrity must be preserved through continuous clearance of damaged organelles and misfolded proteins by autophagy, a lysosome-dependent pathway active within the GSC-niche. Our previous work showed that GSC autophagy is tightly controlled: classical inducers such as nutrient stress and rapamycin fail to trigger it, leading us to hypothesize that it is regulated non-autonomously. Knockdown of the BMP ligand Gbb (gbb-KD) in niche cells upregulated autophagy throughout the germarium and reduced 1B1- positive spectrosomes and fusomes, indicating turnover of GSCs and germline cells. To define when Gbb is required, we knocked it down temporally in niche cells using Gal4-Gal80ts. Vasa-positive cells were most abundant after pupal-stage knockdown but scarce after larval (L1) knockdown, while autophagy stayed elevated from L1 through pupal stages relative to age-matched controls. When gbb was depleted only after adult eclosion, Vasa-positive cells persisted through weeks 1–4 despite elevated germarial autophagy. Together, these data indicate that Gbb acts during development, not adulthood, for GSC maintenance and/or specification. How Gbb signalling connects to autophagy remains open, since germarial autophagy rises whenever Gbb is depleted across ovarian development.

10 Shubham Yadav

National Centre for Cell Science, Pune

Decoding the Gαo -mediated signaling pathway: Identification and characterization of downstream effector molecules

G protein-coupled receptors (GPCRs) represent the largest and most diverse class of membrane receptors in animals and signal primarily through heterotrimeric G proteins composed of α, β, and γ subunits. Gα subunits majorly drive intracellular responses and are classified into four major subgroups: Gαs, Gαq, Gα12, and Gαo, each associated with distinct downstream effectors. Gαs activates adenylyl cyclase to elevate cyclic AMP and regulates growth via protein kinase B (PKB), whereas Gαq signals through phospholipase Cβ (PLCβ) and Gα12 through Rho1. In contrast, Gαo, the most abundant G protein in the brain, plays essential roles in neuronal development and differentiation; however, its downstream effectors remain poorly defined despite decades of research. To address this gap, we aimed to identify and functionally characterize candidate Gαo effectors using the C. elegans and mouse model systems. Mass spectrometry-based analysis of mouse and worm lysates generated a set of conserved candidate interactors, which were further refined through RNAi-based functional screening using egg-laying behavior as a quantitative readout. This approach identified the Rho-family GTPase CDC-42 as a strong candidate effector. To assess this interaction in vivo, co-immunoprecipitation (CoIP) assays were performed, which confirmed the interaction between Gαo and CDC-42 protein. To evaluate whether this interaction is direct, both proteins were purified in bacterial systems and subjected to in vitro binding assays under defined nucleotide conditions. CDC-42 preferentially associated with the active, GTPγS-bound form of Gαo compared to the inactive GDP-bound state, indicating nucleotide-dependent interaction. These results support CDC-42 as a promising candidate Gαo effector and provide a basis for further dissection of the pathway. Building on these findings, ongoing and future studies will aim to determine if the Gαo-binding proteins meet the biochemical and genetic criteria expected of an effector, and positive results would be a breakthrough in understanding the neural signalling through Gαo.

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11 Praneet Wahi

National Centre for Cell Science, Pune

CCR9+ dendritic cells control the central tolerance and gut inflammation

Most thymic dendritic cells (DCs) express chemokine receptor CCR9 and bind to its cognate chemokine CCL25, facilitating their localization into specific thymic compartments. The non-chemotactic functions of CCL25-CCR9 signaling in regulating the DCs phenotype and function in the thymus are not known.C57BL/6 mice were given 2% dextran sodium sulfate (DSS; w/v) in drinking water. Cellular and molecular changes were analyzed using spectral flow cytometry, immunohistological staining, and RT-PCR. DSS treatment resulted in significantly increased colitis, body weight loss, and thymic atrophy in CCR9-/- mice compared to wild-type mice. DSS-treated CCR9-/- mice thymus showed a significant increase in the frequency of CD103hiCD8αα+CD11c+ (migratory) and CD103loCD11b+CCR7+CD11c+ (cDC2) cells compared to CCR9+/+ mice. CCR9-/- mice exhibited impaired positive and negative selection and T cell maturation compared to wild-type controls. The adoptive transfer of purified CCR9+/+ DCs into CCR9-/- mice mitigated the severity of DSS-induced colitis and restored thymic T cell development. Additionally, proteomics and RNA sequencing analyses of thymic tissue from CCR9-/- mice revealed dysregulation of several important molecules involved in antigen processing and presentation, energy metabolism, and pathways that control central tolerance.CCL25-CCR9 signaling plays an important non-chemotactic function in thymic DCs. Understanding the mechanisms that govern central tolerance via CCR9+ DCs offers potential strategies for modulating inflammation and autoimmunity.

Poster Presentations

P1-Z1 Abyaktaguru Dash

National Centre for Cell Science, Pune

Decoding the Molecular Impact of Pathogenic CLTC Variants on Clathrin Assembly

Clathrin-mediated endocytosis is a fundamental process in neurons for synaptic vesicle recycling and crucial for proper brain functioning. Pathogenic mutations in the CLTC gene encoding the clathrin heavy chain (CHC) associated with intellectual disability may disrupt this process at multiple levels, thereby affecting vesicle formation dynamics. Currently, 41 patients with 35 de novo heterozygous CLTC variants have been reported worldwide. The precise molecular and cellular mechanisms by which these variants lead to neurodevelopmental defects remain unclear. Hence, we assessed the molecular impact of these variants by using biochemical assays to understand the alteration of clathrin binding and assembly. We utilized in-vitro supported membrane tube (SMrTs) assays, followed by florescence imaging to understand the adaptor mediated clathrin clustering.

The mutant forms of CHC showed reduced clathrin polymerization compared to the wild-type protein. This decrease could be attributed to impaired binding between CHC and adaptor protein, which may reduce efficient recruitment of clathrin to the tubes. Alternatively, the mutations may disrupt adaptor-mediated clathrin clustering, potentially due to weakened interactions between clathrin triskelia. Such disruptions could hinder clathrin polymerization, leading to lower accumulation of CHC on membrane tubes.

In future we propose to generate iPSC lines harbouring clinically relevant CLTC mutations associated with intellectual disability, providing a physiologically relevant human model system. These iPSC lines will then be differentiated into major neural cell types, followed by detailed cellular and molecular characterization to determine the effect of mutations in CLTC on neurodevelopment.

P2-Z2 Aditi Manjare

National Centre for Cell Science, Pune

Establishing a Drosophila Model to Investigate Trauma-Induced Emesis and Gut–Brain Communication

Traumatic brain injury (TBI) is frequently associated with nausea and vomiting in humans, yet the underlying gut-brain mechanisms remain poorly understood. Drosophila melanogaster exhibits conserved regurgitation-like behaviors, including emesis, making it a genetically tractable model for investigating the underlying gut-brain pathways. This study aims to investigate how these pathways contribute to TBI-induced emesis in Drosophila. We first optimized the starvation period to enhance the emetic response in the flies. Flies were then subjected to mechanical injury using a High-Impact Trauma (HIT) device, and emetic responses were quantified by measuring regurgitated blue dye droplets. TRPA1 mutant and Trhn[01] mutant flies were further examined to assess the contribution of gut sensory and serotonergic signaling to trauma-induced emesis. TBI induced a dose dependent increase in emetic behavior, with males exhibiting stronger responses than females. Moderate trauma produced robust responses with relatively low mortality. Preliminary analysis of mutant lines suggests that TRPA1 contributes to trauma-induced emetic behavior, while the persistence of responses in Trhn[01] mutants indicates additional pathways may be involved. These findings establish a reproducible Drosophila model for studying TBI-induced emetic behavior and provide a novel platform for investigating gut-brain interactions following TBI. This model may facilitate future studies aimed at identifying the molecular basis of post-traumatic emesis and potential therapeutic targets for gut-brain axis disorders.

P1-Z3 Aishwarya Laxmi National Centre for Cell Science, Pune

Aging-Associated Remodeling of Germinal Center Dynamics Drives Loss of B-cell Tolerance and Autoimmunity

Aging is accompanied by declining vaccine efficacy and an increased prevalence of autoantibody associated disorders. Although impaired germinal center (GC) responses are a hallmark of aging, it remains unclear whether defective humoral immunity simply reflects diminished GC activity or results from active reprogramming of B-cell differentiation toward extrafollicular pathways. Here, we investigated the cellular mechanisms underlying age-associated defects in humoral immunity. First, we examined antigen-specific immune responses in naturally aged mice. Following immunization, aged mice exhibited poor GC formation, elevated antigen-specific IgM titers, reduced class-switched IgG1 antibodies, and markedly lower antibody avidity, indicating defective affinity maturation. To determine whether impaired GC responses are accompanied by enhanced extrafollicular differentiation, we next employed an imiquimod (IMQ)-induced lupus model, in which chronic type I interferon signaling drives pathogenic extrafollicular B-cell activation. Notably, IMQ-treated aged mice exhibited higher antinuclear antibody titers and, more importantly, broader reactivity against multiple self-antigens, consistent with type I interferon-driven epitope spreading. Together, these findings demonstrate that protective GC responses are compromised during aging and support the concept that humoral immunity is progressively redirected from canonical GC responses toward pathogenic extrafollicular differentiation. Because sustained type I interferon signalling is a major driver of inflammaging and extrafollicular B-cell responses, we next asked whether cellular senescence is sufficient to initiate this remodelling. Using a D-galactose-induced accelerated aging model, we found that D-galactose-treated mice recapitulated the impaired antibody responses observed in naturally aged mice, demonstrating that senescence-associated inflammation is sufficient to compromise humoral immunity. Collectively, our findings support a model in which age-associated inflammaging enhances type I interferon responsiveness, shifting B-cell differentiation from germinal center to extrafollicular pathways. We propose that this remodeling compromises GC fitness, promotes autoreactive B-cell responses, and contributes to both impaired vaccine-induced immunity and the development of autoimmunity during aging. Ongoing studies will determine whether therapeutic modulation of chronic type I interferon signaling can restore GC integrity, improve affinity maturation and vaccine responsiveness, and preserve B-cell tolerance in the elderly.

P1-Z13 Bhavuk Gupta National Centre for Cell Science, Pune

Spatio-Temporal Analysis of Microbial Community Dynamics Associated with a Large Religious Mass Gathering

Mass gathering events (MGEs) represent extreme, punctuated anthropogenic disturbances to natural environments, yet the structural resilience and ecological dynamics of river microbiomes under such acute stress remain largely understudied. This study provides a spatiotemporal characterization of the riverine microbiome across the Ganges and Yamuna rivers, and their confluence, during one of the world's largest religious mass gathering events. By integrating high-throughput microbial community profiling with comprehensive physicochemical water quality analyses, we investigated ecological dynamics across four distinct temporal phases: pre-event, during the mass gathering, post-event, and the COVID-19 lockdown, which served as a unique period of minimal anthropogenic disturbance. Alpha and beta diversity analyses revealed profound, statistically significant shifts in community composition that were primarily driven by the temporal gathering phases. Differential abundance using MaAsLin3 demonstrated a severe perturbation of the core microbiome, characterized by the displacement of 62 significantly different aquatic taxa and a highly significant influx of about 200 of conditionally rare, microbial taxa. This anthropogenic influx was dominated by key human-associated genera, notably Blautia, Dialister, Pseudomonas, Dorea, and Paracoccus. This biological contamination strongly correlated with a concurrent deterioration in physicochemical water quality indices, notably a spike in Biological Oxygen Demand (BOD) and a drop in Dissolved Oxygen (DO). Overall, our findings demonstrate that large-scale human gatherings can rapidly reshape riverine microbial communities and influence water quality. Although the microbiome exhibited clear signs of recovery following the event, with further restoration observed during the COVID-19 lockdown, these patterns highlight both the vulnerability and resilience of freshwater ecosystems to anthropogenic disturbance. The microbial taxa identified in this study represent potential bioindicators of human-associated pollution and offer valuable insights for environmental surveillance, water quality assessment, and public health risk management during future large-scale mass gathering events.

P2-Z4 Akshay Vijay Rathod

National Centre for Cell Science, Pune

Regulation of Clathrin Mediated Endocytosis by Munsicins

Clathrin-mediated endocytosis (CME) is a fundamental cellular process responsible for the internalization of receptors, nutrients, and signaling molecules. The initiation of CME is tightly regulated and depends on early endocytic adaptor proteins that define the formation of clathrin-coated pits (CCPs) at the plasma membrane. Members of the muniscin family FCHo1, FCHo2, and SGIP1 have emerged as key regulators of early CCP initiation. Increasing evidence suggests that the cellular levels of muniscin proteins play a critical role in promoting the activation of AP-2 and facilitating the assembly of the endocytic machinery. However, the individual contributions of these proteins, their domain-specific functions, and their cargo selectivity remain poorly understood. Understanding how muniscin abundance coordinates CCP nucleation and maturation is essential for elucidating the molecular mechanisms governing clathrin-mediated endocytosis.

P1-Z5 Akshita Gupta

Indian Institute of Science Education and Research, Bhopal (IISERB)

Role of Nucleoporin Nup88 in BCR Signalling Regulation

B cells are an essential component of the adaptive immune system and play a major role in protecting the body against infections. Upon encountering an antigen, B cells become activated and initiate intracellular signalling pathways that regulate proliferation and differentiation into memory and plasma cells. While memory B cells are quiescent cells, plasma cells secrete large amounts of antibodies. In quiescent B cells, nuclear transport activity is relatively controlled, with basal import and export of proteins and RNAs. Immediately after activation, BCRs get internalised, initiating the signalling pathway that leads to the nuclear import of activated transcription factors. On the contrary, a few hours after antigen encounter, activated B cells become transcriptionally active and initiate RNA export for making antibodies. We hypothesise that in this process of B cell differentiation, the nuclear pore complex might also undergo dynamic changes. Our RNA-seq data suggested post activation that transcripts for a few nuclear pore proteins are upregulated. Among these one of the nucleoporins is Nup88, which is a scaffold nucleoporin present on the cytoplasmic side of the nuclear pore complex. We are therefore interested in looking at the changes in Nup88 upon B cell activation. We examined the dynamic localisation of Nup88 during B-cell activation following antigen encounter; we observed that Nup88 co-clusters with internalised B-cell receptors (BCRs). Interestingly, multivesicular bodies-enriched fractions isolated from activated B cells showed increased Nup88 levels, whereas whole-cell Nup88 levels were reduced, indicating selective recruitment into MVBs and secretion into exosomes. We propose that Nup88 may serve as a scaffold linking BCR signalosomes to MVB-derived exosomal pathways, thereby contributing to immune communication.

P2-Z6 Akshssmita Somani

National Centre for Cell Science, Pune

Understanding the Role of VprBP in the MAPK Signaling Pathway During Tumorigenesis

The RAS–RAF–MEK–ERK signaling cascade is frequently dysregulated in colorectal cancer (CRC), driving uncontrolled proliferation, survival, and metastatic progression. While the core mechanisms governing ERK1/2 activation are well-documented, the precise regulatory factors fine-tuning its phosphorylation kinetics and intracellular localization remain incompletely understood. VprBP (DCAF1), a substrate receptor of the CRL4 E3 ubiquitin ligase complex, has been implicated in diverse oncogenic processes; however, its functional intersection with MAPK signaling remains uncharacterized. To elucidate the molecular role of VprBP in modulating ERK1/2 signaling dynamics, stable VprBP knockdown models were established in human CRC cell lines. Western blot analysis demonstrated a marked increase in baseline ERK1/2 phosphorylation following VprBP knockdown; however, subsequent nuclear-cytoplasmic fractionation revealed that VprBP depletion paradoxically diminished the nuclear accumulation of this active ERK population. Consistent with impaired nuclear signaling, functional characterization via colony formation assays showed a significant reduction in clonogenic growth, which correlated with a quantitative RT-PCR-confirmed down-regulation of classical ERK-responsive target genes. Together, these findings identify VprBP as a regulator of ERK signaling in colorectal cancer, demonstrating that disruption of the VprBP–ERK axis impairs nuclear translocation and suppresses tumorigenic potential, thereby providing novel mechanistic insights into MAPK pathway regulation

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P1-Z7 **Aman Sharma** Indian Institute of Science Education and Research, Pune

Cell-free reconstitution reveals the minimal machinery for fast Endophilin-mediated endocytosis

Endocytosis in particular, and vesicular transport in general, require the precise coupling of cargo recognition, membrane deformation, and vesicle scission. While the sequence of events and mechanisms leading to the formation of a clathrin-coated transport vesicle are well established, the principles governing cargo capture and membrane remodeling during clathrin-independent pathways—such as fast Endophilin-mediated endocytosis (FEME)—remain poorly understood. Here, using a bottom-up reconstitution approach with supported membrane templates and purified proteins, we define the minimal machinery sufficient for Endophilin A1-driven cargo internalization and membrane vesiculation. We demonstrate that Endophilin A1 self-assembles on $PI(4,5)P_2$-containing membranes to generate highly curved nanotubes that serve as substrates for Dynamin 2-mediated scission. The coupling of Endophilin A1 and Dynamin 2 reconstitutes a tubulation-coupled fission module that processes membrane tubules into detached vesicles. Mechanistically, Endophilin abundance modulates Dynamin activity via $PI(4,5)P_2$ sequestration, uncovering a lipid-mediated regulatory mechanism that tunes the transition from membrane deformation to fission. FEME serves to manage the endocytosis of the β1-adrenergic receptor by recognition of the third intracellular loop (TIL) of the receptor by Endophilin A1. Remarkably, the TIL is sufficient to nucleate Endophilin A1 assembly and induce membrane tubulation independent of $PI(4,5)P_2$ and membrane electrostatic interactions, establishing cargo as an active driver of membrane remodeling. Reconstitution of this complete cargo–Endophilin–Dynamin axis reveals that cargo engagement temporally precedes Dynamin recruitment. Furthermore, competitive occupancy of the Endophilin SH3 domain by cargo versus Dynamin functions as a molecular switch governing the progression from curvature generation to fission. Together, our findings establish a minimal mechanistic framework for FEME and elucidate how sequential, competitive molecular interactions coordinate cargo capture with vesicle formation.

P2-Z8 **Tanisha Mudaliar**
Apurva Dalve
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In Silico Identification of Pterospermum acerifolium and Dalbergia sissoo as Multitarget Breast Cancer Stem Cell Modulators

Breast cancer stem cells (BCSCs) are a highly tumorigenic subpopulation implicated in tumor recurrence, metastasis, and therapeutic resistance. As bone is the most frequent site for breast cancer metastasis, leading to skeletal deterioration and impaired bone remodeling, there is a growing need for therapeutic agents that simultaneously suppress BCSCs and support bone regeneration. Pterospermum acerifolium and Dalbergia sissoo are medicinal plants that possess osteo-inductive properties and diverse bioactive phytochemicals, making them attractive candidates or dual-action therapeutic discovery. Phytochemicals from both plants were screened using in silico ADME, drug-likeness, and toxicity analyses to identify compounds with favorable pharmacological properties. Candidate molecules were subsequently subjected to target prediction,network-based analyses, and molecular docking against key BCSC-associated receptors, Among the screened compounds, apigenin, luteolin, and kaempferol from P.acerifolium, and tectorigenin, ψ-tectorigenin, 7-O-methyltectorigenin, and 5-hydroxy- 6,7,4′-trimethoxyisoflavone from D. sissoo demonstrated favorable pharmacokinetic profiles. For P. acerifolium, apigenin exhibited the strongest binding toward EGFR (−8.9 kcal/mol), kaempferol showed the highest affinity for AKT1 (−8.2 kcal/mol), while luteolin demonstrated the strongest interaction with MMP9 (−10.7 kcal/mol). Among the D. sissoo phytochemicals, 5-hydroxy-6,7,4′-trimethoxyisoflavone demonstrated the strongest binding to EGFR (−8.2 kcal/mol), while ψ-tectorigenin exhibited the highest affinity toward ADORA2A (−7.3 kcal/mol).These findings demonstrate the modulatory potential of investigated plants and their bioactive molecules. Specifically, P. acerifolium targets EGFR, AKT1 and MMP9, while D. sissoo targets EGFR and ADORA2. Both plants disrupt key components driving breast cancer stem cell tumorigenesis, offering strong foundation for in vitro validation.

P1-Z9 Ashrita singh

National Centre for Cell Science, Pune

Investigating the role of Nup358 -mediated regulation of ER-Mitochondrial contact sites in Neurodegenerative diseases.

Amyotrophic lateral sclerosis (ALS) is a progressive neurodegenerative disorder where the nerve cells, mainly motor neurons, gradually degenerate, causing muscles to become paralyzed. Researchers have found many different biological causes that can trigger ALS, and one of the primary causes appears to be Endoplasmic Reticulum-Mitochondria Contact Sites (ERMCS) disruption. Research has shown that TDP43 and FUS pathogenic mutations lead to disruption of ERMCS by activating GSK3β. However, the mechanistic details of how TDP43 and FUS activate GSK3β is not known. Our laboratory has recently shown that Nup358, a nucleoporin present on the cytoplasmic ER subdomain called annulate lamellae (AL) contributes to ERMCS disruption via regulation of the mTORC2/Akt/GSK3β axis. This observation raises the possibility of a crosstalk between Nup358–TDP-43 and Nup358–FUS. To investigate the potential interconnection between Nup358 and TDP-43/FUS, we performed immunoprecipitation assays to assess the physical interaction between these proteins. The results revealed that both TDP-43 and FUS interact with Nup358. Currently, testing the hypothesis that Nup358 and the ALS proteins (TDP-43 and FUS) coordinate to regulate the ERMCS dynamics through the regulation of GSK3β is underway. To further elucidate the molecular mechanism, ER-mitochondria connectivity will be assessed under Nup358 and TDP-43/FUS depleted or overexpressed condition. This work will also allow us to determine whether the TDP-43–Nup358 and FUS–Nup358 interactions are hierarchically organized in regulating ERMCS.

P2-Z10 Atanu Sahoo

National Centre for Cell Science, Pune

Assessing the role of F-box protein, FBXO31 in NF-κB pathway

NF-kB functions as pleotropic transcription factor that plays a central role in immune response, stress response and other cellular processes. The activation of NF-kB is a double-edged sword. While needed for proper immune system function, aberrant NF-kB activation can lead to different inflammatory diseases, autoimmune disorders and tumorigenesis. It is well established that proteasomal degradation of IkB family proteins is obligatory for NF-kB pathway activation. Till date, F-box protein βTrCP is known to direct proteasomal degradation of IkB. However, which paralogue of βTrCP is involved in IkB degradation not known. Therefore, we speculate that other F-box protein might control the degradation of IkB and performed a F-box screen. Our study reveals that several F-box proteins including FBXO31 direct IkBα degradation. We found that FBXO31 promotes the proteasomal degradation of IkBα. Mechanistically we found that FBXO31 directs proteasomal degradation of IkB through SCF complex and it controls NF-kB activation under different stress conditions. Thus, FBXO31 is critical regulator NF-kB signaling pathway.

P1-Z11 Ayushi Kumari

National Centre for Cell Science, Pune

Exploring the Conserved RNA-Binding Activity of PIP4K2 Across Parasitic and Mammalian Systems

PIP4K2A (phosphatidylinositol-5-phosphate 4-kinase type II**α**) is a highly conserved phosphoinositide kinase that phosphorylates phosphatidylinositol-5-phosphate (PI5P) to phosphatidylinositol-4,5-bisphosphate, thereby regulating cellular phosphoinositide homeostasis. PIP4K2A is ubiquitously expressed across tissues and subcellular compartments and has been implicated in regulating membrane trafficking, autophagy, cellular metabolism, stress responses, and signal transduction pathways. It is also genetically and functionally linked to neuropsychiatric disorders, including schizophrenia. Dysregulation of PIP4K2A has also been associated with cancer progression, metabolic diseases, and neurological dysfunction, highlighting its broad physiological and pathological significance. Beyond its lipid kinase activity, PIP4K2A was recently shown to have RNA binding activity. Additionally, PIP4K2A was found to be imported from the host into apicomplexan parasites Plasmodium and Toxoplasma species, despite the absence of an endogenous PIP4K2A in these organisms. Host-derived PIP4K2A was shown to be essential for parasite survival, suggesting an important role during infection. Furthermore, PIP4K2A associates with parasite transcripts, and subsequent studies have demonstrated that its RNA binding activity is evolutionarily conserved.

These findings suggest that PIP4K2A has a key role in gene regulation in addition to its role in phosphoinositide metabolism. We hypothesise that the RNA binding activity of PIP4K2A is an evolutionarily conserved function that contributes to gene regulatory networks in both parasites and mammals. Our work aims to (i) investigate the import and functional significance of PIP4K2A in non-apicomplexan parasitic species, (ii) characterise the PIP4K2A-mediated gene regulation in neuronal physiology, and (iii) characterise the RNA binding activity of PIP4K2B and PIP4K2C, the two mammalian isoforms of PIP4K2A.

P2-Z12 **Bhavika Jethani** National Centre for Cell Science, Pune

Clathrin Light chain regulates neural development and function in Drosophila melanogaster

Clathrin is a cytosolic protein involved in the intracellular trafficking of a wide range of cargo. It is composed of three heavy chains and three light chains that together form a triskelion, the subunit that polymerizes to form a clathrin coated vesicle. Although the role of the heavy chains in regulating important physiological processes has been well documented, we still lack a complete understanding of how clathrin light chains regulate membrane traffic and cell signaling.
In this study, we use Drosophila melanogaster as a model system to understand the role of clathrin light chains (CLC) in neural development and function. Using CRISPR -Cas9, we show that adult flies exhibit a severe locomotion defect upon the depletion of the light chain. This phenotype is also seen upon neuron-specific loss of the protein indicating that a neuron specific function of the light chain, presumably it's role in synaptic vesicle recycling, is essential for locomotion in adult flies. We also observe defects in neuronal morphology and maturation at the neuromuscular junction in the third instar larvae of the CLC Knockout flies which may be a contributing factor to the behavioral defect. Interestingly, this behavioural defect is observed in larvae only upon heat stress. Other behavioural defects like an increase in water consumption is also seen in these flies indicating an altered ability to sense thirst or satiation. We also observe an altered mushroom body structure in these flies indicating that these flies may have defects in learning and memory formation.

P2-Z14 **Bheem Singh Bhandari** National Centre for Cell Science, Pune

Functional study of the mammalian Nup88 sub-complex of the nuclear pore complex

Nuclear pore complexes are the megadalton assemblies residing in the nuclear envelope of an eukaryotic cell. It directs the import and export of biomolecules between cytoplasm and nucleus, which are highly specific, dynamic and regulated. There are 32 different proteins (Nucleoporins) that build this assembly together. NPC is modular and can be divided into many sub-complexes. One such sub-complex is the Nup88 sub-complex (Nup214•Nup88•Nup62), And located at the cytoplasmic side of the NPC and also their proteins involve in mRNP export. Although its interactors are known but domains wise exact role of these proteins of the mammalian NPC are still unknown. The large sequence difference of the Nup88 sub-complex nucleoporins of the yeast and higher vertebrates, it becomes difficult to delineate and understand the proper mechanism of mRNP transport by the NPC in higher vertebrates. In this study, We aim to investigate the functional role of Nup214 in mRNP export using stable Nup214KD HeLa cells and rescue the function via the domain wise overexpression followed by mRNA quantification of the Nuclear-Cytoplasm fraction.

P1-Z15 **Brindaban Das** National Centre for Cell Science, Pune

FBXO41 restricts breast cancer growth by controlling multifaceted regulation of atypical CDK, CDK5

F-box proteins are substrate recognition components of SCF (Skp1-Cullin1-F-box) E3 ligases, which play vital roles in cell cycle progression, apoptosis, DNA damage repair, cell signaling, and other cellular processes by governing the turnover of major cellular proteins. The human genome encodes 69 F-box proteins, but the function of most of them remains elusive. From our lab, a tumor suppressor screening of F-box proteins showed that FBXO41 inhibits growth of the MCF7 breast cancer cell line through inducing autophagy. Several unpublished data highlighted that FBXO41 can also target oncoprotein CDK5. CDK5, popularly known as atypical CDK, has a crucial role in post-mitotic neurons. Apart from its physiological role, it is a well-known driver of cancer progression. Using biochemical assays, we have observed that FBXO41 directly interacts with CDK5 and promotes its degradation through proteasomal as well as lysosomal pathways. Lastly, our phenotypic data suggest that FBXO41 may be functioning as a tumor suppressor in breast cancer by targeting and facilitating degradation of CDK5.

P2-Z16 Debleena Mukhopadhyay

National Centre for Cell Science, Pune

Study of Pathogenic Huntingtin Aggregates from the perspective of RNA binding proteins

Huntington's disease (HD) is an autosomal dominant disorder that causes progressive neurodegeneration. HD causes neuronal death in the striatum and cerebral cortex, which ultimately manifests as severe motor and cognitive dysfunction. Currently. There has been no cure for HD apart from physiotherapy and speech therapy to help manage the symptoms; the disease only results from the expansion of the CAG repeats, which ultimately results in the formation of toxic Huntingtin (Htt) aggregates. These Htt aggregates have been found to sequester a wide variety of proteins within the aggregate, which might be sent by the cellular system.

In our study, we propose to study the Htt aggregates from the perspective of RNA and RNA-binding proteins (RBPs). We plan to co-transfect S2 cells with HttQ138 along with multiple RBPs to screen for the RBP targets that get sequestered in the aggregates. Additionally, we would study what role these RBPs might play in the formation or clearance of these aggregates. We would also extrapolate our findings using Drosophila melanogaster as our animal model system. Overall, our study aims to check whether targeting RBPs and RNAs in the Htt aggregates can be used as a novel therapeutic method in the future and rescue the toxicity associated with it.

P1-Z17 Felix Christian

National Centre for Cell Science, Pune

Understanding the role of substance P–Tachykinin receptor-mediated dendritic cell function in gut inflammation, and autoimmunity

Neuroimmune communication is a bidirectional interaction between the nervous and immune systems. These interactions are important for integrating signals from various stimuli to mount an appropriate immune response and maintain homeostasis. Dysregulation of these interactions has been implicated in autoimmune diseases, including inflammatory bowel disease (IBD). Substance P (SP), a neuropeptide elevated in patients with IBD, has emerged as a potential pro-inflammatory mediator. As professional antigen-presenting cells, dendritic cells (DCs) integrate environmental cues and regulate T-cell priming, thereby determining the balance between immune tolerance and inflammation. DCs express the neurokinin-1 receptor (TACR1), the primary receptor for Substance P; however, whether SP–tachykinin receptor 1 (TACR1) signaling modulates DC function to shape downstream T-cell responses during intestinal inflammation remains unclear.

We hypothesize that SP signaling through TACR1 regulates DC phenotype and function, thereby shaping T-cell responses and contributing to intestinal inflammation. To test this, bone marrow-derived DCs will be used to investigate the effects of SP during DC activation, antigen presentation, cytokine production, and T-cell stimulatory capacity. The functional consequences of these changes will be assessed using DC-T cell co-culture assays. In parallel, the role of TACR1 signaling in intestinal inflammation will be examined in the dextran sodium sulfate (DSS)-induced colitis model by comparing disease progression and dendritic cell and T-cell phenotypes in TACR1-deficient and wild-type mice. Together, these complementary in vitro and in vivo studies aim to define the role of SP–TACR1 signaling in DC-mediated regulation of adaptive immunity and provide mechanistic insights into neuroimmune communication during intestinal inflammation.

P1-Z19 Juveria Azam

National Centre for Cell Science, Pune

Deciphering the role of FBXW11 in cellular signalling in cancer

FBXW11, a substrate receptor component of SCF E3 ubiquitin protein ligase, plays a crucial role in breast cancer progression, impacting patient outcomes significantly. We recently showed that FBXW11 functions as an oncogene in breast cancer. However, its oncogenic potential is still poorly understood. Serendipitously, we found that FBXW11 positively controls the activation of AKT signalling. We found a significant correlation between FBXW11 expression and AKT activation in triple-negative breast cancer. Furthermore, we found that FBXW11 activates AKT in a growth factor-independent manner. Furthermore, our study demonstrates that overexpression of FBXW11 in breast cancer leads to activation of the AKT pathway under nutrient-deprived conditions. In addition, we found that FBXW11-expressing cells are resistant to AKT inactivation-mediated cell death, indicating that the FBXW11-axis might be targeted for anti-cancer therapeutic intervention.

P2-Z18 Hariom Goswami

National Centre for Cell Science, Pune

Generation of neutralizing human monoclonal antibodies against Rabies virus Glycoprotein

Rabies is a neglected yet fatal zoonotic disease responsible for approximately 59,000 deaths annually, primarily in low- and middle-income countries. Transmission occurs mainly through bites from rabid dogs, although increasing wildlife–domestic–human interactions have contributed to the spread of rabies into previously unaffected regions. While rabies can be prevented through timely post-exposure prophylaxis (PEP), access to Rabies Immunoglobulin (RIG) remains limited due to high production costs, supply shortages, batch variability, and safety concerns. Human monoclonal antibodies (mAbs) offer a promising alternative because of their safety, consistency, and scalability.

In this study, human mAbs targeting the rabies virus (RABV) glycoprotein (G), the primary target of neutralizing antibodies, were generated from B cells isolated from vaccinated individuals following dog-bite exposure. Immunoglobulin heavy- and light-chain genes were amplified from single sorted B cells, cloned into expression vectors, and expressed in HEK293 and CHO cells. Recombinant antibodies were screened for binding to RABV G protein and evaluated for neutralizing activity. Several candidates demonstrated strong binding and broad neutralization against multiple RABV strains.

These findings support the development of human mAbs as alternatives to RIG for rabies PEP. Ongoing in vivo studies will assess their protective efficacy and clinical potential.

P1-Z31 Muskan Suthar

National Centre for Cell Science, Pune

Deciphering the role of p85 in RNA binding activity of PIP4K2a

Cellular signaling being essential and fundamental aspect of life and metabolism governing it. Such signaling wont be possible without proteins interacting with other proteins, finally altering the downstream pathways and molecules involved in it. To study such interactions, we'll need to isolate proteins tagged with some specific sequence which would facilitate purification and solubility as well in case of gst tag. My dissertation work focuses on His tag protein purification- optimization and further protein purification by Ni-NTA beads /column of p85 alpha protein. Parallely, I cloned the lentiviral construct containing PIP4K2A S to N variant via gibson assembly then transfected into FT cells and transduced into HEK T cells, finally validating the expression by Western blotting; hence preparing the stable cell line expressing PIP4K 2A gene tagged with HA.

P2-Z20 Jyoti Chaurasia

National Centre for Cell Science, Pune

Elucidation of Cellular And Molecular Basis Of Pathological Heterogeneity In Amyotrophic Lateral Sclerosis

Amyotrophic lateral sclerosis (ALS) is a severe neurological disease where the upper and lower motor systems degenerate gradually. It progresses from muscle weakness to total paralysis and respiratory failure, and eventually to death, often co-occurring with frontotemporal lobar degeneration (FLTD), with a survival expectancy of 2-5 years. The pathological hallmark is the widespread presence of abnormal aggregates of TAR DNA-binding protein-43 (TDP-43) called cytoplasmic inclusions in various parts of degenerating brain regions, including the spinal cord, motor cortex, and lower brainstem. These inclusions exhibit significant heterogeneity in shape and size within degenerating neurons. Neither is it clear which of the heterogeneous structures drives the disease pathology and neurotoxicity, nor is it fully understood what specific biological factors trigger this heterogeneity. Recent studies of postmortem ALS/FTLD patient brains reveal a cleaved fragment of Annexin A11 (ANXA11) co-aggregated with TDP-43. ANXA11, known to be an ALS risk gene, physiologically functions on cellular trafficking and aids local mRNA translation in neurons through stress granule formation. Increasing evidence indicates that not only known disease mutants but also wild-type ANXA11 and its differently sized cleaved fragments can exhibit different ALS pathological phenotypes. Employing protein engineering and cell biological methodologies, we focus on elucidating independent and co-aggregation mechanisms of ANXA11 and TDP-43 through the development of ALS disease models. We specifically started with questions aimed at understanding the structural dynamics and pathobiological roles of ANXA11 and its disease variants at the levels of phase separation, aggregation mechanisms, and variant-specific contributions to disease heterogeneity.

P1-Z21 Krishna Ashokkumar

National Centre for Cell Science, Pune

Impact of Insulin Deficiency and Hyperglycemia on Bone Remodeling: A Path to Diabetic Osteoporosis

The skeletal system is an intricately specialized and adaptive organ that undergoes incessant remodeling to preserve its structural integrity. Osteoblasts, osteocytes, bone-lining cells, and osteoclasts, along with their progenitor cells, collectively constitute the Bone Multicellular Units (BMU). The principal role of BMU is to ensure the maintenance of normative physiological bone remodeling. Dysfunction in the operations of BMU induces an imbalance in the bone remodeling mechanism, culminating in significant bone pathologies such as osteoporosis. As delineated in the Global Burden of Disease Study 2019, Diabetes has been recognized by both the United Nations and the World Health Organization as one of the five paramount non-communicable diseases (NCDs). The auto-immunological destruction of insulin producing beta cells of the pancreas results in a state of near total insulinopenia leading to development of Type 1 Diabetes Mellitus (T1DM). Insulin is known to stimulate mitosis and inhibit apoptosis in osteoblasts. The deficiency of insulin within the physiological system exerts a detrimental influence on the anabolic regulation of bone remodeling, consequently resulting in diminished bone mineral density (BMD) and an elevated risk of fractures. This pathological state, in which osteoporosis arises because of diabetes, is referred to as Diaporosis or Diabetic Osteopathy. Lack of insulin in the body results in chronic hyperglycemia. Advanced glycation end-products (AGEs) are generated through the non-enzymatic glycation of proteins, including collagen, which subsequently interferes with the adhesion of osteoblasts to the extracellular matrix and diminishes the activity of alkaline phosphatase (ALP) along with the secretion of Osteocalcin by mature osteoblasts. An augmented presence of proinflammatory cytokines is noted, which ultimately induces osteoclastogenesis and inhibits the differentiation of osteoblasts, culminating in skeletal fragility. Our invitro studies revealed that hyperglycemic conditions significantly impair osteogenesis. Similarly, in a T1DM mouse model, insulin deficiency and hyperglycemia compromised trabecular and cortical bone parameters, reducing bone strength and affecting collagen tissue and sub-chondral bone. This highlights the need for therapeutic interventions to address bone loss, prompting us to develop cellular and vesicular treatment strategies.

P2-Z22 Krishna Ch Mondal

National Centre for Cell Science, Pune

Investigation of Nucleocytoplasmic Transport through Central Transport Channel of NPC.

The nuclear pore complex (NPC) is a massive macromolecular assembly embedded in the nuclear envelope that serves as the sole gateway for nucleocytoplasmic transport. The central transport channel (CTC), formed by three phenylalanine-glycine (FG) nucleoporins – Nup62, Nup58, and Nup54 creates a selective hydrogel-like barrier that regulates the passage of biomolecules between the nucleus and cytoplasm. While small molecules (<40 kDa) traverse the NPC passively, larger cargoes require nuclear transport receptors (NTRs) that interact with the characteristic FXFG/GLFG repeat motifs present in the intrinsically disordered N-terminal domains of CTC nucleoporins. Among the CTC components, Nup58 is uniquely distinguished by possessing an unstructured C-terminal domain, in contrast to Nup62 and Nup54, which bear structured alpha-helical C-termini that anchor the complex to the inner ring via interactions with Nup93. This structural distinction suggests that Nup58 may play a non-redundant role in maintaining CTC architecture and transport selectivity. However, the precise functional contribution of Nup58 to nucleocytoplasmic transport and other cellular perspectives remains poorly understood.

In this study, we investigate the structural and dynamic consequences of Nup58 depletion on the central transport channel using molecular dynamics simulations of the Nup62-Nup54 sub-complex, in-vitro self assembly of Nup62-Nup54 FXFG domain, SPA of CTC-Nup93 complex with cellular function of Nup58 based upon lentivirus mediated knockdown and high throughput screening. By characterizing conformational changes, FG-repeat accessibility, and hydrogel network properties in the absence of Nup58, we aim to elucidate its specific role in establishing transport selectivity. Our findings provide mechanistic insight into how individual FG-nucleoporins contribute to the collective gating function of the NPC, with broader implications for understanding transport dysregulation in disease

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P1-Z23 Kshitija Kavali National Centre for Cell Science, Pune

Investigating how Clathrin light chains regulate mitochondrial function in early development

Clathrin mediated endocytosis is a key endocytic pathway involved in regulating cell fate decisions during early development. The basic unit of the clathrin coat is the triskelion structure made up of three heavy and three light chains. While the clathrin heavy chain is known to maintain pluripotency in embryonic stem cells, the role of the Clathrin light chains in development is underexplored. Previously our lab has made and characterized Clathrin light chain knockout mouse embryonic stem cells (mESCs), which show an altered organization of the actin cytoskeleton along with aberrant expression of differentiation markers (Tiwari et al, 2025). During differentiation, pluripotent stem cells undergo a drastic change in mitochondrial morphology, transitioning from small and fragmented to elongated, tubular networks, which is accompanied by a significant increase in the number of mitochondria in mouse and human embryonic stem cells. We therefore characterized whether mitochondrial function was altered in these knockout mESCs. We observed changes in cellular proliferation rates and mitochondrial cristae organization in mESCs upon loss of specific clathrin light chains. Further, we observed that loss of Clta resulted in more tubular mitochondria, while loss of both Clta and Cltb led to a change in the overall shape of the mitochondria from tubular to donut. We also observed an increase in the number of PDH enriched mitochondria-derived vesicles (MDVs) in Clta-/- mESCs. Golgi-derived vesicles mark mitochondrial fission sites. The presence of clathrin light chain A (CLCa) in the Golgi indicate towards potential novel functions for CLCa in initiating mitochondrial fission. Our results suggest a possible role for Clathrin light chains in regulating mitochondrial morphology during early development.

P2-Z24 Lavan Kumar Bansal National Centre for Cell Science, Pune

Sortilin receptor regulates contractile vacuole dynamics by controlling Dop1p trafficking

Contractile vacuole complexes (CVCs) are intricate osmoregulatory organelles comprising of tubular (spongiome) and vesicular (bladder) subcompartments. The mechanisms that underlie their formation and maintenance within the eukaryotic endomembrane network are poorly understood. In Tetrahymena thermophila, we found a key CVC determinant, Sortilin receptor 3 (Sor3), that shows sensitivity to osmotic changes. Sortilins, animal homologs of S. cerevisiae Vps10, are post-Golgi trafficking receptors found in a variety of cell structures, including the trans-Golgi network (TGN), endosomes, secretory vesicles, multivesicular bodies, and the cell surface.

In this study, we found that Sor3p localizes to highly mobile cytoplasmic puncta. We show by inducible knockdown that SOR3 is required for CVC organization and function. SOR3 knockdown increased susceptibility to osmotic shock, tolerated in the wildtype but triggering irreversible lethal swelling in the knockdown cells. The depletion of SOR3, rapidly triggered contraction of the spongiome and extended the bladder contractile cycle, disassembly of CVC, and dispersal of proteins associated with those compartments. Further, we show that the CVC protein Dop1p is localized to the bladder, spongiome and cytoplasmic puncta. The distribution and number of these cytoplasmic puncta are reduced in SOR3 knockdown cells, indicating that Dop1p trafficking relies on Sor3p. Additionally, the cytoplasmic puncta of Dop1p showed colocalization with Sor3p. Notably, a greater extent of colocalization was observed near the CVC. Our findings suggest that Sor3p is required for the organization and function of CVCs in ciliates, highlighting the key role that SOR3 plays in maintaining this complex compartment.

P1-Z25 **Mala Adarsh** National Centre for Cell Science, Pune

Understanding the mechanism of substrate recognition, unfolding and degradation by human proteasomes

Almost all proteins of the human proteome, meet with proteasome for their own destruction. Proteasome is a multisubunit-multicatalytic complex of MW 2000 KDa and the protein degradation machinery in the Ubiquitin-Proteasome System (UPS) in all eukaryotic organisms. The 26S proteasome complex is the major ubiquitin-dependent degradation machinery. However, our recent report suggests the smaller complex – "20S proteasome", which comprises ~50% of the total cellular proteasomes, act as an emergency proteasome under hypoxic stress conditions where it can even degrade globular proteins along with the unstructured protein-substrates. Elevated levels of 20S proteasomes appear to contribute to cell survival under hypoxic stress by degrading damaged proteins. Very surprisingly, without any known dedicated substrate-binding sites and any known unfoldase activities, 20S proteasome is able to proteolyse well-folded substrates. In my PhD project, we aim to elucidate the molecular mechanism of "how 20S proteasome recognises and unfolds its substrates for proteolysis?" We will follow biochemical and Single particle Cryo-EM based-structural studies to understand the molecular mechanism of 20S proteasome function.

P2-Z26 **Madhu Parna Karmakar** National Centre for Cell Science, Pune

Repurposing cAMP-PKA pathway modifier drugs for the treatment of Alzheimer's disease

Tau aggregation is one of the major causes of 8 various tauopathies in the human brain, including AD. Since there are no FDA-approved drugs targeting the primary cause of tauopathies, treatments mainly focus on managing the symptoms and exploring disease-modifying therapies. Nevertheless, our preliminary data, using some FDA-approved cAMP/PKA signal-modifiers, surprisingly show significant evidence for tau degradation in cellular AD models. However, a thorough systematic study is needed to establish these substantial findings in animal AD models. Hence, in this study, we are proposing to develop AD-models in Drosophila and check the efficacy of those cAMP/PKA signal-modifiers along with some potential hormones as novel modifiers for Tau degradation. We will test two small molecules, Forskolin (an Ayurvedic medicine for respiratory diseases), ProstaglandinE1 (FDA-approved drug for erectile dysfunction), and three hormones, Ghrelin, epinephrine, and glucagon, for their efficacy in degrading Tau protein via ubiquitin-proteasome pathway in AD-cell models and then in AD-drosophila models. This study will demonstrate the efficient cAMP/PKA-modifier drug and provide a novel modality to protect against neurodegeneration in Alzheimer's disease.

P1-Z27 **Mohammad Tanzeel Aalam** National Centre for Cell Science, Pune

Acid-sensitive liposomal vesicles for cargo delivery directly to the insect gut: probing post-ingestive mechanisms of food perception

Using Drosophila melanogaster as a model organism provides an opportunity to study questions related to the gut–brain axis because of the wide availability of genetic tools. To study the role of gut–brain axes in food component perception, we often have to manipulate or bypass peripheral sensing using genetic tools in transgenic flies. This makes it genetically cumbersome to manipulate post-ingestive mechanisms in parallel, and such manipulation techniques are further limited in non-genetic model organisms.

To overcome this, we report the development of a liposome-based pH-sensitive vesicle system designed to release encapsulated molecules in response to the acidic conditions of the fly's midgut. These liposomes are stably formed at alkaline and neutral pH while enabling cargo release under acidic conditions. Characterization shows consistent vesicle size, encapsulation efficiency, and pH-dependent release kinetics. Using fluorescent dyes and behavioural assays, we demonstrate the utility of this system to probe gut–brain axis function in vivo. Overall, this vesicle system offers a valuable tool for studying chemosensory and physiological responses to gut inputs in Drosophila by reducing the need for peripheral genetic manipulation in behavioural and neuronal studies.

P2-Z28 Mir Ahmad Habib

National Centre for Cell Science, Pune

Role of CCR6 on innate lymphoid cells during gut inflammation and autoimmunity

Innate lymphoid cells (ILCs) are a class of innate immune cells that lack antigen-specific receptors and expand under the influence of local cytokine milieu. ILCs are deeply integrated into tissue architecture and are crucial for the body's rapid defense against infections, particularly at barrier sites such as the skin and gut. Based on expression of signature transcription factors and effector cytokine secretion, ILCs are grouped into three broad classes: Group 1 ILCs comprise ILC1 and NK cells. Group 2 ILCs include ILC2, and Group 3 includes ILC3. The inflamed gut is known to produce the chemokine CCL20 and drive the recruitment of chemokine receptor CCR6-positive immune cells into the gut. How the CCR6-CCL20 axis alters the recruitment, differentiation, and function of ILCs during gut inflammation remains unknown.

Our data showed that CCR6-knockout mice exhibit delayed gut inflammation when treated with 2% DSS (w/v) in the drinking water. Analysis of ILCs using flow cytometry in the mesenteric lymph nodes (mLN) showed that the frequency of ILC1 (T-bet+) and ILC2 (GATA3+) cells was significantly increased in the DSS-treated wild-type C57BL/6 mice compared to the CCR6-/- or control mice. CCR6-/- mice showed significantly reduced pathological changes with DSS treatment in the gut compared to wild-type mice. Furthermore, upon exposing purified ILCs to recombinant CCL20 in culture, significant increase of T-bet, GATA3 and RORγt expression was observed in wild-type ILCs compared to CCR6-/- ILCs. These results suggest that CCL20-CCR6 signaling influences ILC polarization, which may contribute to their phenotypic plasticity and effector functions during gut inflammation, and provide a new strategy for using CCR6 as a therapeutic target to control gut inflammation and autoimmunity.

P1-Z29 Mithila Chinchalkar

Modern College Ganeshkhind

Stem Cell Characterization in Dugesia punensis: Insights into Regenerative Biology

Freshwater planarians are renowned for their remarkable regenerative capacity, primarily attributed to a population of adult pluripotent stem cells known as neoblasts. The present study investigates stem cell characterization in Dugesia punensis, a recently described freshwater planarian species endemic to Pune, Maharashtra, India. Morphological observations, histological analyses, and cellular characterization were employed to identify the distribution and organization of neoblasts within the body parenchyma. Histological sections revealed densely distributed undifferentiated cells throughout the mesenchymal tissue, with notable exclusion from the pharyngeal lumen and cephalic tip, consistent with the neoblast distribution reported in other triclad planarians. Regenerative responses following amputation demonstrated rapid blastema formation, highlighting the functional role of these stem cells in tissue restoration. The observed cellular architecture supports the exceptional regenerative potential of D. punensis and contributes to the understanding of stem cell biology in indigenous Indian planarian species. This study establishes D. punensis as a promising non-model organism for investigating stem cell dynamics, regeneration, and developmental biology, providing a foundation for future molecular characterization and comparative regenerative studies.

P2-Z30 Monty Vijayvargiya

National Centre for Cell Science, Pune

Crosstalk between Nucleoporin 155 and Nucleoporin 93 in the Nuclear Pore complex

Nuclear Pore Complexes are megadalton assemblies made up of 34 different proteins, that form large conduits for cargo transport between the cytoplasm and nucleus. During transport, the proteins of the NPC undergo numerous rearrangements that allow NPC to transition from a state of constriction to dilation. To understand this mechanism of transition, we aimed at investigating the crosstalk between two critical proteins of NPC, Nup93 and Nup155 that reside in the inner ring of NPC. Nup93 is a versatile linker protein that also interacts with the central transport channel which includes Nup62, Nup58 and Nup54 whereas Nup155 has the membrane binding domain that tethers to the nuclear envelope. To delineate the role of these nucleoporins in NPC dynamics, we mapped the interaction interfaces between Nup93-Nup155 and reconstituted the complexes in-vitro. Previous studies including Cryo-ET maps of the NPC only highlight a single interface between these two proteins for their interaction. Interestingly, we observed that there are multiple interaction sites present between these proteins that provide scope for vigorous rearrangement of proteins within NPC and help achieve the plasticity required for active transport of cargoes. Structural studies using cryo-electron microscopy (cryo-EM) will be used to further visualize these newly identified interaction interfaces and capture their dynamic states at a higher resolution.

P2-Z32 **Namrata Jiya** National Centre for Cell Science, Pune

Microbial Life in Antarctica: Diversity and Adaptation

Antarctica harbours the most extreme, heterogeneous ecological niches which support a diverse range of polyextremophilic microorganisms, making it important to study the microbial diversity of this pristine continent. In the present study, we characterize the bacterial and fungal communities associated with different environmental niches and sediment types of Antarctica using culture-dependent and culture-independent approaches. High throughput sequencing revealed the presence of bacterial phyla like Actinobacteriota (22.5%), Bacteroidota (18.9%), Proteobacteria (18.7%), Acidobacteriota (12%) and Chloroflexi (7.6%); and bacterial genera including Crossiella (14.3%), Rhodanobacter (10.4%), Blastocatella (8.6%) and Pseudonocardia (6.9%) in the accumulated snow and exposed sediments. Metagenomic prediction led to the detection of pathways responsible for carbohydrate metabolism, DNA repair and carbon fixation. DNA metabarcoding using ITS gene showed the occurrence of fungal phyla like Ascomycota (61.7%), Basidiomycota (31.1%) and Chytridiomycota (5.7%); and fungal genera like Glaciozyma (24.8%), Cladosporium (16.8%), Phenoliferia (8.8%), Acrodontium (8%) and Aspergillus (5.4%) in diverse environmental niches like exposed, accumulated snow, deep sea and lake sediments of Antarctica. Culture-dependent approach using polyphasic characterization led to the identification of two novel microorganisms, including a novel yeast-like fungus Leuconeurospora bharatiensis (MCC 10126T), isolated from accumulated snow sediment; and a novel red-pigmented, UV resistant bacterium Deinococcus pantiae (MCC 5347T) from a lake sediment. Genome mining of the Deinococcus strain revealed the presence of metabolic pathways for adaptation including DNA repair, oxidative stress response, desiccation resistance, production of carotenoid pigment and membrane fluidity modulation. The KEGG pathway reconstruction of the Leuconeurospora fungal genome unveiled its cold adaptation potential, represented by pathways for DNA repair, environmental stress defense, membrane stability and protein folding. The fungal genome harboured biosynthetic gene cluster for squalestatin S1, an anti-cholesterol compound.

P1-Z33 **Nasrin Banu Mohammed Faisal** National Centre for Cell Science, Pune

Investigating the Chaperone-mediated regulation of the Memory associated RNA-binding Protein Orb2

A central question in neuroscience is how memories are maintained over a lifetime despite rapid protein turnover.Previous studies have demonstrated that local protein synthesis plays a critical role in sustaining long-term memory.The RNA-binding protein CPEB/Orb2 plays a key role in memory persistence through functional prion-like Oligomerization.Recent evidence indicates that the molecular chaperone Mrj (Mammalian Relative DnaJ) regulates Orb2 oligomerization via direct interaction.Notably,Mrj knockout models exhibit significantly reduced levels of Orb2 oligomers, indicating a critical role of Mrj in maintaining the functional oligomeric state of Orb2.Mrj possesses the ability to self-oligomerize and associate with translating ribosomes, suggesting that it may function as a ribosome-associated chaperone that facilitates the assembly of Orb2 oligomers during translation. The molecular mechanism underlying this novel role of Mrj chaperone remains largely unexplored.Here,we aim to define the structural and functional determinants of Mrj that governs Orb2 Oligomerization.Initial results from this study indicate that the C-terminal domain of Mrj is required for its self oligomerization and for its colocalization with both isoforms of Orb2 but not for its interaction with ribosome.Furthermore a T218A mutation in the C terminal domain of MRJ disrupts its self-oligomerization and its colocalization with Orb2.These findings suggest that higher order assembly of MRJ might be needed for its regulatory function towards Orb2 Oligomerization.This work underscores an emerging paradigm in which molecular chaperone are not merely protein-folding assistants but also play a key role in regulating the functional assembly of the RNA binding protein Orb2.

P2-Z34 Neethu C H

National Centre for Cell Science, Pune

Regulation of cGAS-STING pathway by Nup358

Nup358 is one of the nucleoporins that can be found at the nuclear envelope as well as in the cytoplasm at structures called Annulate lamellae (AL). Many dominant missense mutations in this nucleoporin are linked with a disease condition called Acute Necrotizing Encephalopathy-1 (ANE-1). One of the main characteristics of ANE-1 is the increased levels of pro-inflammatory cytokines in the serum and cerebrospinal fluid (CSF), after an infection often by a virus. How Nup358 mutations lead to increased inflammatory responses is not known. Whether Nup358 plays any role in innate or acquired immunity is not well-understood. As ANE-1 is triggered by infection mostly by viruses, it is possible that the viral nucleic acid (DNA or RNA) may induce the pro-inflammatory condition. One pathway that senses dsDNA in the cytoplasm is the cGAS-STING pathway. As viral infection is characterized by the entry of viral DNA/RNA into the cytoplasm and ANE1 conditions arise usually after a viral encounter, we speculate a potential role for Nup358 in regulating the intracellular DNA/RNA sensing inflammatory pathway.

Results in HeLa and HEK293T cell lines show that Nup358 depletion results in reduction in STING protein levels without any change in the transcript levels. Interestingly, depletion of other nucleoporins like Nup214 or Nup98 does not show the same phenotype as Nup358. Also, co-immunoprecipitation assays suggest an interaction of STING with Nup358. Understanding the mechanism by which Nup358 contributes to restriction of cGAS-STING pathway is underway. Further studies are also focused on testing if ANE-1 linked mutations of Nup358 compromises this function.

P1-Z35 P. Sreeja

Symbiosis School of Biological Sciences

Correlation between citrate synthase gene sequence variation, expression, and citric acid production in Aspergillus niger

Citric acid (CA) overproduction by Aspergillus niger remains an unresolved physiological phenomenon despite over a century of industrial application. Recent metabolic engineering approaches primarily focused on genetically modified laboratory strains by targeting specific genes such as citrate synthase (citA) and the citrate exporter (cexA), which provides limited insight into the natural genetic variation associated with citric acid production. This study investigated the relationship between citA gene sequence variation, citA gene expression, and CA yield among thirty native A. niger isolates. Genetic variation was assessed using Polymerase chain reaction-restriction fragment length polymorphism (PCR- RFLP) employing two iterations of restriction digestion with NcoI, SacI, and HindIII. Restriction profiles were analyzed by agarose gel electrophoresis, fragment sizes were estimated using ImageJ, and genetic relationships were determined using Dice similarity coefficients and UPGMA cluster analysis. A pilot fermentation study was also performed to identify the optimum sampling time for citA transcript by RT- qPCR. PCR- RFLP generated a distinct restriction fragmentation pattern across the isolates, and dendrogram analysis revealed clustering of isolates based on predicted citA sequence variation. The study suggests that the variations in citA gene sequences can be used as a reliable marker for identification of the high citric acid producing A. niger strains from the natural sources. Moreover, it would be interesting to understand the CitA gene expression variation across these isolates and their correlation with citric acid production, if any, to develop a better understanding of the variation in citric acid yields.

P2-Z36 Pratyay Ghosh

National Centre for Cell Science, Pune

Investigate the dynamics of atypical epigenetic regulation during hematopoiesis, emphasizing their contribution to anemia in cancer.

Cancer-induced anemia (CIA) and chemotherapy-induced anemia (ChIA) are major hematological complications that significantly impair patient quality of life and treatment outcomes. While conventional studies have largely focused on histone H3 and H4 modifications, the contribution of atypical histone H2A modifications and variants to disrupted hematopoiesis remains poorly understood. This study aims to investigate the role of atypical epigenetic regulators, specifically H2AT120 phosphorylation (H2AT120p), H2AK119 ubiquitination (H2AK119ub), and the histone variants H2A.Z and MacroH2A1/2, in the pathogenesis of cancer-associated anemia.

Using established syngeneic mouse models of ovarian cancer-associated anemia and complementary in vitro hematopoietic stem and progenitor cell (HSPC) culture systems, genome-wide profiling of these histone marks and variants will be performed through CUT&RUN sequencing. Integration of epigenomic datasets with RNA sequencing and quantitative gene expression analyses will identify key regulatory networks and genes involved in erythropoiesis that are altered under cancerous and chemotherapeutic stress. Particular emphasis will be placed on understanding how aberrant chromatin landscapes influence hematopoietic differentiation and anemia progression.

Furthermore, the therapeutic potential of targeting atypical histone modifications will be evaluated using the VprBP inhibitor B32B3 to reverse H2AT120 phosphorylation–associated defects and restore erythroid gene expression. The study is expected to uncover novel epigenetic mechanisms governing hematopoiesis, identify biomarkers associated with anemia severity, and establish new therapeutic targets for cancer-related anemia. These findings may contribute to the development of precision epigenetic interventions aimed at improving supportive cancer care and patient outcomes.

P1-Z37 Priyanka S Pidugu

Ramaiah University of Applied Sciences

Bioactive Human Amniotic Membrane as a Next-Generation Scaffold for Bone Regeneration

Bone defects arising from trauma, infection, tumour resection, or non-union fractures remain a major clinical challenge, as current grafting options are limited by donor site morbidity, immune-related concerns, and inadequate osteoinductive capacity. This study investigates human amniotic membrane (AM) as a bioactive scaffold for bone regeneration. AM, a placenta-derived tissue, is naturally enriched with extracellular matrix components, growth factors, and immunomodulatory molecules that support cell attachment, proliferation, and tissue repair. In this work, AM was processed using standardized tissue bank protocols, and its structural integrity and scaffold architecture were evaluated. Histological analysis confirmed preservation of matrix organization, while microscopy, mechanical testing, and biochemical assays are being used to assess porosity, mechanical stability, and retention of bioactive factors. Human amniotic mesenchymal stem cells (hAMSCs) and stromal cells were successfully isolated and expanded in culture, displaying characteristic spindle-shaped morphology and high proliferative potential. Their mesenchymal and stromal identity is being validated through surface marker profiling and trilineage differentiation assays. Following osteogenic induction, hAMSCs will be seeded onto AM scaffolds to develop a bone-forming construct. The construct will be assessed for its ability to support osteogenic differentiation, modulate inflammation, and promote angiogenesis, which are critical for successful bone healing. Collectively, this study positions AM as a safe, cost-effective, and clinically relevant scaffold with strong translational potential for the repair of critical-sized bone defects and non-union fractures.

P2-Z38 Priyanka V

M S Ramaiah University of Applied Sciences

Decoding the recurrent glioblastoma multiforme (rGBM) tumor microenvironment (TME) through Explainable AI and single cell transcriptomics

Glioblastoma multiforme (GBM) remains one of the most lethal malignancies, characterized by high rates of recurrence and poor survival outcomes. Despite extensive research, the differences between the tumor microenvironment (TME) of primary (pGBM) and recurrent (rGBM) glioblastoma are poorly understood, which severely limits clinical options for post-recurrence therapy. This study aims to characterize these transitions and investigate signatures of the therapeutic-resistant TME alongside immune remodeling.We utilized bulk (discovery cohort) and single-cell RNA sequencing (validation cohort) data to profile cell-state and TME programs, to assess the shifting landscape of tumor heterogeneity between pGBM and rGBM. We performed differential cell-state analysis, machine learning combined with an Explainable AI (xAI) framework, and pseudobulk expression analyses, alongside cell-cell interaction profiling, to provide a mechanistic interpretation of complex transcriptomic data. Bulk RNA analysis identified T-cell senescence, myeloid dominance (specifically M2like tumor-associated macrophages, or TAMs), IFN-γ-mediated inflammatory signaling, and mesenchymal plasticity as major contributors to the rGBM TME. Our integrative approach generated a high-resolution map of the rGBM TME, identifying specific tumor glial ligand genes correlated with a poor prognosis and interacting with immunosuppressive programs in rGBM patients. This study advances our mechanistic understanding of pro-tumor immunity and therapeutic resistance. These computational insights shed light on the interplay between TAMs and the complex, immune-modulatory, and phagocytic regulatory programs governing the rGBM TME, providing a robust foundation for future efforts to overcome treatment resistance.

P1-Z39 Raksha Bhansali

Indian Institute of Science Education and Research (IISER), Pune

The Suppressor of Clathrin Deficiency 5 (Scd5) Directly Mediates Membrane Fission

Clathrin-mediated endocytosis (CME) is a critical vesicular trafficking pathway that internalises cell-surface cargo. This process involves reshaping the flat plasma membrane into an invagination, followed by membrane scission to release the vesicle. While the GTPase dynamin drives scission in mammalian cells, the mechanism in yeast remains elusive. Our recent work shows that the yeast dynamin Vps1 catalyses the GTP-dependent fission of phosphatidylinositol (4,5)-bisphosphate (PI(4,5)P2) membrane tubules in supported membrane templates (SMrTs). However, deleting VPS1 does not impair in vivo uptake of the CME cargo Mup1, suggesting Vps1 is dispensable for yeast CME and that dynamin recruitment into CME is a recent evolutionary development. While mammalian BAR-domain proteins recruit dynamin, current yeast models suggest the endocytic BAR-domain Rvs161-167 heterodimer facilitates fission via membrane phase separation and actin forces. Yet, on SMrTs, purified Rvs161-167 induces only nanotube constriction, not full scission. This suggests either a reconstitution limitation or the involvement of an unidentified factor. Notably, the essential gene SCD5 (Suppressor of Clathrin Deficiency 5), a genetic suppressor of clathrin heavy chain defects, regulates late-stage secretory transport to rescue clathrin-deficient yeast lethality. Remarkably, we discover that purified Scd5 directly catalyses the fission of PI(4,5)P2 membrane nanotubes in a nucleotide-independent manner. This is the first report characterizing the direct membrane-binding and scission capabilities of Scd5. Future work will dissect the mechanisms underlying Scd5 membrane activity and evaluate its necessity in Mup1 uptake.

P2-Z40 Ravish Javed

National Centre for Cell Science, Pune

How does SARS-CoV-2 protease PLpro modulates host UPS function

Understanding the alteration in structural and functional dynamics of host macromolecular complexes upon virus infection is crucial in understanding disease mechanism. My doctoral research is centered on investigating how one of the most vital proteases of SARS-CoV2 influence the human Ubiquitin-Proteasome System (UPS) and alter host protein homeostasis. The UPS maintains a healthy immunity by degrading the pathogen derived proteins. However, a few pathogens during evolution have developed mechanisms to hijack host defense system, of which antagonizing ubiquitin signaling is one of them. Interestingly one of the SARS-CoV2 protease, mimics a human deubiquitinase enzyme (DUB) & removes ubiquitin units from the target proteins in host cells and reverses protein function, preferably for its own survival and therefore modulate the host machinery thus perhaps understanding this interaction becomes one of the crucial avenues.

P1-Z41 Riddhi Bharadwaj

National Centre for Cell Science, Pune

Direct Bacterial Stimulation Alters Hematopoietic Stem Cell Transcriptional Identity and Activated Innate Immune Signalling

Hematopoietic stem cells (HSCs) sustain lifelong blood production and can sense microbial signals, yet how bacteria influence HSC function remains unclear. This study investigates transcriptional and signaling changes in mouse bone marrow HSCs following bacterial stimulation. LSK $CD150^{+}$ $CD48^{-}$ HSCs were stimulated in vitro with heat-killed Pseudomonas aeruginosa (HKPA) or Staphylococcus aureus (HKSA) (MOI 50). Bulk RNA-seq was performed at 12 h post-stimulation, and activation of NF-κB, ERK, and c-Jun pathways was assessed by western blot (1–6 h). HKPA stimulation resulted in 317 upregulated and 83 downregulated genes. Stemness-associated genes, including Erg, were reduced, while upregulated genes included lymphoid markers (Ebf1, Cd79a), innate receptors (Tlr2, Tlr6, Nod2), transcription factors (Gata2, Gata3), and inflammatory mediators (Il1β, Ccl3, Cd69). Both HKPA and HKSA induced progressive activation of NF-κB, ERK, and c-Jun signaling. These findings demonstrate that bacterial exposure rapidly shifts HSCs from a stemness program toward inflammatory activation and lineage priming, potentially compromising self-renewal. Targeting TLR and NOD signaling pathways may offer strategies to preserve HSC function during infection.

P2-Z42 Sampurna Guha

Indian Institute of Technology Gandhinagar

Biochemical characterization of the amino-truncated human THAP9 isoform

THAP9 (THanatos-Associated Protein 9), belonging to the human THAP family, is homologous to the well-characterised Drosophila P-element DNA transposase (DmTNP), with similar domain architecture containing an N-terminal THAP domain, Oligomerization domain, RNase H-like catalytic domain, and C-terminal domain. Despite sharing this similarity, human THAP9 lacks terminal inverted repeats (TIRs) and target site duplication (TSDs) and is predicted to be a molecularly domesticated transposon-derived gene. However, human THAP9 retains its catalytic activity and can mobilise the P-element transposon in HEK293 cells.

The THAP domain of DmTNP is an established DNA-binding domain which binds to P element DNA. However, the role of the THAP9 THAP domain is unknown, although it is predicted to bind DNA. Interestingly, NCBI has recently annotated novel human THAP9 isoforms that lack the amino-terminal end, including the THAP domain and oligomerization domain. Here we demonstrate that these truncated human THAP9 isoforms surprisingly retain significant DNA integration activity. This suggests that the THAP domain and oligomerization domain of THAP9 are not essential for its catalytic activity. It is interesting to note that our bioinformatic analysis has previously predicted an intrinsically disordered region between the THAP domain and the oligomerization domain of THAP9. Thus it is tempting to assume that this predicted disordered region, by virtue of its inherent structural flexibility, may regulate THAP9 function. These intriguing observations lead us to question the exact roles of the amino-terminal domains of THAP9 including the disordered region as well as the physiological relevance of the truncated THAP9 isoforms.

P1-Z43 Sannidhya De Indian Institute of Science Education and Research (IISER), Pune

α-Synuclein acts as a physiological mitochondrial uncoupler to regulate membrane potential

The homeostatic maintenance of mitochondrial membrane potential (MMP) requires a precise balance between potential generation and regulated dissipation. While the mechanisms driving MMP generation via the electron transport chain (ETC) are highly conserved, those governing physiological dissipation remain poorly understood. In specialized brown adipose tissues, uncoupling proteins (UCPs) facilitate proton permeation across the inner mitochondrial membrane for thermogenesis, utilizing fatty acids as intermediates. However, the mechanisms responsible for homeostatic uncoupling in non-adipose systems remain elusive. Hypothesizing that an uncoupling pathway would require membrane-active proteins, we screened brain lysates for membrane integrity-disrupting (MID) factors using Supported Membrane Templates (SMrT). In this assay, highly curved membrane nanotubes undergo catastrophic fission when exposed to membrane-active proteins. By profiling SMrTs composed of diverse lipid species, we identified the Parkinson's disease (PD)-linked protein α-synuclein (α-syn) as a novel regulator of membrane integrity. This activity showed strict specificity for templates containing the mitochondrial lipid cardiolipin (CL). We discovered that while α-syn is sufficient to disrupt CL-containing membrane nanotubes, its activity is strictly regulated on planar membranes. On these flat surfaces, it preserves membrane integrity but permits proton permeation through membrane defects. Validating this screening rationale, knocking out α-syn in mammalian cells caused significant mitochondrial hyperpolarisation. Mechanistically, a fraction of α-syn localizes to the mitochondrial intermembrane space, where it actively dissipates membrane potential by generating transient membrane defects that allow proton leakage. Remarkably, familial PD-linked α-syn mutants lacked defect-forming capabilities, and could not rescue mitochondrial hyperpolarisation. Together, our findings establish a native, physiological role for α-syn as a generic mitochondrial uncoupler, directly linking PD pathogenesis to a loss-of-function failure in membrane potential regulation.

P2-Z44 Sanskriti Chavan Institute of Bioinformatics and Biotechnology (IBB), SPPU

Mechanistic insights into free fatty acid induced lipotoxicity and its role in the development of insulin resistance

Type 2 Diabetes mellitus (T2DM) is a metabolic disorder characterized by the development of insulin resistance and impaired glucose homeostasis. Skeletal muscles play a major role in maintaining glucose homeostasis and are important sites for insulin-stimulated glucose uptake. They are susceptible to lipotoxic conditions arising from excess free fatty acid exposure in nutrient excess conditions, similar to those in T2DM, confounded with obesity. Previous studies have shown that exposure to saturated free fatty acids, such as palmitic acid and stearic acid induce oxidative stress that disrupts insulin signaling, whereas unsaturated fatty acids have been reported to show protective effects against lipotoxic stress. In this study, we investigated the effects of saturated and unsaturated FFAs on skeletal muscle using the mouse-derived C2C12 and the rat-derived L6 skeletal myotubes, alongside a physiologically relevant human model generated by differentiating umbilical cord-derived mesenchymal stem cells (UCMSCs) into multinucleated skeletal myotubes. Antioxidants such as glutathione have been known to show protective effects against free fatty acid- induced lipotoxicity. GSH was found to be effective against PA- mediated cytotoxicity by reducing intracellular lipid accumulation. Further, the effect of cell secretome therapy or conditioned media on saturated fatty acid-mediated lipotoxic stress in these lines was attempted. Collectively, these findings
demonstrate the effects of saturated and unsaturated fatty acids on skeletal muscle and highlight the potential of antioxidant treatment. The results from these investigations will be presented.

P1-Z45 **Sarvesh Raykar** National Centre for Cell Science, Pune

Conformation Sensitive docking by BMP2K/AAK1 promotes fidlity of AP2 phosphorylation during CME.

Clathrin-mediated endocytosis (CME) is a major vesicular transport mechanism in eukaryotes for internalization of cell surface transmembrane protein cargo. In vertebrates, CME is dependent on the recruitment of adaptor protein AP2 to the membrane. Regulation of endocytosis is largely dependent on post translational modifications of key endocytic proteins including AP2 which is phosphorylated on T156. Yet how these PTMs are regulated is not known. Here our work identifies a novel docking motif of kinase BMP2K which phosphorylates AP2. Using gene edited cells we demonstrate that absence of BMP2K leads to endocytic defects inside the cells. Additionally in our study we try to address the long standing question of how BMP2K can recognize different conformations of AP2 by demonstrating the presence of two domains BMP2K (346-389) and BMP2K(735-808) which can differentiate between different conformations of AP2 and thereby regulates phosphorylation in context dependent manner.

P2-Z46 **Savani Jayant Pathak** Agharkar Research Institute

Neighbours matter: Identifying Niche-derived regulators in Stem Cell Autophagy

Autophagy is a lysosomal degradation process that clears damaged organelles and misfolded proteins to maintain cellular homeostasis. Most studies have examined its cell-autonomous regulation, yet a few reports indicate that autophagy can also be controlled by a cell, non-autonomously. The factors mediating this non-autonomous regulation remain largely unknown. To identify them, we conducted a targeted screen in the Drosophila germarium, the anterior tip of the ovary, and an ideal tissue to study communication between germline stem cells (GSCs) and their somatic niche. Using an in silico screen, we identified candidate secretory and membrane-bound factors expressed in niche cells that may regulate autophagy in GSCs (and GCs). We then used the UAS-Gal4 system to knock down each candidate by RNAi specifically in the niche and assayed its effect on autophagy in the neighboring GSCs (and GCs). We used two classic readouts: Ref(2)P, whose accumulation indicates impaired autophagy, and Atg8a, which translocate from the cytoplasm to the autophagosome upon induction of autophagy. Autophagy was monitored by mCherry-Atg8a localization to autophagosomes and by Ref(2)P/p62 clearance. Several candidates altered both Atg8a localization and Ref(2)P levels in GSCs (and GCs), indicating that these niche-derived factors regulate autophagy in GSCs (and GCs) non-cell-autonomously. Further work is underway to define the mechanisms by which these proteins regulate autophagy in GSCs (and GCs).

P1-Z47 **Seinjuti Nath** National Centre for Cell Science, Pune

Understanding the proteome and metabolome alterations in secondary organ upon triple negative breast cancer metastasis

Triple-negative breast cancer (TNBC) is among the most aggressive subtypes of breast cancer and accounts for a large proportion of cancer-related deaths in India and globally. TNBC exhibits a strong tendency for organotropism, the preferential metastasis of cancer cells to specific distant organs, particularly the lungs and liver. Although circulating tumor cells (CTCs) can disseminate throughout the body, the molecular mechanisms that drive their selective colonization of these organs remain poorly understood. In this study, 4T1 cells were cultured and orthotopically injected into the mammary fat pad of female BALB/c mice to establish a spontaneous TNBC metastasis model.

To investigate the molecular events underlying organotropic metastasis, we performed comprehensive proteomic and metabolomic profiling of the peripheral tumor microenvironment (TME) in the lungs and liver. Characterizing the peripheral proteomic landscape and associated metabolic pathways enables the identification of molecular alterations that create a permissive metastatic niche and support tumor cell survival, colonization, and outgrowth in these organ-specific microenvironments. Overall, the objective of this project is to uncover organotropism-associated molecular drivers, characterize the peripheral tumor microenvironment proteomic landscape and metabolomic pathways, and identify supportive signalling networks involved in TNBC metastasis. These findings will contribute to a deeper understanding of the molecular landscape governing organ-specific metastatic spread and may reveal potential biomarkers and therapeutic targets for metastatic TNBC.

P2-Z48 **Shiva Kumar Tomar** National Centre for Cell Science, Pune

Exploring the role of an F-box Protein, FBXO34 in Cancer

Cancer, remains one of the leading causes of mortality worldwide. This high mortality rate is attributed to the dysregulation of various cellular processes, including the turnover of proteins via ubiquitination. The SCF (Skp1-Cullin1-F box proteins) E3 ligase complex, a pivotal unit in ubiquitination, features highly variable F-box proteins. Despite their significance, the role of these proteins in cancer regulation remains underexplored. Our lab screened several F-box proteins and observed significant growth inhibition by FBXO34 (F-box Only 34 protein) in the MCF-7 cell line. Thus, we hypothesize that FBXO34 may act as a tumor suppressor. We aimed to understand its role in cancer phenotypes and its important mechanism in malignancy. Further investigation revealed that FBXO34 functions as a tumor suppressor by inhibiting the proliferation, colony formation ability, and migration of breast cancer cells. Notably, FBXO34 downregulates the protein level of Vimentin, a key mesenchymal marker involved in the migration and invasion of cancer cells. Further, we plan to conduct additional key experiments to understand the underlying mechanism of tumor suppression. So far, the data suggest that FBXO34 plays a critical role in the cancer regulation and degrades a key oncogenic protein. These findings highlight the importance of FBXO34 in the regulation of breast cancer progression. Consequently, our study may discover a new molecular player associated with cancer malignancy and potentially lead to novel therapeutic strategies.

P1-Z49 Shivarkar Sejal Sachin Indian Institute for Science Education and Research, Pune

A Novel Mechanism Regulating Endosomal Protein Sorting

Cellular compartmentalization relies on a precise balance between membrane protein recycling and degradation, yet the underlying regulatory mechanisms remain poorly understood. These pathways converge at the endosomal hub, where protein cargoes are sorted into distinct membrane tubules for transport to target organelles. In yeast, while major sorting pathways—such as the retromer complex—rely on a trimeric cargo-selective complex (CSC) coupled with membrane-remodelling proteins, the sorting nexin Mvp1 operates independently of the CSC to traffic the transmembrane protein Vps55. The mechanisms coordinating Mvp1-mediated membrane remodelling and cargo recognition remain elusive. Here, we demonstrate that the unstructured N-terminal region of Mvp1 encodes an intrinsic autoinhibitory signal, restricting membrane binding and subsequent tubulation. This autoinhibited state exhibits minimal affinity for the endosome-specific lipid phosphatidylinositol 3-phosphate (PI3P), preventing premature tubule formation. Crucially, we show that this autoinhibition is relieved upon engagement of the N-terminus with Vps1, a downstream fission protein belonging to the Dynamin superfamily. Ultimately, our findings uncover a tightly regulated, novel mechanism of cargo sorting that may represent a widespread paradigm in eukaryotic membrane trafficking. Ongoing and future work will evaluate whether this regulatory mechanism is evolutionarily conserved in mammalian systems by investigating Sorting Nexin 8 (SNX8)—the mammalian ortholog of yeast Mvp1—and Dynamin.

P2-Z50 Sourabh Yadav National Centre for Cell Science, Pune

Antagonizing or deficiency of muscarinic receptor 3 (CHRM3) inhibits tumor growth

Emerging evidence suggests that neurons interact with immune cells via neurotransmitters, thereby modulating their phenotype and function during cancer progression. Neurotransmitters, such as acetylcholine, produced by neurons signal through either nicotinic or muscarinic acetylcholine receptors (CHRMs). These receptors are known to increase cell division, proliferation, and metastasis in colon cancer cells. However, the mechanism by which CHRMs modulate immune cell function in colon cancer remains unclear.

A subcutaneous tumor was induced in wild-type C57BL/6 or CHRM3-/- mice by injection of the mouse colon cancer cell line MC38. Expression of muscarinic receptors and other molecules in various tissues and immune cells was analyzed using quantitative RT-PCR, Immunohistochemistry (IHC), ELISA, and multicolor spectral-flow cytometry.

Our results showed that various immune cells and the colon cancer cell line MC38 express diverse muscarinic receptors (CHRM 1-5). Furthermore, MC38 tumors in CHRM3-/- mice exhibited reduced tumor growth, accompanied by increased tumor-infiltrating CD8 and CD4 T cells, a higher frequency of effector T cells (Tc1 and Th1), and a decrease in immunosuppressive T cells (Foxp3+ Tregs). Intraperitoneal injection of a CHRM3 antagonist, 4-DAMP, significantly reduced MC38 tumor growth in wild-type mice. Furthermore, antagonist treatment significantly altered the frequency and number of intratumoral cytotoxic CD8+ T cells (Tc1) and helper CD4+ T (Th1) cells. Adoptive transfer of tumor-primed lymph node cells from CHRM3-/- mice in tumor-bearing NRG mice (RAG1-/-IL-2R -/- mice; lack T, B, and NK cells) showed a significant protection from colon tumor growth compared to mice that received CHRM3+/+ tumor-primed lymphocytes.

Our results showed that CHRM3 signaling modulates anti-tumor immunity, suggesting that interfering with CHRM3 signaling is an interesting therapeutic strategy to boost anti-tumor immunity and control tumor growth.

P1-Z51 Sourav Ganguly

IIT Gandhinagar

Characterisation of the nucleotide-binding properties of the THAP domain of hTHAP9

Transposable elements (TEs) are genomic architects that have shaped genomic landscapes through the course of evolution. Human THanatos-Associated Protein 9 (hTHAP9) is a TE-derived protein, homologous to the well-studied active Drosophila P-element transposase (DmTNP). Interestingly, hTHAP9 retains the ability to excise and integrate P-element DNA. Members of the THAP family of proteins are distinguished by the presence of an N-terminal THAP domain having a Zn- coordinating C2CH motif within a conserved β-α-β secondary fold, which has been shown to strongly bind DNA in DmTNP. Our preliminary observations demonstrate that the hTHAP9-THAP domain binds DNA with low affinity; however, the specific binding site and its crucial binding residues remain elusive. To investigate the DNA-binding characteristics of hTHAP9, we performed multiple sequence alignment of its THAP domains with DmTNP and other THAP homologs, as well as structure-based alignment. In comparison to DmTNP, hTHAP9-THAP domain had two short insertions within the C2CH motif, and the C-terminal end of the DmTNP-THAP domain had a longer loop. To study the roles of these regions, hTHAP9 mutants (by residue removal or addition) were created, and their ability to bind and integrate DNA was investigated via biochemical assays. Preliminary studies demonstrate that the hTHAP9 insertion mutant had a significantly increased ability to integrate DNA as compared to wild-type. Interestingly, in our MD simulation study, one deletion mutant showed an increased solvent-accessible surface area per residue compared to wild-type, suggesting an enhanced ability to interact with DNA. Future comprehensive analysis of hTHAP9 mutants will help elucidate its DNA- binding functions.

P2-Z52 Subhankar Das

National Centre for Cell Science, Pune

Deciphering the role of Hematopoietic PBX Interacting Protein (HPIP) in Multiple Myeloma pathogenesis

Multiple myeloma (MM) accounts for 13% of all haematological malignancies worldwide and is a result of abnormal proliferation of clonal plasma cells. In preliminary investigation, using SWATH-MS it was observed that HPIP was significantly upregulated in MM cell lines (MM1S and U266B1) in comparison to control cell line (RPMI 1788). For validation, western blot experiment was done, which showed similar results to SWATH-MS i.e. HPIP was found significantly upregulated in MM1S *&* U266B1, compared to RPMI 1788. Further, qRT-PCR data showed that the HPIP gene is not only upregulated at the protein level, but it is also significantly upregulated at the RNA level in MM1S and U266B1 in comparison RPMI 1788. Analysis of patient plasma cell transcriptomic datasets from the Gene Expression Omnibus (GEO) database revealed HPIP expression was significantly elevated during the progression from precursor conditions to full-blown stages i.e. MGUS, SMM, and MM, when compared to normal plasma cells [GEO accession: GSE47552]. HPIP knockdown in the U266B1 is achieved and cell counting assay showed that the growth of U266B1-shHPIP has significantly decreased compared with U266B1-SCR. Furthermore, SWATH-MS comparing U266B1 control and HPIP knockdown was performed to identify HPIP targets in MM. Bioinformatic pathway analysis using ShinyGO 0.85 showed pathways like apoptosis, the VEGF signalling pathway, the AMPK cascade, the phosphatidylinositol system, and the HIF-1 signalling pathway. Candidate members of the pathways that were significantly altered are, KRAS, PIK3CB, PIK3C2A, PIK3R2 etc. These proteins will be further validated by western blot. Immunoprecipitation Mass Spectrometry was performed and identified PARP1, YB-1, XPO1 and MMTAG2 as a critical interacting partners of HPIP in MM. Currently, we are validating these interacting partners and understanding the molecular mechanism of HPIP mediated MM pathogenesis.

P1-Z53 Sudarshan Mandal

National Centre for Cell Science, Pune

Neuroimmune Communication of Muscarinic (M3) Receptor in Psoriasiform Skin Inflammation

Psoriasis is a chronic, recurrent inflammatory disease characterized by keratinocyte hyperproliferation, epidermal thickening, scaling, and immune cell infiltration. Although it is a disorder of immune dysregulation, recent studies suggest that the nervous system influences skin immunity. Acetylcholine (ACh), an important neuroimmune mediator, modulates immune-cell function through muscarinic acetylcholine receptors. In our study, we investigated the contribution of M3R signaling using imiquimod (5%, w/v)-induced psoriasiform skin inflammation in C57BL/6 mice.

Our data showed that Imiquimod (IMQ) treatment of M3R-deficient mice significantly attenuated disease severity, as evidenced by reduced erythema, epidermal thickness, and splenomegaly compared with wild-type (WT) mice. Immunohistological analysis of skin tissues showed that M3R knockout mice had reduced expression of TLR7 and the endothelial marker CD31, indicating diminished inflammatory and angiogenic responses. Flow cytometric analyses revealed a marked reduction in $CD11b^+$ myeloid cells, $Ly6G^+$ neutrophils, Ly6C+ monocytes, and $CD11c^+$ dendritic cells within the inflamed skin of M3R Knockout mice compared to wild-type mice. Treatment of wild-type mice with the M3R pharmacological antagonist 4-DAMP reduced the psoriatic score to levels similar to those of M3R-deficient mice. Histological examination further demonstrated reduced inflammatory T-cell infiltration ($\gamma\delta TCR^+$ and RORγt+) in M3R-deficient mice compared with WT mice.

Collectively, these findings showed that M3R is a critical regulator of psoriasiform skin inflammation. Further, we provide a mechanistic insight into cholinergic neuroimmune signaling through M3R, which promotes inflammatory cell recruitment, angiogenesis, and type 17 immunity, thereby regulating psoriasis.

P2-Z54 Sukirtee Sahoo

National Centre for Cell Science, Pune

Role of estrogen receptor signaling on immune response to Human Papillomavirus vaccine antigen

Sexual dimorphism in vaccine responses is well documented, with females mounting stronger immune responses than males across multiple vaccine platforms. Sex hormones are thought to contribute to this disparity, yet the mechanisms through which hormonal signaling regulates antigen-specific adaptive immunity remain poorly understood. Recent evidence suggests that hormone receptor expression on lymphocytes may directly regulate immune responses. Given that the Human Papillomavirus (HPV) vaccine is administered to adolescents during a period of significant hormonal transition, it offers a unique model to investigate hormone-mediated regulation of vaccine immunity. We therefore hypothesized that estrogen receptor (ER) signaling modulates immune responses induced by immunization with HPV vaccine antigen.

To study this, HPV16 virus-like particles (VLPs) were used as a model vaccine antigen to immunize the C57BL/6 female mice. Immunization with HPV16 VLPs increased ER expression in secondary lymphoid organs, suggesting an important role for ER in vaccine-induced immune activation. Furthermore, in vitro analyses of stimulated female murine B cells indicated that ER signaling may regulate intracellular pathways involved in antibody diversification. Pharmacological modulation of ER signaling altered activation-induced deaminase (AID) expression, suggesting a potential role for ER signaling in regulating antibody diversification.

These findings provide preliminary evidence that ER signaling may contribute to the regulation of vaccine-induced B-cell responses. Ongoing studies employing ovariectomy and hormone replacement models aim to define how hormonal status influences HPV vaccine-induced immunity. Studying these mechanisms may explain hormone-specific differences in vaccine responses and inform strategies to improve vaccine efficacy.

P1-Z55 Suraj Kumar Meena National Centre for Cell Science, Pune

Elucidating the mechanism of 26S Proteasome impairment in hypoxic microenvironment in Cancer

Solid tumor proliferating cells that are farther from blood vessels develop a hypoxic region and lose direct access to oxygen and nutrients. The hypoxic microenvironment induces oxidative stress, leading to the accumulation of unfolded or misfolded proteins, which in turn cause proteotoxicity. To maintain proteostasis and survive, tumor hypoxic cells may rely on the major protein degradation system, that is, the Ubiquitin-proteasome system (UPS). In UPS, the free 20S complexes are relatively resistant to oxidation damage compared to the 26S Proteasome holo-complex. In our previous report, we discovered that the 20S proteasome often plays a role as an emergency proteasome under stress conditions, such as oxidative stress and hypoxia. Under hypoxia 26S proteasome breaks down to form free 20S proteasome complex which acts as an independent proteolytic enzyme. Altering the proteasome species ratio may be a strategy that cells utilise to survive under proteotoxicity. However, the underlying mechanism controlling this disassembly of 26S Proteasome into its 20S core and 19S regulatory sub-complex during hypoxia is still not clearly understood. My PhD research aims to investigate the dynamic behaviour of the proteasome system and how it undergoes structural and functional modifications in response to hypoxic stress. The study offers new insights into how tumor cells regulate their proteasome activity to survive in a hypoxic microenvironment. This will elucidate the survival strategy of cancer tumor and identify the therapeutic possibilities by obstructing proteasome regulation and enhancing the susceptibility of tumor cells to oxidative stress and anticancer therapies.

P2-Z56 Swati Tanti National Centre for Cell Science, Pune

Understanding the molecular mechanism of serotonin receptor HTR2B - driven neuroimmune crosstalk and its effects on anti-tumor immunity

Serotonin signaling has been identified as an important regulator of tumor development and immune regulation in colorectal cancer. HTR2B is one of the serotonin receptors that are increasingly recognized as crucial modulators of tumor signaling in the microenvironment. Recent research reveals that HTR2B promotes colorectal cancer cell proliferation, migration, and metastasis via pathways including ERK and Akt/mTOR-dependent signaling, as well as immune-related processes such as macrophage polarization and inflammatory signaling. However, the exact role of HTR2B in influencing the tumor microenvironment and the underlying signaling pathways, remains poorly understood. In this study, we are investigating HTR2B signaling in colorectal cancer using a combination of flow cytometry, qRT-PCR, Western blotting, and HTR2B-knockout cell lines. Our preliminary results indicate that HTR2B knockout cells exhibit slower growth in vivo and impaired tumor formation, suggesting that HTR2B may contribute to tumor viability and progression. By analyzing downstream signaling events and cellular phenotypes, this work aims to define how HTR2B regulates the tumor microenvironment and cancer cell behavior. We also aim to identify the immune cells that play a significant role in the tumor microenvironment and how these immune cells and HTR2B interact to shape it. These findings might provide mechanistic insight into serotonergic signaling in colorectal cancer and highlight HTR2B's potential as a therapeutic target for immune-based interventions.

P1-Z57 Uma Swaminathan Indian Institute for Science Education and Research, Pune

ATG18-mediated membrane budding and sequestration are critical for both vacuole shape maintenance and autophagy

Organelle homeostasis relies fundamentally on proteins that actively remodel cellular membranes. While the phosphoinositide-binding PROPPIN family protein ATG18 is known to be crucial for autophagy and vacuolar membrane maintenance in yeast, its exact molecular mechanism has remained poorly defined. In this study, we uncover a novel membrane-remodeling activity of ATG18. Integrating in vitro reconstitution, quantitative microscopy, and yeast cell biology, we demonstrate that ATG18 drives membrane sequestration by forming bud-like structures on phosphoinositide-containing bilayers. Disrupting the oligomerization interface—identified from the recently solved membrane-bound structure of ATG18—selectively abolishes this budding activity while leaving membrane-binding capability completely intact. In vivo, these oligomerization-deficient mutants fail to rescue autophagic defects and induce abnormally enlarged vacuoles, validating the physiological relevance of this interface. Furthermore, because ATG18 complexes with the lipid transfer protein ATG2, we propose that ATG18-mediated membrane sequestration directly orchestrates the vectorial flow of lipids from donor compartments to the nascent autophagosome. Ultimately, our work identifies the molecular determinants governing ATG18-driven membrane remodeling, offering new insights into how this structural activity maintains vacuolar architecture and fuels autophagy.

P2-Z58 Vandna Maurya National Centre for Cell Science, Pune

Identification and characterization of MFS transporter proteins associated with lysosome-related organelles in Tetrahymena thermophila

In the ciliate Tetrahymena thermophila, a subset of secretory proteins can be exocytosed via specialized vesicles called mucocysts. Mucocysts belong to the very broad family of lysosome-related organelles (LROs) including secretory LROs in animal cells, whose formation involves contributions from both secretory and endocytic trafficking. Despite the importance of LROs, the biogenesis pathway of these organelles in animals is not fully understood. Being a simpler organism than animals, Tetrahymena makes it easier to study LRO biogenesis that could be applicable to animals.

We extended the analysis of mucocyst/LRO biogenesis in Tetrahymena, focusing on its reliance on the major facilitator superfamily (MFS) of transporter proteins, by taking advantage of the many tractable features of this single-cell model organism. MFS is one of the largest and most diverse superfamily of secondary active transporters conserved from bacteria to humans. They are responsible for transporting essential molecules, including drugs and toxins, enabling them to be potential drug targets. However, MFS transporters in humans and other higher eukaryotes have received limited attention due to their biological complexity.

In this study, we began with expression profiling and found two MFS transporter genes (MFS8 and MFS9) in Tetrahymena that are targeted to mucocysts and associated with endolysosomal pathways. Cells lacking the MFS8 and MFS9 genes show defects in the mucocyst exocytosis, while the formation remains unaffected. Our findings indicate that MFS8 and MFS9 play essential roles in regulating mucocyst exocytosis, providing new insights into how evolutionally conserved MFS proteins co-ordinate the trafficking and exocytic efficiency of secretory cell organelles.

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