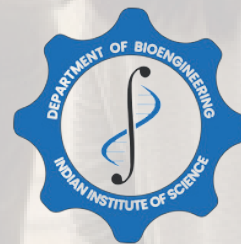


11th Annual Bioengineering Symposium

The Department of Bioengineering (BE) at IISc is an interdisciplinary hub that integrates engineering and life sciences. Research activities in BE are spearheaded by the students and faculty, who have procured competitive grants for studies related to bioengineering research. The Annual Research Symposium unites students, researchers, and professionals to explore breakthroughs in diagnostics, imaging, immune engineering, systems biology, and biomedicine.



EVENT BROCHURE

9-10, JANUARY 2025

TCS-SMART X HUB, IISC



Schedule

DAY

TIME

EVENT

9TH JANUARY
(THURSDAY)

1:30 PM (onwards)	Registration
2:30 PM	Welcome speech Prof. Kaushik Chatterjee, Chair, BE
2:35 PM	Dr. Neha Sharma Senior Advisor, British High Commission in India
3:00 PM	Poster session (tea, coffee, snacks)
5:00 PM	Ratnasri K (Trailblazer talk)

10TH JANUARY
(FRIDAY)

10:00 AM	Inaugural speech Prof. Navakanta Bhat, Dean, Interdisciplinary Sciences
10:10 AM	Introduction to Prof. Sanjay Biswas Memorial lecture
10:15 AM	Prof. Sanjay Biswas Memorial lecture- Prof. Vishal Rai, IISER Bhopal
11:00 AM	Tea/Coffee Break
11:15 AM	Prof. Prerna Sharma (Spotlight Talk)

Schedule

DAY

TIME

EVENT

**10TH JANUARY
(FRIDAY)**

11:50 AM	❖	Sarthak Sahoo (Trailblazer Talk)
12:05 PM	❖	Manasa Veena (Trailblazer Talk)
12:20 PM	❖	Prof. Rachit Agarwal (Spotlight Talk)
1:00 PM	❖	LUNCH
2:15 PM	❖	Prof. Siddharth Jhunjunwala (Spotlight talk)
2:50 PM	❖	Shivaani E (Trailblazer talk)
3:05 PM	❖	Dr. Janani Venkatraman (Spotlight talk)
3:40 PM	❖	Amrapali Datta (Trailblazer talk)
3:55 PM	❖	Arjun SV (Trailblazer talk)
4:05 PM	❖	Tea/Coffee Break
4:20 PM	❖	Dr. Ajay Tijore (Spotlight talk)
4:55 PM	❖	Closing remarks and vote of thanks

SANJAY BISWAS MEMORIAL LECTURE



Dr. Vishal Rai

Department of Chemistry,
IISER Bhopal

“Human behavior inspired protein engineering”

Our pursuit to understand human behavior starts from the day we are born. Since it offers an interplay of multiple dimensions, it can serve as a model and provide insight into other complex questions. We will discuss how it enabled the evolution of knowledge and core principles for precisely engineering proteins and antibodies. It rendered the technologies for homogeneous antibody-drug conjugates (ADCs) for directed cancer chemotherapeutics and fluorophore conjugates (AFCs) for imaging-guided tumour surgery. Besides, it creates an opportunity to realize precision therapeutics.

Dr. Vishal Rai obtained his Ph.D. in Chemistry from IIT Bombay under Prof. I. N. N. Namboothiri (2003-2008). He subsequently held a postdoctoral position and MITACS-Elevate fellow position in Prof. Andrei Yudin's group at the University of Toronto, Canada (2008-2011). His contributions to peptide macrocycles created the platform for Encycle Therapeutics. Later, he joined the Department of Chemistry at IISER Bhopal in 2011. His research group is leading the development of chemical technologies for the precision engineering of proteins. He is the Founder and Director of Plabeltech Private Limited. The state-of-the-art protein and antibody engineering technologies. He is an invited Fellow of the Royal Society of Chemistry (FRSC, UK) and an elected Fellow of the National Academy of Sciences (NASI, India). Recently, he was awarded the Rashtriya Vigyan Puraskar: Vigyan Yuva - Shanti Swarup Bhatnagar (2024).

Spotlight Sessions

“ Building Biomoneta: A hardware startup in a digital world”

Dr. Janani Venkatraman
CEO, Biomoneta



Janani received her scientific training via a PhD from the Indian Institute of Science. She helped found first Bugworks, a cutting-edge antibiotics discovery company, and then Biomoneta, the inventor of one of the world's most effective air treatment technologies with the potential to revolutionize infection prevention and the delivery of hospital-grade environments in resource-poor areas.

“UK – India Engineering Biology: A Collaborative Path to Global Solutions”

Dr. Neha Sharma
Senior Advisor, British High Commission in India



Neha leads on science policy and partnerships in Biotechnology, Engineering Biology, Anti-microbial Resistance and Neurosciences. She plays a pivotal role in fostering UK-India collaborations across government, academia, and industry to address global health and challenges and drive innovations in life-sciences. Her research experience encompasses natural product chemistry, with a particular focus on MS-based identification of molecular targets for natural products demonstrating therapeutic potential against ovarian cancer.

Spotlight Sessions

“ Collective phototaxis of Chlamydomonas reinhardtii”

Dr. Prerna Sharma
Associate Professor, IISc



Prof. Prerna Sharma has been a faculty member at IISc since 2014. She is an experimentalist with research interests in soft matter and biophysics.

“Immune Responses to Inflammatory Events in the Background of Chronic Inflammatory Conditions – the neutrophil perspective”

Dr. Siddharth Jhunjunwala
Associate Professor, IISc

Siddharth Jhunjunwala is an Associate Professor in the Department of Bioengineering, Indian Institute of Science. His research interests are in understanding the interactions of immune cells with biomaterial implants, especially in the context of type-2 diabetes.



Spotlight Sessions

“ Tuberculosis drug delivery and organoids”

Dr. Rachit Agarwal
Associate Professor, IISc

Rachit Agarwal is an associate professor at the Indian Institute of Science, Bangalore, India. His scientific interests are in developing biomaterial-based delivery vehicles for the treatment of inflammatory and infectious diseases.



“Mesenchymal stem cell osteogenesis by ultrasound-generated nanoscale mechanical perturbations”

Dr. Ajay Tijore
Assistant Professor, IISc

Ajay joined the Department of Bioengineering in 2021, where his 'Mechanobiologics Lab' works on investigating the effect of mechanical forces on cancer cell growth and regulating stem cell fate using custom-built microfluidic devices and micro/nanoscale biomaterials. Currently, he holds the R.I. Mazumder Young Investigator position.



Trailblazer Talks

“Rapid emergence of colistin resistance progresses through phenotypic tolerance and gene duplication”

Ratnasri K, Ph.D. Student

Colistin is considered a crucial last-line antibiotic and is often used to combat multidrug-resistant bacteria in humans and animals. However, many bacterial species have been documented to develop resistance to colistin. In addition, clinical bacterial isolates frequently exhibit mixed populations where a predominant susceptible variant masks the existence of low-frequency and unstable colistin heteroresistant (HR) cells. However, the mechanism for the emergence of HR variants and the establishment of permanent resistance to colistin is not well understood. We use a combination of drug kill assays, single cell imaging, genome sequencing, and mathematical modeling, to demonstrate that HR cells can spontaneously emerge within a few generations, even in laboratory strains of bacteria when exposed to colistin. An isogenic susceptible population consists of phenotypically tolerant cells that die at a slower rate than average susceptible cells when exposed to the drug. With continued exposure, these phenotypic tolerant cells acquire gene duplications in various stress and metabolic pathways that allows them to survive. This gene duplication (GD) ‘adaptation’ albeit is associated with growth fitness defects. Hence, the GD variants are unstable and easily lost in the absence of colistin. However, the occasional acquisition of site-specific point mutations in two component signalling genes associated with colistin resistance leads to stable colistin resistance in the population while the GD variants are lost. Overall, our study reveals that phenotypic tolerance, rapid gene duplication, and resistance fixation by site-specific mutations, rather than pre-existing resistant subpopulations, is a previously unexplored pathway for the emergence of colistin resistance, highlighting the need to target tolerant and transient bacterial intermediates to improve antibiotic treatment strategies.

“Dynamical modeling of proliferative-invasive plasticity and IFN γ signaling in melanoma reveals mechanisms of PD-L1 expression heterogeneity”

Sarthak Sahoo, Ph.D. Student

Phenotypic heterogeneity of melanoma cells contributes to drug tolerance, increased metastasis, and immune evasion in patients with progressive disease. Diverse mechanisms have been individually reported to shape extensive intra-tumor and inter-tumor phenotypic heterogeneity, such as IFN γ signaling and proliferative to invasive transition, but how their crosstalk impacts tumor progression remains largely elusive. Here, we integrate dynamical systems modeling with transcriptomic data analysis at bulk and single-cell levels to investigate underlying mechanisms behind phenotypic heterogeneity in melanoma and its impact on adaptation to targeted therapy and immune checkpoint inhibitors. We demonstrate that the emergent dynamics of our regulatory network comprising MITF, SOX10, SOX9, JUN and ZEB1 can recapitulate experimental observations about the co-existence of diverse phenotypes (proliferative, neural crest-like, invasive) and reversible cell-state transitions among them, including in response to targeted therapy and immune checkpoint inhibitors. These phenotypes have varied levels of PD-L1, driving heterogeneity in immunosuppression. This heterogeneity in PD-L1 can be aggravated by combinatorial dynamics of these regulators with IFN γ signaling. Our model predictions about changes in proliferative to invasive transition and PD-L1 levels as melanoma cells evade targeted therapy and immune checkpoint inhibitors were validated in multiple RNA-seq data sets from in vitro and in vivo experiments. Our calibrated dynamical model offers a platform to test combinatorial therapies and provide rational avenues for the treatment of metastatic melanoma. This improved understanding of crosstalk among PD-L1 expression, proliferative to invasive transition and IFN γ signaling can be leveraged to improve the clinical management of therapy-resistant and metastatic melanoma.

Trailblazer Talks

“Differential interfacial dynamics between oncogenic mutants and host epithelium dictate the outcome of cell-competition during cancer initiation across tissues”

Amrapali Datta, Ph.D. Student

Carcinomas, which constitute a majority of all cancers, arise from genetic alterations in healthy epithelial cells. Clinical data reveal that cancer driver genes are often mutated in a tissue-dependent manner, being altered in some cancers but not others. For instance, HER2 is a prominent driver in breast cancer, while oncogenic Ras are more prevalent in lung cancer. Additionally, often the prognosis and the therapeutic outcome are dependent on which cancer is being treated- raising the question of why certain mutations are more aggressive in specific tissues. This study investigates the mechanical aspects of the host epithelium, specifically how the interfacial dynamics between oncogenic mutants and wild-type cells influences the outcome of cell-competition during cancer initiation. Using mammary and bronchial epithelial models, we show that tissue-specific differences in interfacial tension between mutant and wild-type cells dictate the behavior of oncogenic populations. In mammary epithelia, higher interfacial tension between the two populations leads to the suppression of HRas-V12 mutants, while in bronchial epithelia, lower interfacial tension allows the mutants to evade elimination and exhibit enhanced growth and invasiveness. Our findings highlight the importance of mechanical cues in shaping the tissue-specific responses to oncogenic mutations, offering new insights into the events associated with early cancer development. By integrating computational modeling and experimental analyses, we propose that targeting the early mechanical interactions between transformed and wild-type cells could offer novel strategies for preventing tumor initiation.

“Nanoscale ligand spacing regulates mechanical force-induced cancer cell killing”

S Manasa Veena, Ph.D. Student

Cancer cells sense and respond to their extracellular environment, with differences in nanoscale ligand spacing affecting their drug resistance, migration and plasticity. Emerging reports show that stretch/ultrasound-mediated mechanical forces promote apoptosis (mechanoptosis) by increasing myosin contractility. Since myosin contractility is critical for nano-ligand spacing-regulated cell behavior, we study the effect of in vivo mimicking ligand spacing on cancer cell apoptosis upon ultrasound treatment. Gold nanoparticle arrays were created with 35, 50, and 70 nm spacings and functionalized with cyclic-RGD peptides. Interestingly, the highest level of apoptosis was observed on 50 and 70 nm spaced ligands, where an increase in myosin contractility, peripheral Piezo1 channel localization and enhanced Piezo1-mediated calcium influx were observed. Perturbing cell-matrix interactions through the compound Cilengitide (cyclic RGD pentapeptide) increases mechanoptosis on 35 nm ligand spacing to similar levels observed on 50 and 70 nm. Thus, nanoscale-level changes in binding domains regulate mechanoptosis through cell-matrix mediated mechanotransduction, and the synergistic action of ultrasound and nanomolar doses of Cilengitide can ultimately be applied to enhance tumor treatment.

Trailblazer Talks

“A novel photoacoustic contrast scaffold for noninvasive real time assessment of highly metabolic tumors”

Arjun S. V., Ph.D. Student

Noninvasive assessment of enhanced metabolic activity in tumors with imaging methods forms an essential part of clinical diagnosis, prognosis and therapy. Among the imaging methods widely used in the radiology clinic, the current gold standard is nuclear medicine or Positron Emission Tomography (PET) with ^{18}F -FDG an ^{18}F analog of 2-deoxy glucose. However, PET involves a radioactive isotope (^{18}F) and for precise anatomical image needs to be accompanied with another imaging modality-computed tomography (CT) that employs ionizing radiation (X-rays). Therefore, a drawback of PET/CT is the accumulation of radiation dosage that often limits its utility in monitoring therapy response at short intervals. Photoacoustic tomography, a hybrid of ultrasound and optical imaging has the potential to overcome the limitations of PET/CT with its high spatiotemporal resolution, its ability to provide both anatomical and functional information with the same modality, and most importantly non-involvement of any radiation or radioactivity. Therefore, with the right contrast agent, photoacoustic tomography can complement or even substitute PET imaging of metabolic tumors that are at a depth of $\sim 5\text{cm}$ from the surface of skin. In this study, we show our design and preclinical translation of such a photoacoustic contrast agent, in a metabolic tumor model of 4T1. Our contrast agent, GPc is a water soluble, small molecule that harbors four glucose moieties conjugated to an NIR-dye Zinc phthalocyanine. It demonstrated rich photoacoustic contrast and sensitivity, good cellular uptake in two aerobic cancer cell lines (OVCAR3 and 4T1), and excellent biochemical stability and minimal cytotoxicity at concentrations admissible for in vivo studies. In vivo serial photoacoustic imaging in a 4T1 tumor model, showed excellent uptake and high accumulation of GPc in tumor by 4 hours and washout of the tumor within 12 hours of intravenous (i.v) administration of the agent. Ex vivo studies further confirm that the agent is predominantly cleared through the hepatobiliary route. Overall, this probe offers to be a promising alternative for the assessment of therapy response in a longitudinal manner.

“Unified mechanistic model of glycemic and body weight regulation for optimizing incretin-based therapies”

Shivaani E., M.Tech. Student

Obesity and type 2 diabetes (T2D) are major global health challenges, affecting millions worldwide. Incretin-based therapies, leveraging hormones like glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic polypeptide (GIP), have shown significant potential in improving glycemic control and promoting weight loss. These hormones enhance insulin secretion, suppress glucagon, slow gastric emptying, and reduce appetite, making them key targets for therapeutic intervention. Drugs such as GLP-1 receptor agonists (e.g., semaglutide) and dual GLP-1/GIP receptor agonists (e.g., tirzepatide) have delivered promising clinical outcomes, with emerging therapies like the dual GLP-1/amylin receptor agonist cagrisema, the triple GLP-1/GIP/glucagon agonist retatrutide, and oral semaglutide further expanding the therapeutic landscape.

Here, we develop an integrated mechanistic model to elucidate the dynamic interactions between insulin, glucagon, and incretin hormones, their role in glucose homeostasis, and their contributions to weight regulation. The model predicts key therapeutic outcomes, such as blood glucose levels and weight loss, and helps optimize dosing regimens to maximize efficacy while minimizing side effects. Additionally, it provides insights into the synergistic effects of combination therapies, offering a framework to design more effective treatment strategies for obesity and T2D.

Poster Sessions

Poster Number	Presenter Name	Title of the Poster
1	Saivishak Saikumar	In-situ hydrogel-based system for Sprifermin delivery for Osteoarthritis therapy
2	Sibani Jani	Engineering a 3D tissue model to investigate the role of aging in lung fibrosis
3	Roshni P	Metabolic profiling of tumors with 3T magnetic resonance spectroscopy (MRS)
4	Alapati Mohan Sai Surya Teja	Automation of quantification in Uniculture plate for Rapid Diagnosis of Urinary Tract Infection
5	Akhilesh Agarwal	Multifunctional Bioglass Particles for Bone Regeneration and Immunomodulation
6	Bidita Samanta	Variability in the impact of dicarbonyl stressors on intravasation-relevant extracellular matrices
7	Vibhor Gaikwad	Role of receptor Guanylyl Cyclase C (GC-C) in intestinal immunity
8	Prem Singh Anant	Immunomodulatory GelMA hydrogels: balancing LPS-Induced inflammation for enhanced wound repair

Poster Sessions

Poster Number	Presenter Name	Title of the Poster
9	Chandan Sringi	Role of Idiopathic pulmonary fibrosis in initiating lung cancer in 3D ALI (air-liquid interface) cultures of lung cells
10	Partha Sarthi Dey	Multiscale modeling of hypoxic tumor microenvironment
11	Saksham Jain	Biomaterial-based pulmonary delivery of mycobacteriophages for prophylaxis against Tuberculosis
12	Sejal Khanna	Understanding the 3D epigenomic landscape of acquired drug resistance in estrogen receptor-positive (ER+) breast cancer
13	Akanksha Sarothia	Quantitative characterization of Neural Cell Migration
14	Shounak Sanyal	Investigating the role of resource limitation in the spatial distribution of F-actin species during the Immunological synapse
15	Vijaya Vaishnavi	Three-dimensional (3D) Hydrogel Culture Systems for Studying Host-Pathogen Interaction in Tuberculosis
16	Syed Tameemuddin	Noninvasive Assessment of Hemodynamic Changes in Ageing vs. Young Rodent Brain with Photoacoustic Imaging Tomography