GLOBAL IMMUNOLOGY SUMMIT-2026

Unravelling Immunity: From Molecular Signals to Systemic Functions

19th – 21st January 2026 | BRIC-ILS, Bhubaneswar, India



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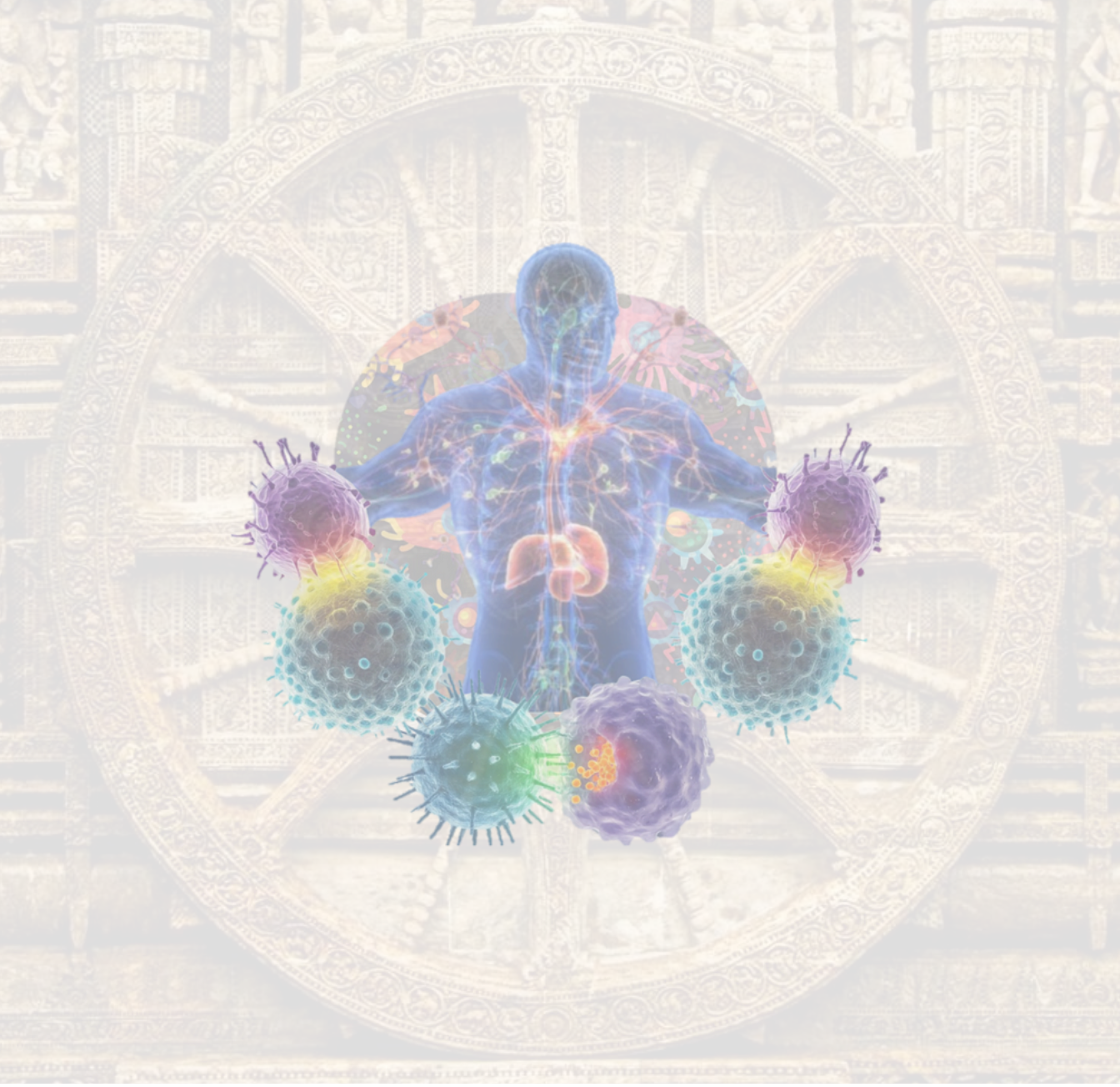
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“The cover highlights a mesmerizing fusion of science and heritage, capturing the essence of the Global Immunology Summit 2026. Inspired by the majestic Konark Sun Temple of Odisha, it symbolizes the eternal cycle of balance and coordination that defines the immune system. At the center, the human body surrounded by immune cells represents the intricate coordination among cellular players that sustains our immune system. The design places this system within the Sun Temple’s iconic wheel, symbolizing the continuous cycle of balance between molecular signals and systemic functions. The depiction of cellular interactions reflects the depth and complexity of immunity - inviting the global scientific community to explore, unravel, and illuminate the mysteries of immune regulation through this prestigious summit.”
(Scientific illustration by *Mr. Sourya Prakash Nayak*, BRIC-ILS)

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DIRECTOR'S MESSAGE



Dr. Debasis Dash
Director, BRIC-Institute of Life Sciences
Email: director@ils.res.in

It gives me immense pleasure to warmly welcome all our distinguished speakers and participants to the Global Immunology Summit – 2026 (GIS-2026) at the BRIC–Institute of Life Sciences, Bhubaneswar.

At ILS, we strive to deepen our understanding of biological systems and translate this knowledge into meaningful advances in human health. Our research spans immunology, host–pathogen interactions, genomics, molecular medicine, and systems biology, with a strong emphasis on integrating fundamental discovery with translational impact. GIS-2026 aligns closely with this mission by bringing together cutting-edge research across innate and adaptive immunity, inflammation, infectious diseases, immunogenomics, and immunotherapy.

This abstract book reflects not only technical excellence, but also the spirit of curiosity, collaboration, and interdisciplinary thinking that drives scientific progress. Whether exploring molecular signalling pathways, immune regulation, or therapeutic innovations, the work presented here advances our collective understanding of immune function in health and disease.

I encourage participants to look beyond individual studies, identify connections across disciplines, and explore opportunities for collaboration. Meetings such as these strengthen our scientific community and inspire the next generation of ideas and innovation.

I thank the organizers and participants for their dedication and wish you a stimulating and productive meeting.

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Organizing Committee

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Director, BRIC-ILS



Dr. Ganesan Karthikeyan
Director, BRIC-THSTI



Prof. Srinivas Patnaik
Dean, School of Biotechnology, KIIT-DU

Convenor & Co-Convenor:



Dr. Sunil K. Raghav
BRIC-ILS



Dr. Bhawna Gupta
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Sumanta Kumar Kolay
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Sridhar Behera
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Braja Mohan Behera
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Safet Pradhan
Lab Technician

PROGRAM SCHEDULE

Day 1: 19th January 2026, Monday

Time	Schedule
08:00 - 09:00	Registration and Welcome
Inaugural Session	
09:00 - 09:30	• Welcome Address **Dr. Debasis Dash,** Director, BRIC-ILS, BBSR **Dr. Srinivas Patnaik,** Dean,School of Biotechnology, KIIT-DU, BBSR • Inaugural Address **Dr. Sanghamitra Pati,** Director, ICMR-RMRC, BBSR • GIS Journey **Dr. Amit Awasthi,** BRIC-THSTI, Faridabad • **Dr. Sunil K. Raghav,** Convenor • Vote of Thanks **Dr. Bhawna Gupta,** KIIT-DU
SESSION 1 **Theme: Autoimmune Disorders & Inflammation** **CHAIR: Prof. B. Ravindran & Prof. N. K. Mehra**	
Keynote Talk 09:30 – 10:05 (30+5 Mins)	**Dr. Vijay Kumar Kuchroo,** Harvard, USA **Title: Dark Kinases as Checkpoint molecules that regulate induction of anti-tumor immunity**
10:05 – 10:25 (15+5 Mins)	**Dr. Maheswara Reddy Emani,** GenenTech, USA **Title: Predictive in vitro profiling of LNP-induced innate immune response using an iPSC-derived monocyte model**
10:25 – 10:45	**Dr. Dipayan Rudra,** ShanghaiTech University, China **Title: Novel Insights on Regulatory T cell Mediated Tumor Immune Tolerance**
10:45 – 11:05	**Dr. Pragyan Acharya,** AIIMS, Delhi **Title: Lipid–Immune Crosstalk as a Driver of Dysregulated Inflammation in Acute-on-Chronic Liver Failure**
11:05 – 11:10	Q/A for Students **(Dr. Avinash Bajaj, Prof. Avinash Sonawane)**

11:10 – 11:25	**Tea Break & Networking, Ground floor, Front Lawn of Auditorium Building (Poster Set-up) & G+5 basement**
SESSION 2 **Theme: Autoimmune Disorders & Inflammation** **CHAIR: Prof. J. Dandapat & Prof. G.C. Kundu**	
11:25 – 11:45	**Prof. Lionel Apetoh,** Indiana University, USA **Title: Molecular regulation of T_H9 cell differentiation and anticancer properties**
11:45 – 12:05	**Prof. Rupesh K. Srivastava,** AIIMS Delhi **Title: Harnessing the "Gut-Immune-Bone axis" as a novel Immunoporotic strategy in enhancing Bone-Health in Post-Menopausal Osteoporosis**
12:05 – 12:25	**Dr. Amit Awasthi,** THSTI, Delhi **Title: Ionic immune regulation limits intestinal inflammation by promoting cholic acid-mediated IL-10 induction**
12:25 – 12:45	**Robert Balderas,** BD Biosciences **Title: A proteomic and transcriptomic assessment of innate and adaptive responses from normal human PBMCs**
12:45 - 12:55 (8+2 mins)	**Delegate Faculty Talk: Dr. Zaigham Abbas Rizvi,** THSTI, Faridabad **Title: Long-Term Systemic Sequelae of SARS-CoV-2: A Multi-Omics Analysis of Neuroinvasion and Extrapulmonary Pathology in Longitudinal Animal Models**
12:55 – 13:05	Q/A for Students **(Dr. Avinash Bajaj, Dr. Pragyan Acharya)**
13:05 – 14:00	**Lunch Break & Poster discussions, ILS Central Lawn & G+5 building ground floor**
SESSION 3 **Theme: Infectious Diseases & Host Immunity** **CHAIR: Dr. Narottam Acharya & Dr. Satish Devdas**	
Keynote talk (30+5 Mins): 14:00 – 14:35	**Prof. David A Hafler,** Yale University, USA **Title: Host Genetic Architecture between Epstein–Barr Virus Activity and Multiple Sclerosis Reveals Shared Pathways**

14:35 – 14:55	**Dr. Imroze Khan,** Ashoka University, Delhi **Title: Viewing Immunity in the context of Ageing and Evolution**
14:55 – 15:15	**Dr. Hiyaa Ghosh,** NCBS, Bangalore **Title: Microglial diversity: Do specific subtypes respond to specialized need?**
15:15 – 15:35	**Prof. Avinash Sonawane,** IIT Indore **Title: TRPV4 ion channel exerts differential immunoprotective response in host cells during early and late infection phase of *Mycobacterium tuberculosis* infection**
15:35 – 15:55	**Dr. Subhasis Chattopadhyay,** NISER, BBSR **Title: Cell Mediated Immune Regulation during Chikungunya Virus (CHIKV) Infection, Experimental Immune Suppression and Cancer Immunity**
15:55 – 16:00	Q/A for Students **(Prof. Himanshu Kumar, Dr. Gaurav Sharma)**
16:00 – 16:15	**Tea Break & Networking, Ground floor & Front Lawn of Auditorium Building**
16:15 - 16:45	**Poster session 1 & Assessment (47 Posters), G+5 building ground floor**
SESSION 4 Theme: Infectious Diseases & Host Immunity CHAIR: Dr. Sunil K. Raghav & Dr. Debasis Dash	
Keynote talk (30+5 Mins): 16:45 – 17:20	**Dr. Riitta Lahesmaa,** Turku Bioscience Centre, University of Turku, Finland **Title: Regulation of human T cell response**
17:20 - 17:40	**Dr. Prafullakumar Tailor,** NII, Delhi **Title: Decoding the specificity within Interferon regulatory factors in dendritic cell differentiation and innate immunity**

17:40 - 18:00	**Dr. Rajiv Kumar,** BHU, Varanasi **Title: Characterization of Cytotoxic CD4+ T Cells in Visceral Leishmaniasis Patients**
18:00 - 18:20	**Dr. Santosh Chauhan,** CCMB, Hyderabad **Title: Mechanism of innate immune responses**
18:20 - 18:40	**Dr. Gaurav Sharma,** PGIMER, Chandigarh **Title: Diversity of Killer-cell Immunoglobulin like Receptors Influence Graft vs Host disease after Hematopoietic Stem Cell Transplantation**
18:40 - 18:45	Q/A for Students **(Prof. Himanshu Kumar, Dr. Rupesh K. Srivastava)**
18:45 - 18:52	**Sponsor Talk: SPT Labtech**
18:52 - 19:00	**Sponsor Talk: Vision Group**
19:00 onwards	**Cultural Program & Dinner, ILS Central Lawn**

Day 2: 20th January 2026, Tuesday

Time	Schedule
SESSION 1 **Theme: Immunogenomics & Immunometabolism** **CHAIR: Dr. Dipayan Rudra & Dr. Devinder Sehgal**	
Keynote talk (30+5 Mins): 09:00-09:35	**Dr. Bart Everts,** Leiden University Medical Center, Netherlands **Title: Regulating the regulators: how nutrient sensors control dendritic cell-driven tolerance**
09:35 - 09:55	**Dr. Nimesh Gupta,** NII, Delhi **Title: Gut Metabolite Shape Antibody Quality By Impairing Tfh-B Cell Collaboration**
09:55 - 10:15	**Dr. Ranjan Nanda,** ICGEB, Delhi **Title: Tango of Host Amino Acid Metabolism and *Mycobacterium tuberculosis* Infection**
10:15 -10:35	**Prof. Kushagra Bansal,** JNCASR, Bangalore **Title: Regulators of genome superstructure in the immune system**
10:35 – 10:55	**Prof. Himanshu Kumar,** IISER Bhopal **Title: Immunometabolic role of PANK4 in regulation of TLR7/9-mediated innate immunity**
10:55 – 11:15	**Dr. Avinash Bajaj,** RCB, Faridabad **Title: Metabolite-mediated Regulation of Immunoproteasome Steers the Chemotherapy Response in Hot and Cold Tumour Microenvironment**
11:15- 11:35	**Dr. Sunil K. Raghav,** ILS, Bhubaneswar **Title: Nuclear Receptor co-repressors NCOR1 & SMRT fine-tune Immune-tolerance and inflammation**
11:35 - 11:40	Q/A for Students **(Dr. Savneet Kaur, Dr. Rajiv Kumar)**

11:40 - 12:00	**Tea Break & Networking, Ground floor & Front Lawn of Auditorium Building**
SESSION 2 **Theme: Clinical and Translational Immunology** **CHAIR: Prof. Hans-Acha Orbea & Dr. Sunil K. Raghav**	
Keynote talk (30+5 Mins): 12:00: 12:35	**Prof. Bart Deplancke,** EPFL, Lausanne **Online Talk: Bridging Morphology and Transcriptomics in Immunology through Single-Cell Phenomics**
12:35 – 12:55	**Dr. Savneet Kaur,** ILBS, Delhi **Title: Disruption of Mesenteric Lymph Node Immune Surveillance Drives Bacterial Infections in Advanced Liver Disease**
12:55 - 13:15	**Dr. Chandrashekara. S,** ChanRe Centre, Bangalore **Title: Disruption of Mesenteric Lymph Node Immune Surveillance Drives Bacterial Infections in Advanced Liver Disease**
13:15 - 13:25	**Delegate Faculty Talk: Dr. Vandana D. Pradhan,** NIIH, Mumbai **Title: IFN levels and IFN signature identify patient subsets with distinct clinical and immunological characteristics in SLE**
13:25 - 13:30	Q/A for Students **(Dr. Punit Prasad, Prof. Kushagra Bansal)**
13:30-14:30	**Lunch Break and Poster discussions, ILS Central Lawn & Ground floor of G+5 building**
SESSION 3 **Theme: Clinical and Translational Immunology** **CHAIR: Dr. Sunil K. Arora & Prof. Gaurishankar Sa**	
Keynote Talk (30+5 Mins): 14:30 – 15:05	**Prof. Wilfried Ellmeier,** Medical University of Vienna **Title: Online talk: Histone deacetylases and the control of $CD4^+$ T cell-mediated immunity**

15:05 – 15:25	**Dr. Girdhari Lal,** NCCS, Pune **Title: NK-1R signaling restrains Foxp3^{+} Treg differentiation and function during gut inflammation and autoimmunity**
15:25 – 15:45	**Dr. Brian Hafler,** Yale School of Medicine, USA **Title: Single-cell analysis identifies shared inflammatory glial states across neurodegenerative central nervous system diseases**
15:45 - 16:05	**Dr. Devinder Sehgal,** NII, Delhi **Title: SPD_1629 Mediates Uracil Uptake and Is Required for Streptococcus pneumoniae Fitness and Virulence In Vivo**
16:05 – 16:10	Q/A for Students **(Dr. Ranjan Nanda, Dr. Nimesh Gupta)**
16:10 – 16:25	**Tea Break, Ground floor & Front Lawn of Auditorium Building**
16:25 - 17:20	**Poster session 2 & Assessment (47 Posters), G+5 building ground floor**
17:20 - 17:40	**Dr. Sumit Kumar Hira,** University of Burdwan, West Bengal **Title: Breaking the Metabolic Lock: Sphingolipid-Driven T Cell Exhaustion in Immunotherapy-Resistant Colorectal Cancer**
17:40 – 18:00	**Dr. Sunil K. Arora,** PGIMER, Chandigarh **Title: Checkpoint molecules an evasion mechanism in HIV-Mtb co-infection**
18:00 – 18:20	**Prof. Mahaboobkhan Rasool,** VIT, Vellore **Title: IL-17A signalling blockade: A powerful therapeutic modality to combat rheumatoid arthritis and psoriasis**
18:20 - 18:30	**Delegate Faculty Talk: Dr. Ravi Verma,** JNU, Delhi **Title: Unravelling Mucosal Immunity–Microbiota Interactions: From Host Defence to Beneficial Symbiosis**

18:30 - 18:40	**Delegate Faculty Talk: Dr. Deepak Kumar Rathore,** THSTI, Faridabad **Title: Immune Protection After Quadrivalent Influenza Vaccination Among Elderly Indians**
18:40 – 18:47	**Sponsor Talk: Premas Life Sciences**
18:47 - 18:55	**Sponsor Talk: Bencos Life Sciences**
18:55 onwards	**Bus transportation to Padmaja Hotel**
19:00 onwards	**Gala Dinner @ Padmaja Hotel Rooftop** **Padmaja Hotel, Niladri Vihar**

Day 3: 21st January 2026, Wednesday

Time	Schedule
SESSION 1 **Theme: Immunotherapy & Technical advancements** **CHAIR: Dr. Satish Devdas & Dr. Shantibhusan Senapati**	
Keynote talk (30+5 Mins): 09:00 – 09:35	**Prof. Gaurisankar Sa,** Bose Institute, Kolkata **Title: Cancer immunotherapy and beyond**
09:35 – 09:55	**Prof. Dipankar Nandi,** IISc Bangalore **Title: Interferon-gamma-induced nitric oxide responses: Yes to NO!**
09:55 – 10:15	**Prof. Atul Johri,** JNU, Delhi **Title: Use of reverse vaccinology approach for the development of vaccine against *Streptococcus pyogenes* infections**
10:15 – 10:35	**Dr. Syed Faisal,** NIAB, Hyderabad **Title: Bridging Innate and Adaptive Immunity: Next-Generation Adjuvants for Veterinary Vaccines**
10:35 - 10:45	**Delegate Faculty Talk: Dr. Pijus Barman,** Mahindra University, Hyderabad **Title: Impact of aging on monocyte function and breast cancer progression**
10:45 - 10:50	Q/A for Students **(Dr. Sumit Kumar Hira, Dr. Punit Prasad)**
Student Talk **Judges: Prof. Atul Johri, Prof. Dipankar Nandi, Prof. GauriSankar Sa, Dr. Satish Devdas**	
10:50 – 11:00	**Student Talk 1: Sarwareddy Kartik Kumar,** AHERF, Hyderabad **Title: Harnessing Tumor-Derived Extracellular Vesicle-Primed Dendritic Cell Exosomes for both Immunogenic and Cytotoxic Targeting of Oral Squamous Cell Carcinoma**

11:00 – 11:10	**Student Talk 2: Amlan Priyadarshee Mohapatra,** ILS, Bhubaneswar **Title: The heterogeneity in normal pancreatic fibroblasts and its implication in CAFs mediated tumor immune-modulation and pancreatic cancer progression**
11:10 – 11:20	**Student Talk 3: Mohammad Kadivella,** NIAB, Hyderabad **Title: From Structure to Immunity: Characterizing Leptospira FlaB as a Potent Vaccine Adjuvant**
11:20 – 11:30	**Student talk 4: Subhasish Prusty,** ILS, BBSR **Title: Hsp16.3ΔC4, a C-terminal truncated mutant, confers strong cellular and humoral immunity against pulmonary tuberculosis**
11:30 – 11:40	**Student talk 5: Parnika Sahoo,** IISER Tiruvanantpuram **Title: Co-option of the host ACVR1-iron axis drives iron remodelling and enables a permissive niche for *Toxoplasma gondii* replication**
11:40 – 11:50	**Student talk 6: Pranati Sar,** Silver Oak Inst. of Science, Ahmedabad, Gujarat **Title: Host Genetic and Epigenetic Regulation of MHC-II as a Predictor of Malaria Clinical Outcomes**
11:50 - 12:00	**Student talk 7: Agalya Vanamudhu,** ICMR-NIRT, Chennai **Title: Profiling Complement Proteins in Pediatric Cases of Tuberculosis and SARS-CoV-2 Seropositivity**
12:00 - 12:10	**Student talk 8: Anand Kumar Keshri,** IIT-Mandi, HP **Title: Hyperactivated CCR5⁺ Neutrophils Drive Gastric Inflammation and Epithelial Injury in Helicobacter pylori Infection**
12:10 – 12:17	**Sponsor Talk: Beckman Coulter**
12:17 – 13:00	**Valedictory function and Award Ceremony** **Travel Awards (8), Poster Awards (10), Oral Presentation Young Scientist Awards (3)**
13:00 – 14:00	**Lunch, ILS Central Lawn**
14:00 Onwards	**Buses to Konark & Puri for interested Delegates / & Invited speakers** **Outside ILS (Buses will leave at 14:15h)**

INVITED SPEAKER TALK ABSTRACTS

Dr. Vijay K. Kuchroo
Harvard Medical School, USA
Email: vkuchroo@evergrande.hms.harvard.edu

Dark Kinases as Checkpoint molecules that regulate induction of anti-tumor immunity

Expression of co-inhibitory receptors or "checkpoint" molecules, such as CTLA-4, PD-1, TIM-3, LAG-3 and TIGIT on effector T cells is a key mechanism for ensuring immune homeostasis. Dysregulated expression of co-inhibitory receptors on CD4+ T cells promotes autoimmunity while sustained overexpression on CD8+ T cells promotes T cell dysfunction or exhaustion, leading to impaired ability to clear chronic viral infections and cancer. Immune checkpoint blockade (ICB) treatment by blocking CTLA4 and PD-1 has revolutionized cancer therapies, yet current ICB response rates are still relatively low. This suggests the need to discover novel checkpoint molecules that can be targeted and synergize with current checkpoint therapies to promote anti-tumor immunity.

More than 50% of the kinase genome is comprised of dark kinases whose function and targets are completely unknown, and their function in the immune system is completely unknown. Using novel genetic and pharmacological tools we have identified a critical role for a dark kinase PI5P4Kγ (Pip4k2c) in regulating anti-tumour immunity. Specific deletion of Pip4k2c in dendritic cells (DCs) resulted in a significant reduction in tumour burden, concomitant with an increased frequency of intratumoral DCs and expansion of highly functional CD4+ and CD8+ effector T cells. To explore the therapeutic potential of targeting PI5P4Kγ, we generated a highly selective small molecule degrader and demonstrate that degradation of endogenous PI5P4Kγ alone delays tumour growth and enhances anti-tumour immunity. Together our results show that DC-specific loss of a dark kinase Pip4k2c profoundly increases anti-tumour immunity, and that targeting endogenous PI5P4Kγ may be a promising strategy to increase anti-tumour immune responses, when used with existing checkpoint molecules.

Dr. Emani Maheswara Reddy
Genentech, USA
Email: emani.maheswara-reddy@gene.com

Predictive in vitro profiling of LNP-induced innate immune responses using an iPSC-derived monocyte model.

Lipid nanoparticles (LNPs) are a powerful drug delivery platform advancing vaccines and gene therapies. However, their efficacy and safety are closely linked to innate immune activation, and a reliable in vitro model to predict immune responses across diverse LNP compositions remains lacking. Conventional models, such as PBMCs, are limited by donor variability and inconsistent sensitivity. To address this, we developed a cytokine profiling platform using induced pluripotent stem cell (iPSC)-derived monocytes (iMonocytes), a physiologically relevant cell type that gives rise to macrophages and dendritic cells and plays a key role in immune surveillance and inflammation. iPSCs provide a renewable, uniform monocyte source for consistent, high-sensitivity LNP screening. When tested with LNPs of graded immunostimulatory potency, iMonocytes showed improved reproducibility and strong correlation with in vivo cytokine responses. This platform enables evaluation of cargo- and dose-dependent effects, providing a robust and scalable tool for preclinical assessment and rational design of LNP therapeutics.

Dr. Dipayan Rudra
ShanghaiTech University, China
Email: drudra@shanghaitech.edu.cn

Novel Insights on Regulatory T cell Mediated Tumor Immune Tolerance

Evolution of the immune system is a key to efficient defense of the host against pathogens. At the same time unwanted immune response against self, needs to be tightly regulated to avoid catastrophic autoimmunity. While self-reactive thymocytes could be eliminated through the cell intrinsic mechanism of central tolerance, an additional mechanism of immune regulation by the so-called Regulatory T (Treg) cells has become a major focus of immune research for the last two decades. Treg cells, which are dependent on the fork-head family transcription factor Foxp3 for their generation and function, are a specific subtype of CD4+ lymphocytes critical for suppressing autoimmunity and uncontrolled immune response against pathogens. Contrary to beneficial roles against inflammation, due to their inherent immune-regulatory properties, Tregs are obstructive to anti-tumor immune responses, and are therefore well-recognized targets with enormous therapeutic promise in cancer immunotherapy. An important conceptual bottleneck to therapeutically targeting Treg cells is the fact that systemic inhibition of Treg function in order to boost anti-tumor response may cause life-threatening autoimmunity. Therefore, tumor-specific mechanisms of Treg mediated immune tolerance needs to be identified. We recently discovered that alternative glucose utilization pathway through the Glucose transporter Glut3, which is primarily a transporter expressed in neurons, is crucial. Interestingly, Glut3-mediated glucose uptake results in optimal utilization of glucose towards the so called Hexosamine Biosynthetic Pathway, and post translational O-GlcNAcylation of downstream factors, that orchestrates the homeostasis and function of Treg cells specifically at tumor sites. The relevance of these findings in light of tumor specific functionality of Treg cells and their implications in the context of human cancer types will be discussed.

Dr. Pragyan Acharya
AIIMS Delhi
Email: pragyan.acharya@aiims.edu

Lipid–Immune Crosstalk as a Driver of Dysregulated Inflammation in Acute-on-Chronic Liver Failure

Acute-on-chronic liver failure (ACLF) is characterized by profound systemic inflammation, immune dysfunction, and multiorgan injury, yet the contribution of circulating metabolic mediators to immune reprogramming remains incompletely understood. We investigated whether plasma lipid alterations in ACLF exert direct immunomodulatory effects across blood and tissue compartments. Targeted plasma lipidomic profiling performed across two temporally independent ACLF cohorts revealed consistent depletion of polyunsaturated lysophospholipids and enrichment of monounsaturated phospholipids and triacylglycerols, indicating a reproducible disease-associated lipid milieu. To assess functional relevance, healthy donor leukocytes were exposed ex vivo to ACLF-derived plasma lipids, resulting in suppression of multiple Toll-like receptors and induction of the neutrophil activation marker CD177, thereby recapitulating ACLF-like immune signatures. Lipid uptake by leukocytes was confirmed by targeted mass spectrometry, demonstrating selective enrichment of triacylglycerol species linked to disease severity. Proteomic profiling of circulating neutrophils and post-mortem liver and kidney tissue from ACLF patients identified a shared neutrophil degranulation program across compartments, suggesting a systemic immune effector state rather than organ-restricted inflammation. Integrative transcriptional and proteomic analyses highlighted PPARγ as a central regulatory node, and in silico docking demonstrated high-affinity binding of ACLF-associated lipid species to PPARγ, comparable to known ligands. Together, these findings indicate that circulating lipids in ACLF are not passive metabolic bystanders but active drivers of innate immune reprogramming and neutrophil effector function. Lipid-mediated modulation of TLR signaling, neutrophil degranulation, and PPARγ-centered transcriptional pathways may represent a mechanistic link between metabolic dysregulation and immune dysfunction in ACLF, offering potential avenues for therapeutic targeting.

Dr. Lionel Apetoh
Indiana University School of Medicine, USA
Email: lapetoh@iu.edu

Molecular regulation of T_H9 cell differentiation and anticancer properties

We and others previously reported that T_H9 cells, a novel subset CD4 T cells secreting IL-9 and IL-21, featured a superior ability over all other T cell subsets to prevent melanoma progression upon adoptive transfer. This is due to their ability to eliminate antigen-loss variants, self-renew, and activate other immune effector cells in vivo. Previous studies from our group and others also revealed that the phosphorylation of key intracellular mediators such as STAT1, STAT5, NF-kB, and the kinases Fyn, Itk, TAK1 and mTOR promote T_H9 cell differentiation. The molecular mechanisms negatively controlling T_H9 cell differentiation and anticancer activity remain however incompletely understood. We will discuss how phosphorylation events control T_H9 cell effector functions and anticancer activity.

Dr. Rupesh K. Srivastava
AIIMS Delhi
Email: rupeshk@aiims.edun

Harnessing the "Gut-Immune-Bone axis" as a novel Immunoporotic strategy in enhancing Bone-Health in Post-Menopausal Osteoporosis

Our group has coined the term "Immunoporosis" to highlight the immune system's role in osteoporosis and has previously shown the involvement of Bregs in postmenopausal osteoporosis (PMO). While dysbiosis has been implicated in PMO, it remains unclear whether the dysbiotic gut microbiota (GM) directly contributes to Breg dysregulation. Therefore, in this study, we determined the role of dysbiosis in Breg dysfunction and explored whether restoring GM balance under estrogen-deficient conditions can mitigate inflammatory bone loss by modulating the immunoporotic function of Bregs. Our results indicated that GM plays a pivotal role in the onset and progression of PMO by modulating the Bregs. Both clinical and preclinical PMO conditions demonstrated an increase in endotoxin-producing bacteria and a decrease in SCFA-producing bacteria, contributing to inflammatory bone loss. Notably, BC supplementation significantly improved bone mineral density, strength, and microarchitecture by enhancing the anti-osteoclastogenic and immunomodulatory functions of Bregs. Bregs from Ovx mice exhibited impaired capacity to suppress osteoclastogenesis and reduced ability to induce Tregs while failing to inhibit Th17 differentiation. In contrast, Bregs from Ovx + BC mice showed restored function, effectively inhibiting osteoclastogenesis, promoting Treg differentiation, and suppressing Th17 responses. HPLC analysis revealed significantly reduced SCFA levels in Ovx mice, whereas BC treatment elevated SCFA production, underscoring their role in maintaining bone health. in vitro assays further confirmed that SCFAs suppress osteoclastogenesis in a dose-dependent manner and promote the differentiation of both Bregs and Tregs while inhibiting Th17 cell differentiation.Altogether, the present study delves deeper into the role of immune homeostasis and GM profiling in the pathophysiology of PMO. Altogether, the findings of the present study open novel therapeutic avenues, suggesting restoration of GM composition as one of the viable therapeutic options in mitigating inflammatory bone loss under PMO conditions via modulating the Bregs

Dr. Robert Balderas
BD Bioscience
Email: robert_balderas@bd.com

A proteomic and transcriptomic assessment of innate and adaptive responses from normal human PBMCs

Assessing T cell responses to both adaptive and innate triggers is crucial for understanding immune activation and regulation. Utilizing anti-CD3/CD28 beads to stimulate T cells provides a controlled invitro approach to analyze the adaptive immune response. These beads mimic antigen-presenting cells, engaging T cell receptors and co-stimulatory pathways, thus leading to robust T cell proliferation/activation. Using this approach allows one to quantify the strength and characteristics of the T cell response to specific antigens.

In contrast to anti-CD3/28, assessing bystander T cell responses through an innate trigger, such as poly I:C, highlights the role of innate immunity in modulating T cell activity. Poly I:C (a TLR3 agonist) mimics viral infections, stimulating specific receptors that enhance the immune response even in non-specific T cells. By studying T cells from the 2 individual donors under both adaptive and innate conditions, we show how prior T cell activation might influence bystander effects, potentially revealing mechanisms of immune memory and cross-reactivity. In this study we will assess both response utilizing and integrated approach of flow cytometry and single cell WTA and deep receptor profiling with CITEseq.

Together, these measurements provide valuable insights into T cell dynamics and plasticity of response, informing vaccine development and therapeutic strategies against infections and cancers.

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Host Genetic Architecture between Epstein–Barr Virus Activity and 2 Multiple Sclerosis Reveals Shared Pathways

Epstein–Barr virus (EBV) is strongly implicated as an essential environmental trigger of multiple sclerosis (MS), yet the host genetic mechanisms governing EBV activity and how infection triggers the disease are not known. We developed a pipeline to quantify EBV DNA from whole-genome sequencing data and applied it to population-scale cohorts. Using this pipeline, we performed a cross-ancestry genome-wide association study (GWAS) of EBV DNA positivity in 617,186 individuals and identified 39 independent susceptibility risk loci, with the strongest associations in the HLA region. We validated this finding in our independent cohort (N=104) and found that qPCR-confirmed EBV DNA positive individuals were markedly enriched in the top decile of polygenic risk score containing newly discovered loci. There was a significant overlap with genetic variants associated with MS risk and severity including MS susceptibility alleles, HLA-DRB104:04 and HLA-A02:01. Polygenic risk score and Mendelian randomization analyses further supported a causal influence of EBV activity on MS, largely driven by HLA variation, with possible relevance to other autoimmune diseases. By establishing an EBV-optimized single-cell RNA-seq method, we identified EBV-infected B cells, primarily in memory B cells and plasma cells, from MS and healthy individuals. Notably, EBV-infected memory B cells showed strong upregulation of risk genes from both the EBV and MS GWAS, suggesting that MS susceptibility genes are preferentially activated within EBV-infected B cells. Together, these findings define a genetic and cellular framework linking EBV infection to MS initiation.

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Viewing Immunity in the context of Ageing and Evolution

Evolutionary theory predicts that the strength of natural selection wanes in late life and becomes negligible beyond the reproductive window. During this period, harmful mutations can accumulate, accelerating ageing, characterised by an overactive immune system that causes immunopathology, along with higher morbidity and death rates. The question is: why doesn't natural selection eliminate these harmful age-specific maladaptive traits of immunity? What drives the evolution of age-specific variation in immunity and infection outcomes? To answer these questions, we used experimental evolution lines of Drosophila melanogaster populations with divergent reproductive schedules and ageing rates (e.g., 'early reproduction-faster ageing' vs. 'late reproduction-slower ageing') to explore whether features of immune ageing coevolve with ageing rate and reproductive schedule. For example, does immune senescence appear earlier in early-reproducing and early-ageing populations? While our results support this prediction, we also observed that individuals experienced better post-infection health near their reproductive peak, which then declined steadily thereafter. In a subsequent transcriptomics study, we detected both qualitative and quantitative declines in immune efficacy beyond reproductive ages. While maintaining optimal immune regulation is critical for improved health after infection, immune responses often become less specific, exemplified by increased Toll-responsive antimicrobial peptides targeting Gram-negative bacteria. At the same time, there is a decline in immune effectors, such as IMD-regulated AMPs, which previously provided specific protection against pathogens at younger ages. This shift may contribute to higher mortality following infection. Overall, our findings offer a rare conceptual framework for understanding the evolutionary basis of the decline in immunity and post-infection health with ageing, opening avenues for more detailed molecular studies of the regulatory networks involved.

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Microglial diversity: do specific subtypes respond to specialized need?

Microglia diversity is well appreciated in the field, especially with revelations from single cell transcriptomics studies. However, the functional relevance of this diversity remains unclear. Also, whether the varied transcriptional profiles merely present a transient cellular 'state' or if these are specialized 'subtypes' with specific functions remain unclear. We investigated this question using a controlled brain injury model and investigated microglial manifestations during the acute response and recovery phase. Our data indicate that microglia in the normal healthy brain actually have distinct subtypes with potentially specialized functions. I will present our unpublished data regarding microglial response during acute and recovery phases of brain injury, thereby eluding some insights into the possible functional relevance of microglial diversity.

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TRPV4 ion channel exerts differential immunoprotective response in host cells during early and late infection phase of Mycobacterium tuberculosis infection

*Mycobacterium tuberculosi*s (Mtb) proliferates within myeloid cells by subverting host immune responses as a prerequisite for progression to active tuberculosis disease. The non-selective calcium (Ca^{2+}) channel TRPV4, expressed in innate immune cells, plays an important role in the regulation of host immune responses and has emerged as a potential target of Mtb-mediated immune evasion. Our previous work showed that Mtb modulates TRPV4 expression and subcellular localization to avoid killing by macrophages during early infection phase. Trpv4-knock-out mice showed transiently higher mycobacterial burden and reduced proinflammatory responses. However, Trpv4-deficient mice were found to be superior to wild-type ones in controlling Mtb infection in the chronic infection phase. We also found TRPV4-positive neutrophils in close proximity to human TB granulomas, suggesting a role for TRPV4 in TB pathogenesis. Here, we investigated the effect of pharmacological inhibition of TRPV4 on the progression of disease as well as in the regulation of neutrophil function during Mtb infection in TB mice model. Mtb infection down-regulated TRPV4 expression in human neutrophils and macrophages resulting in decreased intracellular Ca^{2+} levels. TRPV4 inhibition in neutrophil-macrophage coculture cells increased reactive oxygen species production that enhanced necrotic cell death to favour intracellular Mtb survival. Mice treated with TRPV4 agonist displayed reduced pulmonary Mtb burden compared to antagonist-treated mice despite lower levels of pro-inflammatory cytokines during early infection. During the chronic phase of infection, TRPV4 antagonist administration significantly increased lung bacterial burden, reduced alveolar macrophage infiltration, and elevated level of $CD8^+$ NKT cells. Collectively, our findings demonstrate that TRPV4 plays a critical and phase-dependent role in host defense against Mtb. Ameliorated chronic infection in the absence of Trpv4 and its expression in human TB lesions indicate TRPV4 as putative target for host-directed therapy.

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Cell Mediated Immune Regulation during Chikungunya Virus (CHIKV) Infection, Experimental Immune Suppression and Cancer Immunity

Our research aims to study the regulatory capacity of different cellular receptors and pathways in various contexts of cell mediated immunity (CMI). We seek to identify and strategically utilize these pathways for modulating immune cells responses for the amelioration of Chikungunya virus (CHIKV) infection, immune dysfunction associated to immune suppression and cancer immunity. Our recent works show an important role of TLR4 as a co-receptor and immune-regulatory molecule during CHIKV infection. Moreover, we have worked on different pathways of immune regulation associated to cancer immunity and immune-suppression. Recently we have demonstrated that targeting Neuropilin-1 (NRP1) could slow cancer progression and reduce peripheral Treg-mediated immunosuppression, making it a promising approach for future cancer immunotherapies, especially in combination with other treatment. Moreover, we have shown that CHIKV modulates cancer-induced immunosuppression through IL-10 dependent pathways, providing new insights into viral-cancer interactions. This research may contribute to developing novel antiviral immunotherapies and virotherapies beneficial for cancer patients and immunocompromised individuals. Together, we are exploring multiple immune-regulatory pathways for a proper management of immune responses to diseased conditions.

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Regulation of human T cell response

Regulation of the immune system is essential for preventing autoimmunity and enabling effective anti cancer responses. Regulatory T cells (Tregs) maintain immune tolerance by balancing pro and anti inflammatory signals, controlling effector T cell activity, and suppressing autoreactive cells. However, within tumors, Tregs can hinder anti cancer immunity by suppressing beneficial T cell responses. Our studies aim at identifying new means to control human Treg differentiation and function as well as that of human effector T cells. I will present our recent results including novel regulators of human T cell response.

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Decoding the specificity within Interferon regulatory factors in dendritic cell differentiation and innate immunity

The DC subsets are collection of heterogeneous populations that are broadly categorized into plasmacytoid DCs (pDCs) and classical DCs (cDC) represented by the phenotypically diverse cDC subsets (cDC1 and cDC2). IRF family members; Irf8 is essential for pDC and cDC1subtype whereas Irf4 is required for the cDC2 subtype development. A spontaneous IRF8R294C mutation in BXH2 mouse model abolishes the development of cDC1 subtype without impairing pDC development and eliminates production of IL12p40, while retaining that of type I IFNs. BATF3 cooperates with IRF8 towards development of cDC1 while higher expression of Irf8 can compensate for the absence of Batf3 for cDC1 development. In our recent study by employing global gene expression analysis, we revisit the role of Irf8 and Irf4 in defining the DC diversity and innate immune pathways.

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Characterization of Cytotoxic CD4+ T Cells in Visceral Leishmaniasis Patients

Control of Visceral leishmaniasis (VL), a parasitic disease caused by Leishmania donovani, requires a robust CD4+ T cell response to control parasite replication by interferon-gamma (IFN-γ) production and activation of macrophages. However, in VL patients, the anti-parasitic CD4+ T cell responses are ineffective for reasons that still are unclear. Our recent study reporting a transcriptional signature of CD4+ T cell isolated from the peripheral blood of active VL patients showed enhanced expression of genes related to cytotoxicity. This study investigates the cytotoxic potential of CD4+ T cells in VL patients, focusing on the expression of the key cytotoxic molecules granzyme B (GZMB), granulysin (GNLY), perforin (PRF), natural killer cell granule protein 7 (NKG7) and the degranulation capacity of CD4+ T cells during infection. We observed significant upregulation of these cytotoxic markers in CD4+ T cells from VL patients, particularly prior to anti-parasitic drug treatment (day 0), suggesting activation of these cells. However, the degranulation capacity, as indicated by CD107a expression, was comparable between VL patients and endemic controls (ECs), suggesting a potential functional impairment in the cytotoxic response. Yet, antigen-specific granzyme B secretion in whole blood cultures and intracellular GZMB production in CD4+ T cell subsets (notably Th1, Th9 and Th17/22 cells) were enhanced in VL patients, indicating a robust antigen-specific response, though this did not translate into effective parasite control. These findings highlight a paradox whereby CD4+ T cells from VL patients have heightened production of cytotoxic molecules, but lack translocation of CD107a to the cell surface, which is associated with an inability to eliminate parasites. This study provides new insights into the immune dysfunction in VL, highlighting a potential role for the cytotoxic phenotype that develops in parasite-specific CD4+ T cells. Targeting these pathways may offer novel therapeutic strategies to enhance immune responses and improve clinical outcomes in VL.

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Mechanism of innate immune responses

The innate immune system constitutes the first line of defense against invading pathogens, yet the molecular mechanisms governing host detection and response to infection remain incompletely understood. In this talk, I will present our recent findings identifying a novel RNA sensor that plays a critical role in regulating host responses to viral invasion. We demonstrate how this RNA-sensing pathway modulates innate immune signaling to shape effective antiviral defenses. In addition, I will discuss our work on the immunity-related GTPase IRGM, highlighting its function as a key regulator of immune responses. Our findings suggest that targeting IRGM represents a promising strategy to enhance both antiviral and anti-tumor immunity. Together, these studies provide new insights into innate immune regulation and reveal potential therapeutic avenues for infectious diseases and cancer.

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Diversity of Killer-cell Immunoglobulin like Receptors Influence Graft vs Host disease after Hematopoietic Stem Cell Transplantation

Killer-cell Immunoglobulin-like receptors (KIR), are transmembrane glycoproteins expressed on surface of Natural killer (NK) cells belong to a family of polygenic and polymorphic gene cluster mapped to a smaller region of chromosome 19q13.4, constituted by 15 coding genes and 2 pseudogenes. KIR receptors interact with a highly polymorphic gene cluster – Human leukocyte antigen (HLA) class I molecules (acts as ligands), regulating licensing, tolerance and activation of NK cells thus influencing several infectious and autoimmune diseases. Intricate balance mediated through HLA/KIR axis regulate post allogenic hematopoietic stem cell transplant (HSCT) allo-immune responses viz. Graft versus Leukemia (GvL) towards positive side while resulting Graft versus Host disease (GvHD) on the other side. However, the population specific influence of this clinically relevant KIR loci is largely unknown from Indian population, therefore, we analyzed this genomic locus in the healthy individuals and HSCT patient-donor pairs of Indian origin using allele specific PCR and next generation sequencing (NGS) based genotyping. Based on presence of Inhibitory and activating KIR genes, these were grouped into Haplotypes AA and Bx (including AB and BB). We observed a high frequency of Bx haplotypes in both healthy and HSCT patient donor pairs. Presence of Bx Haplotypes in donors is linked to reduced rate of GvHD in allogenic HSCT ($p=0.041$). In Haploidentical transplants, donors with AA haplotype transplanted to patients with Bx haplotype, significantly increased the risk of GvHD ($p=0.05$). These results suggest assessment of these KIR genes could support clinical decision in context of donor selection and GvHD management post HSCT.

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Regulating the regulators: how nutrient sensors control dendritic cell-driven tolerance

Dendritic cells (DCs) play a crucial role in regulation of adaptive immunity by controlling priming and polarization of T cell responses. Is has become clear that metabolic rewiring is fundamental to immune cell function including that of DCs. Central regulators of cellular metabolism are the nutrient sensor AMP-activated Kinase (AMPK) and its upstream activator Liver Kinase B1 (LKB1). Yet their role in DCs biology and function are only starting to be revealed. Our recent work suggests a key, but complex role for this AMPK signaling axis in regulating DC metabolism and T cell priming. We found the role for LKB1 to be distinct from AMPK; whereas LKB1 in DCs appears to be crucial for regulating central tolerance and Th17 responses, we found AMPK to be essential for DC-dependent control of peripheral regulatory T cell responses. I will discuss these findings alongside the consequences of targeting this signaling axis for outcome of inflammatory disease. These insights provide new mechanistic insight into metabolic pathways that control T cell priming by DCs and offer potential new therapeutic targets.

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Gut Metabolite Shape Antibody Quality By Impairing Tfh-B Cell Collaboration

Protective antibody responses depend not only quantity, but also on antibody quality, an attribute shaped within germinal centers through precise collaboration between T follicular helper (Tfh) cells and B cells. Our work focuses on this critical immune interaction to understand how durable and broadly protective humoral immunity is established. Using a controlled human vaccination model with the live-attenuated Japanese encephalitis vaccine (SA14-14-2), we conducted a three-year longitudinal study in vaccinated adults. We observed that humoral immunity was often limited and short-lived: only about two-thirds of individuals achieved seroconversion, and neutralizing antibody levels declined sharply within two years. Importantly, sustained seroconversion was strongly associated with robust, antigen specific Tfh cell responses. In contrast, around 18% of the non-seroconvertors showed weakened Tfh activity and defective T–B cell collaboration. To uncover the underlying mechanisms, we performed integrated immune and metabolomic analyses. This revealed a gut microbiota–derived metabolite that negatively regulates Tfh differentiation and disrupts germinal center function. Using functional assays across multiple antigen-specific and viral infection mouse models, we confirmed that this metabolite directly impairs Tfh development and B-cell help, thereby limiting the generation of protective humoral immunity. Together, our findings uncover a new dimension of immune regulation in which gut-derived metabolites directly shape T–B cell interactions and the quality of humoral immunity. By defining this metabolite–immune axis, our study provides a mechanistic explanation for inter-individual variability in vaccine responses and highlights new opportunities for designing next-generation vaccines that integrate immune and metabolic health.

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Tango of Host Amino Acid Metabolism and Mycobacterium tuberculosis Infection.

Upon *Mycobacterium tuberculosis* (Mtb) infection, host amino acid metabolism is perturbed at organismal, cellular and molecular levels. In this presentation, we will discuss about recent activities on how methionine, a sulfur containing essential amino acid, and its downstream metabolic pathways gets perturbed upon Mtb infection. Using isotopomer analysis, we observed that carbon units from methionine fuel polyamine metabolism, diverting the methyl group from SAM production towards the nucleotide salvage pathway. Interestingly, upon methionine supplementation in ex vivo and in vivo conditions, Mtb is cleared effectively, by increasing Il-6, Il-1B and TNF-α levels. Methionine supplementation reported to reduce drug induced liver injury (DILI) in tuberculosis patients and our findings suggest that it might have preventive and therapeutic benefit.

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Regulators of genome superstructure in the immune system

The mammalian immune system is a complex network of cellular players that function in synchrony to protect the body from potential pathogenic threats. The ability of immune cells to maintain themselves in the absence of pathogenic infection as well as their proficiency to respond to environmental stimuli is dependent on the cooperative activity of various molecular regulators that impact the transcriptional output of these cells. Recent data highlights the critical role of genome superstructure in gene regulation; however, how the regulators of genome superstructure shape the immune cell differentiation, activation and their function is only beginning to emerge. We are seeking to provide a comprehensive understanding of how molecular machines regulating genome superstructure dynamics integrate with gene regulatory machinery in the immune cells and I plan to discuss our recent work towards this goal

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Immunometabolic role of PANK4 in regulation of TLR7/9-mediated innate immunity

Innate immune responses are intimately linked to cellular metabolism, yet the molecular connections between metabolic reprogramming and antiviral defense remain incompletely defined. Here, we identify PANK4, an atypical member of pantothenate kinase family involved in coenzyme A (CoA) biosynthesis, as an unexpected regulator of RNA virus including influenza virus pathogenesis. Unlike other pantothenate kinases, PANK4 lacks canonical kinase activity but has been implicated in metabolic regulation. We show that influenza virus infection, in conjunction with pantothenic acid, induces PANK4 expression, which promotes viral replication by enhancing glucose uptake and glycolytic activity. Loss of PANK4 curtailed viral replication, reduced expression of glycolytic regulators, and heightened host antiviral defenses. Mechanistically, PANK4 interacts with UNC93B1 to suppress TLR7 and TLR9 mediated cytokine responses, thereby acting as a negative regulator of nucleic acid–sensing innate immune pathways in various cell-types. Furthermore, viral proteins NS1 and PB1 exploit PANK4 to amplify replication and immune evasion.

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

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

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Nuclear Receptor Co-repressors NCoR1 & SMRT fine-tune Immune-tolerance and inflammation

TLR-specific activation in antigen presenting cells (APCs) like macrophages and dendritic cells involves coordinated metabolic and transcriptional rewiring to elicit effective immune responses. The regulatory factors underlying this fine control of immune tolerance and inflammation (Yin and Yang) in health and disease remains elusive. We have recently reported that NCoR1 ablation generates immune-tolerant DCs through enhanced IL- 10, IL-27 and SOCS3 expression. Whereas, NCoR2 (SMRT) loss of function renders DCs highly inflammatory. High throughput transcriptional and metabolic analysis of NCoR1 and SMRT-depleted DCs hinted towards the involvement of discrete signalling pathways and metabolic processes to fine-tune the generated immune response. We found that NCoR1-depleted DCs meet their energy requirements through AKT-mTOR-HIF-1α- axis mediated glycolysis and CPT1a-driven β-oxidation to induce tolerogenic responses. Combined inhibition of HIF-1α and CPT1a using KC7F2 and etomoxir skewed the tolerogenic responses and rescued the decreased Th1 effector functions by compromising the overall tolerogenic signatures, specifically IL-10 cytokine (. On the other hand, SMRT- depleted DCs depicted reduced glycolysis and mTOR expression with enhanced anaplerotic glutamine metabolism to facilitate the truncated TCA cycle and succinate oxidation to maintain the inflammatory status. Here, inhibition of succinate transport with DEBM in sync with mTOR induction by Mhy-1485 skewed the inflammatory status. Physiologically, in DSS induced colitis model, treated SMRT-depleted DCs reduced the disease burden. Interestingly, In murine (DSS) and human inflammatory diseases (RA, Crohn's and SLE), SMRT and NCoR1 showed differential patterns of immunogenic and tolerogenic gene expression. Overall, we demonstrated that NCoR1 and SMRT levels in APCs define the fine-balance of tolerance and inflammation and thereby defines susceptibility towards inflammatory diseases and bacterial infections.

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Bridging Morphology and Transcriptomics in Immunology Through Single-Cell Phenomics

Understanding how immune cell morphology reflects underlying molecular state remains a fundamental challenge in immunology and cancer biology. This gap is particularly evident in acute myeloid leukemia (AML), where diagnostic workflows rely on invasive bone marrow aspiration followed by cytogenetic and molecular profiling. These procedures are time-consuming, non-standardized across centers, and often delay initiation of targeted therapy, despite mounting evidence that early intervention improves clinical outcomes. Morphology has historically played a central role in AML classification, and recent advances in imaging and deep learning now revive this modality as a quantitative, reproducible, and information-rich phenotypic layer. However, a major barrier has been the absence of technologies that can directly connect morphological cues to cellular transcriptomic and genetic states at single-cell resolution. I will introduce IRIS (Bues et al., BioRxiv, 2025), a newly developed single-cell phenomics platform that deterministically couples high-resolution imaging with deep transcriptomic profiling from the same individual cells. IRIS enables scalable, morphology-anchored molecular analysis and is compatible with both high-quality 3′ scRNA-seq and PacBio long-read sequencing, allowing detection of full-length transcripts, gene fusions, and genetic variants within single cells. This integrated framework makes it possible to dissect how genomic lesions and transcriptional programs shape cellular form and function. I will illustrate this potential using AML as a model, demonstrating how IRIS can characterize disease heterogeneity, identify morphology-associated molecular states, and provide rapid phenotypic readouts relevant for diagnosis. More broadly, IRIS establishes a generalizable platform for immune cell phenomics, offering a powerful bridge between morphology, transcriptomics, and functional state with broad applications in translational immunology.

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Disruption of Mesenteric Lymph Node Immune Surveillance Drives Bacterial Infections in Advanced Liver Disease

The gut-liver axis represents an integrated network of metabolic, neuroendocrine and immune interactions between the gastrointestinal tract and the liver. In health, gut epithelial and vascular barriers, together with local and systemic immunity of gut and liver, limit bacterial translocation (BT) into the circulation. Intestinal and mesenteric lymphatic vessels transport bacteria, microbial products and leukocytes to draining mesenteric lymph nodes (MLNs) for immune surveillance. In patients with advanced liver disease and cirrhosis, this tightly regulated gut-liver immune system fails, contributing to a high burden of bacterial infections. In our lab, we are studying the role and relevance of intestinal lymphatic vessels and MLNs in containing BT and preventing bacterial infections during chronic liver disease. In this talk, I will focus on how gut lymphatic vessels and MLNs contain BT and prevent bacterial infections in health, and how their dysfunction drives immune failure during chronic liver disease. MLN immune cell subsets and protein signatures associated with liver disease, with particular emphasis on the extracellular matrix proteoglycan, Versican (VCAN) will be highlighted. Mechanistic studies from our lab on how VCAN upregulation in MLN stroma is linked to excessive extracellular matrix deposition, impaired T-cell activation and migration, and the creation of an immunosuppressive niche that allows bacterial persistence in MLN will be discussed. Finally, translational relevance of our findings showing a strong association between circulating VCAN levels and infection-related mortality in critically ill cirrhotic patients, supporting its potential as a biomarker of immune dysfunction and prognosis will be provided.

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Precision medicine in Autoimmune rheumatic disease- need an integrated approach

Advances in immunology and molecular medicine have transformed the understanding and management of immune-mediated diseases, particularly autoimmune rheumatic diseases (AIRD). Conditions once considered organ-specific or primarily infectious are now recognized to involve complex immune dysregulation that evolves over time. In AIRD, therapeutic goals have shifted from merely preventing end-organ damage to modifying underlying immune pathways. While precision medicine has achieved major success in oncology and, to some extent, infectious diseases, its application in AIRD remains challenging because of disease heterogeneity, overlapping clinical phenotypes, and dynamic immunopathogenic mechanisms.

Autoimmune rheumatic diseases arise from an interplay of genetic susceptibility, environmental triggers, and immune dysfunction, leading to loss of self-tolerance and activation of multiple inflammatory pathways. This multistep and evolving nature limits the utility of single biomarkers or static diagnostic criteria. Both syndromic and criteria-based diagnostic approaches are currently used, but neither fully captures disease complexity or supports early, individualized intervention. Increasing evidence highlights significant heterogeneity both across and within diseases such as rheumatoid arthritis and systemic lupus erythematosus, further complicating precision-based decision-making.

Recent advances in targeted immunomodulation, including cytokine inhibitors, receptor blockers, and cell-directed therapies, have demonstrated that pathway-specific interventions can improve outcomes in selected patient subsets. Strategies such as patient clustering, composite biomarker models, and immune pathway profiling offer promise for improved stratification, although current predictive accuracy remains suboptimal. An integrated approach combining clinical features, immunological biomarkers, molecular signatures, and advanced analytics, including artificial intelligence, is essential to advance precision medicine in AIRD. Multidisciplinary collaboration and dynamic, data-driven models are required to guide individualized therapy selection, optimize outcomes, and move beyond population-based treatment paradigms.

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Histone deacetylases and the control of CD4+ T cell-mediated immunity

The differentiation of T helper cell subsets and their acquisition of effector functions are accompanied by changes in gene expression programs, which in part are regulated and maintained by epigenetic processes, such as chromatin modifications. Reversible histone acetylation at lysine residues is one of the best-studied histone modifications. It is controlled by histone acetyltransferases (HATs) and histone deacetylases (HDACs), which are considered transcriptional co-activators and co-repressors, respectively. In addition, many non-histone proteins are also acetylated and the acetylation status affects their functional properties, demonstrating that HDACs mediate effects beyond the epigenetic regulation of gene expression. HDAC inhibitors are used clinically for the treatment of certain types of hematological malignancies. Preclinical animal models indicate that HDACs also are promising targets to treat T cell-mediated immune diseases. In my presentation I will provide an overview about HDAC function in general and will then discuss results from ongoing studies of my laboratory about the characterization of the acetylproteome landscape in Th subsets as well as the regulation of CD4+ T cell immunity by HDAC1 and HDAC2 in the context of autoimmunity and infection.

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NK-1R signaling restrains Foxp3^{+} Treg differentiation and function during gut inflammation and autoimmunity

Neuroimmune communication at the gut-brain axis regulates gut inflammation and autoimmunity. Neurokinin receptors (NKRs; also known as tachykinin receptors, TACR) are expressed on neuronal and non-neuronal cells, and their expression is altered during inflammation. However, the mechanisms by which NK-1R signals influence CD4 T cell phenotype and function, and how these changes contribute to the regulation of gut inflammation, remain to be elucidated. C57BL/6 mice were given 2% dextran sodium sulfate (DSS) in drinking water to induce gut inflammation. NKR expression on various tissues and immune cells was analysed using quantitative RT-PCR, immunohistological staining, and multicolour spectral flow cytometry. Quantitative RT-PCR analysis of colonic biopsies from patients with inflammatory bowel disease (IBD) and irritable bowel syndrome (IBS) demonstrated elevated expression levels of NK-1R and NK-2R compared to control individuals. In a DSS-induced colitis model, administration of the NK-1R antagonist CP96345 or the NK-2R antagonist MEN10376 resulted in a marked reduction in gut inflammation and colitis severity. In contrast, the NK-3R antagonist Osanetant showed no significant effect. Multicolour spectral flow cytometry revealed that antagonism of TACRs significantly decreased the frequency of effector cells ($CD4^{+}CD44^{+}ROR\gamma t^{+}$) and increased regulatory CD4 T cells ($CD4^{+}Foxp3^{+}CD73^{+}CD62L^{+}LAP1^{+}$) within the Peyer's patch relative to controls. Furthermore, CD4 T cells were found to express NKRs, and inhibition of NK-1R during in vitro differentiation promoted Foxp3^{+} Treg development and enhanced their suppressive function both in vitro and in vivo.

This study demonstrated that antagonism of tachykinin facilitates the differentiation of regulatory CD4 T cells. We introduced a novel NKR-targeted approach for managing gastrointestinal inflammation and treating gut-associated inflammatory diseases in clinical settings.

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Single-cell analysis identifies shared inflammatory glial states across neurodegenerative central nervous system diseases

Age-related macular degeneration (AMD), Alzheimer's disease (AD), and progressive multiple sclerosis (MS) are neurodegenerative diseases of the central nervous system. Microglial dysregulation contributes to neuronal damage in AD and MS, but the cellular mechanisms driving chronic inflammation and vision loss in AMD remain unclear. We hypothesized that shared microglial activation states contribute to neurodegeneration across diseases and that the human retina could serve as a model to study these mechanisms. To identify cellular drivers of AMD, we performed single-nucleus RNA sequencing on retinal tissue from 11 individuals with AMD and 6 age-matched controls, generating the first single-cell atlas of AMD across disease stages. Disease-associated microglial states were compared with single-nucleus RNA-sequencing data from AD and MS. Using topological data analysis and diffusion geometry, we characterized cell clusters, identified pathogenic populations, and computed differentially expressed genes. Our analysis revealed two activated glial populations enriched in nonexudative AMD: one microglial and one astrocytic subset. These activated microglial states were also present in AD and MS, indicating a conserved inflammatory signature across neurodegenerative conditions. In neovascular AMD, we identified a novel microglia-to-astrocyte signaling axis driving VEGFA expression, a key mediator of pathological angiogenesis, which was validated in vitro and in vivo. This work presents the first single-nucleus transcriptomic atlas of the human retina in AMD and highlights cell-type specific drivers of disease. Our findings reveal shared microglial and astrocytic activation states across neurodegeneration and suggest that microglia-targeted interventions could preserve vision in AMD and slow neurodegeneration in AD and MS.

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SPD_1629 Mediates Uracil Uptake and Is Required for *Streptococcus pneumoniae* Fitness and Virulence *In Vivo*

Streptococcus pneumoniae relies on host-derived nutrients to sustain proliferation and virulence, yet the mechanism of uracil acquisition has remained undefined. In this study, we identified and functionally characterized SPD_1629 as the primary uracil transporter required for pneumococcal fitness and pathogenesis. Using the toxic uracil analogue 5-fluorouracil (5-FU) we observed dose-dependent growth inhibition in vitro that was specifically reversed by excess uracil, indicating a dedicated uptake pathway. Bioinformatic screening identified SPD_0267, SPD_1141 and SPD_1629 as potential candidates. Genetic analysis showed that deletion of spd_1629 (D39Δspd_1629) conferred partial resistance to 5-FU, whereas deletion of spd_0267 or spd_1141 did not, and the triple mutant phenocopied D39Δspd_1629. In contrast to growth in rich medium, D39Δspd_1629 failed to grow in a uracil-sufficient chemically defined medium, and [3H]-uracil uptake assays directly validated SPD_1629 specificity for uracil. In mouse infection models, D39Δspd_1629 exhibited significantly reduced bacterial loads in lung, blood and spleen, and a marked competitive index defect vis-a-viz the wildtype strain. Deletion of spd_1629 also decreased capsular polysaccharide levels, correlating with attenuated virulence in pneumonia and sepsis models. This phenotype links uracil salvage to capsule biosynthesis and highlight the critical role of SPD_1629 in in vivo fitness and virulence.

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Breaking the Metabolic Lock: Sphingolipid-Driven T Cell Exhaustion in Immunotherapy-Resistant Colorectal Cancer

Despite the success of immune checkpoint inhibitors in several cancers, colorectal cancer (CRC)—particularly microsatellite-stable (MSS) tumors—remains largely refractory to immunotherapy. These tumors exhibit immune exclusion and profound dysfunction of tumor-infiltrating $CD8^+$ T cells. To understand the mechanisms underlying this resistance, we integrated single-cell RNA sequencing data from independent CRC cohorts to map the tumor immune microenvironment. Our analysis revealed a continuum of $CD8^+$ T cell exhaustion, with tumor-derived T cells displaying marked metabolic paralysis. Notably, sphingolipid metabolic pathways were highly enriched along the exhaustion trajectory, and phospholipid phosphatase 1 (PLPP1) emerged as a key upregulated regulator.

Functional assays demonstrated that ceramide accumulation and disruption of sphingosine-1-phosphate signaling promote T cell exhaustion, mitochondrial dysfunction, and oxidative stress. Inhibition of sphingolipid synthesis significantly reduced exhaustion markers and restored mitochondrial health. These findings identify sphingolipid metabolism as a druggable immunometabolic checkpoint and suggest metabolic reprogramming as a promising strategy to restore T cell function in CRC.

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Checkpoint molecules an evasion mechanism in HIV-Mtb co-infection

People living with Human Immunodeficiency virus (PLWHA) having Mycobacterium tuberculosis (Mtb) co-infection experience faster progression to AIDS and remains a challenge for disease management. Understanding of the immune-activation, breakdown of immune defence and synergistic effect between HIV and Mtb may provide essential information regarding key signature pathways involved in the pathogenesis of HIV disease in co-infected host. Earlier studies from our lab and others showing genetically diversified evolution of HIV in the co-infected host suggest dysregulated immune-mechanisms in coinfected individuals and led us to hypothesize that Mtb modulates key regulatory mechanisms to evade the immune response in coinfected host. Targeting some of these key pathways may lead to innovative strategies for improving the management of these individuals. A comparative assessment of gene and surface expression of regulatory molecules on T-cells of treatment-naïve recruited human cohorts from North India revealed a significantly dysregulated expression of many regulatory genes including the host intrinsic factors and immune-checkpoint molecules on both the CD4 and CD8 T-cell subsets of HIV-Mtb co-infected individuals in comparison to HIV mono-infected and other groups. The expansion of 'terminally exhausted' T-cells in co-infected individuals correlating positively with viral load negatively with CD4 T-cell count conforms to faster disease progression. Additionally, advanced analysis of bulk mRNA sequencing (RNAseq) of peripheral blood mononuclear cells from these individuals revealed dysregulated primary pathways including Interferon signalling, cytokine signalling, antiviral mechanisms through IFN-stimulated genes, and negative regulation of viral mRNA translation and peptide chain elongation. These findings highlight the Mtb mediated dysregulated molecular signature in HIV-Mtb co-infected individuals, which may collectively contribute to enhanced viral replication and increased cell death leading to faster disease progression in co-infected host. These findings underscore the need for further exploration into the possibility of targeting multiple molecules collectively that may offer adjunct immune-therapeutic options for better management of co-infected individuals.

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IL-17A signalling blockade: A powerful therapeutic modality to combat rheumatoid arthritis and psoriasis

Therapeutic targeting of IL-17A in autoimmune conditions has demonstrated efficacy in mitigating the pathological manifestations of rheumatoid arthritis and psoriasis. Our laboratory has developed strategies employing small-molecule inhibitors of IL-17A, specifically cyanidin, in the context of rheumatoid arthritis and psoriasis. Recently, we identified cyanidin as a potent therapeutic agent capable of restoring the impaired efferocytosis mechanism in rheumatoid arthritis and modulating the ferroptotic program in keratinocytes in psoriasis, both of which are regulated downstream of the IL-17A signaling pathway. Our experimental findings suggest that cyanidin has the potential to restore the impaired efferocytosis machinery of synovial macrophages by regulating the expression of the metalloproteinase ADAM17 via the JAK1-JAK3/STAT signaling axis downstream of IL-17A. Our previous research on cyanidin has notably demonstrated its capacity to restore immune tolerance and enhance the expression of PD-L1 negative checkpoints. Recent findings from our laboratory strongly indicate that cyanidin holds significant promise for broader translational applications, thereby reinforcing its potential as a therapeutic agent for rheumatoid arthritis (RA) and psoriasis. This is further substantiated by our recent patent grant for a pharmaceutical composition aimed at treating psoriasis. While we acknowledge the limited success of IL-17A targeted biologics in clinical settings for RA, we continue to observe substantial pathological effects of the cytokine in exacerbating symptoms associated with joint disease. Therefore, we suggest that cyanidin, a small-molecule inhibitor, should be considered a therapeutic target in the clinical management of RA and psoriasis.

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Cancer immunotherapy and beyond

Immunotherapy has revolutionized cancer treatment by enabling the body's immune system to target and destroy tumor cells. Approaches such as immune checkpoint blockade and adoptive cell transfer have shown remarkable promise, establishing themselves as a new frontier in oncology. However, a significant number of patients fail to respond or eventually develop resistance to these therapies. One major contributor to this challenge is the accumulation of T-regulatory cells within the tumor microenvironment. These cells suppress immune activity, fostering a tolerogenic environment that shields the tumor from immune attack. Advanced single-cell genomic analyses have highlighted the complexity and heterogeneity of these Treg populations in cancer. At the heart of Treg activity lies the transcription factor FOXP3, which orchestrates immune suppression through a dynamic network of interacting partners, including epigenetic modulators and immune tolerance regulators. These findings have shifted research efforts toward understanding how to disrupt Treg cell function to restore effective anti-tumor immunity. This seminar will explore the evolving landscape of cancer immunotherapy, with a focus on the mechanisms by which Treg cells contribute to treatment resistance. We will discuss current strategies aimed at reprogramming the tumor microenvironment, from one of immune suppression to immune activation, and highlight novel therapeutic targets that could help extend the benefits of immunotherapy to a broader patient population

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Interferon-gamma-induced nitric oxide responses: Yes to NO!

Interferon-gamma (IFN-γ), the sole member of the type-II interferon family, is well known to protect the host from infectious diseases as well as mount anti-tumor responses. Our laboratory has been studying the in vitro roles of IFN-γ-induced nitric oxide (NO) on tumor cell growth and macrophage responses. IFN-γ-activation increases glycolytic flux and reduces mitochondrial function in nitric oxide (NO)- and reactive oxygen species (ROS)-dependent manner in the H6 hepatoma tumor cell line. These processes are accompanied by Hypoxia inducing factor (HIF)-1α stabilization and HIF-1α-dependent augmentation of the glycolytic flux. The IFN-γ enhancement of lactate production also occurred in other NO-producing cell lines: RAW 264.7 monocyte/macrophage and Renca renal adenocarcinoma. However, two other tumor cell lines, CT26 colon carcinoma and B16F10 melanoma, did not produce NO and lactate upon IFN-γ-activation. HIF-1α stabilization upon IFN-γ-activation led to lower cell growth of B16F10 but not CT26 cells. Clinical studies have shown the crucial roles of IFN-γ for successful cancer immunotherapies and the possible implications of our study will be discussed. In addition, this talk will also highlight some of our recent insights into IFN-γ-modulated genes identified by RNA seq. The roles of several genes and novel pathways induced IFN-γ-induced NO will be discussed. Finally, the roles of some inhibitors of IFN-gamma-induced NO will be discussed along with their potential in vivo effects in pre-clinical models of disease.

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Use of reverse vaccinology approach for the development of vaccine against *Streptococcus pyogenes* infections

Streptococcus pyogenes, also known as Group A *Streptococcus* (GAS), is a gram-positive human pathogen which causes impetigo, pharyngitis, scarlet fever, necrotizing fasciitis, STSS (Streptococcal Toxic Shock Syndrome) and post-infection effects which involves ARF (Acute Rheumatic Fever), RHD (Rheumatic Heart Disease). Global estimates indicate 18 million cases of GAS infections occur every year, with 1.78 million cases turning into severe ailments. Amongst infections RHD affecting 15.6 million people globally which results in 282,000 new cases annually. Increased cases of GAS infections were reported not only from less developed medical settings like in African countries but also from well developed medical setting like in many countries of Europe. Henceforth, WHO has given ninth rank to GAS among the highest infectious bacteria and has updated its Bacterial Priority Pathogens List (BPPL) based on antibiotic resistance. The current pipeline related to vaccine development includes candidates based on M protein and non-M protein antigens, Based on M protein, few vaccines preparations like 26 valent, 30 valent, and J8 have reached clinical trials phase I/ II. One of the major impediments in GAS vaccine development involve high genetic diversity of antigen targets, lack of relevant animal models, and cross-reaction of antibodies with antigens resulting in autoimmune disorders. Till now, not a single GAS vaccine has been approved or licensed for human use. We have shown that SPy_2191 protein conserved across GAS serotypes (98%) prevalent in developed and developing countries and was found to be significantly protective against various invasive GAS serotypes. Additionally, protective immunity to GAS is not well understood. Therefore, in the present study signal peptide and His-tag sequence free SPy_2191 vaccine formulated with a human-compatible adjuvant i.e., alum was used to check its protective potential against GAS infections. We found that our vaccine formulation imparted a strong serum antibody response (total IgG), with iso-typing profiles leaning towards the IgG1 and IgG2b subclasses. Increased levels of Th1-specific (IFN-γ, TNF-α and IL-2), Th2-specific (IL-4) and Th17-specific (IL-17a) were observed in immunized mice as compared to control suggests antigen re-stimulation mimics its potentiality for a memory-recall response against GAS infections. High levels of chemokines (MCP-1/ CCL2, MIP-1α/ CCL3, MIP-1β/ CCL4 and RANTES/ CCL5) indicate orchestrated a strong but controlled immune response in immunized mice. The cell-mediated immune response provides valuable insights into the production of various T-cell populations in immunized mice indicating antigen recall response and the development of long-term immune memory. Importantly, our in-vitro and in-vivo toxicology study showed that SPy_2191 protein is non-toxic. This study will form the basis of SPy_2191 pre-clinical trials in the pursuit of an effective vaccine preparation to deal with GAS infections.

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Bridging Innate and Adaptive Immunity: Next-Generation Adjuvants for Veterinary Vaccines

Enhancing vaccine efficacy in livestock remains a global priority, particularly for pathogens where existing adjuvants fail to induce durable or sterilizing immunity. Alum is most widely used and safest adjuvant in both Animal and Human Vaccines. However, it induces short term immunity mainly antibody response and to lesser degree cell mediated immunity. Due to limited immunostimulatory activity they are not able to efficiently activate innate immune response which are drivers for adaptive immunity. Hence there is a need to enhance the potency of existing adjuvants or develop new adjuvant formulation. Keeping in view the cost, timeline and other technical hurdles associated with developing new adjuvant formulations, enhancing the potency of existing clinical adjuvant would be smarter strategy that will save time and money especially in context of application in veterinary field. Monophosphoryl Lipid A (MPLA) is a well-known Toll-like receptor 4 (TLR4) agonist and a clinical adjuvant. However, its large, heterogeneous structure complicates synthesis, increases costs, and occasionally leads to toxicity. Synthetic small-molecule TLR agonists present a promising alternative, offering simpler synthesis, lower costs, reduced toxicity, and enhanced potency when combined with other adjuvants. Our group focuses on engineering next-generation adjuvants that strategically activate innate immune pathways to drive strong and long-lasting adaptive responses across species. We identified and characterized NSF-951, a small-molecule TLR4 agonist, and developed optimized formulations—including an Alum-based system—that significantly enhanced dendritic cell activation, cytokine production, and antigen-specific memory responses. These formulations demonstrated robust immunogenicity in murine models using both bacterial and viral antigens and produced sustained antibody and memory responses in goats. Parallel work on Leptospira Lipid A revealed its unique ability to activate TLR4 with controlled reactogenicity, achieving sterilizing protective immunity in a hamster model. Through Indo-US collaborative program our group is developing novel FMD vaccine candidates using liposomal and aqueous formulations combining TLR4 and TLR7 agonists to elicit broad, durable immunity in large animals. Together, these studies demonstrate how rational design of innate-targeting adjuvants can effectively bridge innate sensing and adaptive protection, offering a translational pathway toward next-generation veterinary vaccines.

FACULTY SHORT TALK ABSTRACTS

Delegate Faculty Talk

Long-Term Systemic Sequelae of SARS-CoV-2: A Multi-Omics Analysis of Neuroinvasion and Extrapulmonary Pathology in Longitudinal Animal Models

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Post-acute COVID-19 syndrome (PACS) is increasingly recognized as a cause of persistent neurological dysfunction, yet the mechanisms linking prior SARS-CoV-2 infection to long-term neurodegeneration remain poorly understood. To elucidate the cellular and molecular mechanisms driving PACS-associated neurodegeneration, we used a multi-model longitudinal cohort study involving hACE2 mice and Syrian hamsters infected with SARS-CoV-2 Omicron BA.1, followed for up to one-year post-infection. Persistent viral signals were detected in the brain during the post-acute phase, coinciding with sustained activation of the innate immune compartment, characterized by enrichment of pro-inflammatory macrophages and neutrophils and prolonged elevation of TNFα and IFNγ up to 180 days post-infection. Integrative proteomic and metabolomic analyses revealed a convergent pathological signature involving TNFα signaling, calcium dysregulation, mitochondrial stress, and apoptotic pathways, accompanied by impaired bioenergetic homeostasis. These molecular alterations were associated with mitochondrial depolarization, increased oxidative stress, and enhanced apoptosis in brain immune and neural populations. PACS animals also exhibited elevated circulating neurodegeneration-associated biomarkers and persistent neurological and behavioral deficits. Our findings define a chronic innate immune–mitochondrial axis that drives progressive neurodegeneration during PACS. This study provides mechanistic insight into how sustained inflammatory signaling following SARS-CoV-2 infection contributes to long-term neurological sequelae and highlights innate immune and mitochondrial pathways as potential therapeutic targets for post-COVID neurological disease.

IFN levels and IFN signature identify patient subsets with distinct clinical and immunological characteristics in SLE

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The interferon (IFN) system is dysregulated in Systemic Lupus Erythematosus (SLE) and represents a potential therapeutic target. However, a comprehensive analysis of different IFN types remains limited. The present study aimed to assess role of circulating levels of IFN types and IFN score to evaluate their association with disease activity in SLE patients from Western India. SLE patients with IFN levels above third quartile were categorized as 'IFN-high' groups where their association with clinical and autoantibody profile was investigated. Additionally, cluster analysis based on autoantibodies profile was performed to identify distinct immunophenotypic endotypes.This cross-sectional study included, clinically diagnosed SLE patients (n=115). Disease activity was assessed using SELENA-SLEDAI. Serum levels of IFNα and IFNλ1-λ4 were detected using ELISA, while IFNγ levels were detected using bead-based assay. IFN score was determined using RT-qPCR. Statistical analyses included Mann-Whitney U and Kruskal-Wallis tests, Spearman correlations, chi-square tests, and logistic regression analysis. Hierarchical clustering based on autoantibody profiles was performed.

Dysregulation in IFN pathways with a significant correlation between IFNα and IFN score with SLEDAI suggested their potential utility as possible biomarkers for monitoring SLE disease activity. The association of different 'IFN-high' subgroups with specific clinical and autoantibody profile further suggested that patient stratification according to their IFN profile. This can guide the development of more targeted therapeutic approaches for SLE disease management.

Delegate Faculty Talk

Unravelling Mucosal Immunity–Microbiota Interactions: From Host Defence to Beneficial Symbiosis

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Dysregulation of intestinal microflora contributes to inflammatory disorders by impairing the immunosuppressive functions of Foxp3⁺ regulatory T (Treg) cells. Although commensal microbiota influence Treg generation, the key microbial and dietary factors governing this process remain unclear. We identified Bifidobacterium bifidum as a potent inducer of Foxp3⁺ Treg cells with diverse antigen specificities toward both dietary and commensal antigens. Notably, cell surface β-glucan/galactan (CSGG) polysaccharides of B. bifidum were revealed as the principal effector molecules responsible for Treg induction. Purified CSGG recapitulated the bacterium's Treg-promoting activity through the programming of regulatory dendritic cells (DCs). The resulting Treg cells maintained stable Foxp3 expression, exhibited strong suppressive capacity, and effectively ameliorated experimental colitis, highlighting the immunomodulatory potential of CSGG-producing microbes. Complementing the microbial findings, we also demonstrate that dietary glucose serves as a critical determinant of retinal dehydrogenase (RALDH) activity in small intestinal CD103⁺CD11b⁺ DCs—an essential step for converting vitamin A into retinoic acid (RA), thereby enabling induced Treg (iTreg) differentiation. Using germ-free mice on antigen-free diets, we show that proteins, starch, and minerals are dispensable for this process, whereas sufficient glucose availability is vital for establishing RALDH activity and supporting DC-mediated iTreg generation. Together, these findings reveal how microbial polysaccharides and dietary glucose synergistically shape intestinal immune tolerance by programming tolerogenic DCs and promoting Foxp3⁺ Treg differentiation. This integrated mechanism offers promising therapeutic avenues for restoring mucosal homeostasis in inflammatory bowel diseases.

Immune Protection After Quadrivalent Influenza Vaccination Among Elderly Indians

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Influenza virus continues to pose a significant public health challenge and economic burden due to its ability to cause annual seasonal epidemics and unpredictable pandemics. Despite widespread vaccine use, achieving broad and durable protection remains a significant challenge. This limitation underscores a fundamental gap in our understanding of human immunology, particularly the mechanisms required to induce long-lasting and broadly protective immune responses at the population level. The phase IV clinical trials for Quadrivalent influenza vaccine in India and Europe will identify vaccine responsiveness in different vulnerable populations. For this purpose, human serum samples from vaccinated Indian elderly (≥ 60yrs)
were tested for the presence of influenza specific neutralizing antibodies by performing conventional serological assays and T cell phenotyping.
The antibody response detected using hemagglutinin specific total IgG antibody titer in the serum, whereas the hemagglutination inhibition (HAI) titer led us to screen responses to relevant influenza strain in vaccinated individuals. The viral micro neutralization assay (MN) performed to determine the cross reactivity to related virus strains. We quantified the proportion of vaccine efficacy mediated by post-vaccination and observed significant response differences in H1N1, H3N2 and B lineage strains towards the vaccine used in the study. This data can be used as the serological background level of the vulnerable population and the study will able to determine the vaccine immunogenicity and effectiveness towards next generation vaccine.

Delegate Faculty Talk

Impact of aging on monocyte function and breast cancer progression

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Aging is the greatest risk factor of breast cancer, and two-thirds of breast cancer occur in women over 50 years of age. Tumor associated macrophages (TAMs) trigger an immune suppressive condition by inducing regulatory T cells which support tumor progression instead of cancer cell clearance. In this context, aging causes extensive remodeling of mammary gland cells including induction of immune regulatory macrophages. Aging also induces cellular senescence that promotes cancer progression. However, the link between cellular senescence and immunosuppression in aging leading to breast cancer progression is unclear. Our data from single cell RNA-sequencing and flow cytometry analysis revealed upregulated expression of senescence and immune regulation associated receptor CD74 in aged mouse monocytes. We have also found that a subset of Cd74-high monocytes, which is enriched in aged mice has downregulated cell cycle checkpoint and upregulated immune regulatory gene expression, validating CD74's role in cellular senescence and immune regulation in aging. We aim to investigate CD74's role in connecting cellular senescence and immunosuppression in breast cancer mouse models, and test whether combination therapy of senolytic drugs or CD74 inhibitor can improve immune checkpoint blockade therapy for breast cancer.

STUDENT SHORT TALK ABSTRACTS

Oral Talk 1 and Poster No. 85

Harnessing Tumor Derived Extracellular Vesicle-Primed Dendritic Cell Exosomes for both Immunogenic and Cytotoxic Targeting of Oral Squamous Cell Carcinoma

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Immunotherapy has emerged as a promising approach to improve cancer outcomes; however, the clinical translation of cell-based methods is restricted by scalability and safety concerns. In this study we investigate the exosome-based cell free immunotherapy platform for oral squamous cell carcinoma (OSCC), aiming to overcome the scalability and safety constraints associated with conventional cell-based immunotherapies. Dendritic cell-derived exosomes (Dexosomes) were generated by priming mature dendritic cells, differentiated from the THP-1 cell line, with tumour-derived extracellular vesicles (TEVs) obtained from KB1 oral cancer cells and subsequently loading these vesicles with metformin. Dexosomes showed a size range of 30-200 nm with spherical morphology, negative zeta potential, and expression of exosomal markers CD9, CD81, HSP70, and TSG101. TEV-primed dexosomes demonstrated elevated expression of immunostimulatory markers CD80, CD86, and HLA-DR. Functional assays demonstrated that these dexosomes significantly enhanced lymphocyte activation in co-culture models. Metformin-loaded TEV-dexosomes (TEV-Dex-Met) were prepared via sonication, confirming efficient drug encapsulation and retention of vesicular integrity. Upon internalisation by KB1 OSCC cells, TEV-Dex-Met induced a substantial increase in cell death, ROS generation, and migration inhibition when compared to free metformin. Further, clinical validation using dexosomes derived from histopathologically confirmed OSCC patient samples indicated a significant enhancement of autologous lymphocyte activation ($p < 0.005$), while TEV-Dex-Met further amplified immune responses and produced marked cytotoxicity (>50%) in resistant primary tumour cultures ($p < 0.0001$). These findings underscore the translational potential of TEV-primed dexosomes as a scalable, biocompatible, and personalized therapeutic platform that integrates immune stimulation and targeted cytotoxicity, offering a promising strategy for treating advanced OSCC.

Oral Talk 2 & Poster No. 90

The heterogeneity in normal pancreatic fibroblasts and its implication in CAFs mediated tumor immune-modulation and pancreatic cancer progression

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Heterogeneous populations of carcinoma associated fibroblasts (CAFs) are identified in human and mice pancreatic ductal adenocarcinoma (PDAC). A better understanding of the novel subpopulations of CAFs and their functional significance in the tumor micro-environment (TME) will provide rationale and strategy for targeted therapies against PDAC. Thus, our objective was to underscore the heterogeneity underlying the normal pancreatic fibroblasts (PFs) which can contribute to the CAF heterogeneity and to check the tumorigenic and/or therapy modulating properties of specific CAF subtypes originating from the PF. Thus, we conducted single cell analysis of mouse pancreatic fibroblasts and further examined the tumor modulating properties of a specific inflammatory marker 'LY6C' expressing fibroblasts using flow-cytometry, bio-plex, qRT-PCR and other relevant techniques in an in-vitro setting. Our study results suggest that, normal mice pancreatic fibroblasts exhibit distinct molecular heterogeneity with several subtypes showing inflammatory phenotypes and similarities with CAF subtypes of pre-neoplastic and neoplastic pancreas. Importantly, the tumor modulating properties of LY6C +ve and -ve fibroblast subtypes have been studied in-vitro which also dictated changes in the percentage of myogenic and inflammatory fibroblasts in presence of cancer cells. Further, the inflammatory/immune-phenotypes of these fibroblasts subtypes are characterised and the immunomodulatory effects of these fibroblasts were also studied. Thus, together this study highlights the molecular diversity of fibroblasts of normal mice pancreas and their similarity with CAF heterogeneity in mice PDAC and also elucidates the role of LY6C -ve and +ve fibroblasts in PDAC progression and tumor immune-modulation suggesting their decisive potential in PDAC outcomes.

Oral Talk 3 & Poster No. 84

From Structure to Immunity: Characterizing Leptospira FlaB as a Potent Vaccine Adjuvant

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Flagellin is a well-known TLR5 agonist used as a vaccine adjuvant, with Salmonella FliC serving as the classical model. However, the immunostimulatory potential of Leptospira flagellin (FlaB), an endoflagellar protein with distinct structural features, remains largely unexplored. To address this gap, we evaluated the adjuvant activity of Leptospira FlaB in comparison with Salmonella FliC using a combination of computational modeling, in vitro assays, and in vivo immunization studies. In silico docking and molecular dynamics simulations showed strong interaction of Leptospira FlaB with mouse, human, and bovine TLR5 than those observed for FliC. Consistent with these predictions, FlaB triggered TLR5-dependent IL-8 induction in HEK-TLR5 reporter cells and demonstrated activation of RAW264.7, THP-1, iBMDM, and bovine macrophages. In vivo, LigA formulated with FlaB elicited higher antigen-specific IgG responses than LigA alone, though the responses remained lower than those induced by LigA + FliC. Engineering a FlaB–LigA fusion protein substantially improved immunogenicity relative to co-administered FlaB and LigA, indicating that covalent linkage enhances the adjuvant effect. Interestingly, the addition of Alum to the fusion protein diminished antibody responses, suggesting interference between Alum and the flagellin–TLR5 signaling axis. Overall, this study provides the first detailed characterization of Leptospira flagellin as a TLR5-targeting agonist and demonstrates that it can effectively augment LigA immunogenicity, particularly in a fusion format. These findings highlight the adjuvant potential of Leptospira flagellin.

Oral Talk 4 & Poster No. 86

Hsp16.3ΔC4, a C-terminal truncated mutant, confers strong cellular and humoral immunity against pulmonary tuberculosis

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Despite its widespread use, the Bacillus Calmette-Guérin (BCG) vaccine offers limited and variable protection against adult pulmonary tuberculosis (TB), underscoring the pressing necessity for next-generation vaccines capable of eliciting long-lasting protective immunity. We report here the development and protective efficacy of a subunit vaccine derived from a C-terminal truncated variant of the *Mycobacterium tuberculosis* (*Mtb*) Hsp16.3 protein (Hsp16.3ΔC4). We have utilized both in vitro and in vivo methodologies to evaluate the efficacy of a subunit vaccination against tuberculosis. Flow cytometry, ELISA, and bead-based multiplex immunological assays were employed to measure antigen-specific immune responses, encompassing B- and T-cell activation, cytokine production, and IgG antibody titres. CFU assay and histology were performed in order to evaluate bacterial load and lung pathology. Subcutaneous immunization with Hsp16.3ΔC4 significantly reduced pulmonary bacterial burden and pathology by enhancing both cellular and humoral responses. Notably, co-administration of Hsp16.3 mutant with BCG synergistically augmented Th1 immunity, characterized by elevated IFN-γ and IL-2 levels, and increased frequencies of activated memory T and B cells. Co-administration with BCG was associated with higher levels of IL-17A, IL-12p40, IL-9, MIP-1β, and GM-CSF production necessary for bacterial control. Adoptive transfer experiments further demonstrated that $CD4^+$ T cells serve as the main effectors responsible for conferring protection, underscoring the central role of antigen-specific Th1 immunity in mediating vaccine-induced defence against Mtb.Together, these findings position the Hsp16.3 mutant as a stand-alone vaccine candidate on its own, as well as in conjugation with BCG, applicable to both infant immunization regimens and (re)vaccination approaches for adults.

Oral Talk 5 & Poster No. 83

Co-option of the host ACVR1-iron axis drives iron remodelling and enables a permissive niche for Toxoplasma gondii replicatio

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Intracellular pathogens rely on host signalling networks to establish a permissive niche; however, the host kinases co-opted during infection remain poorly defined. To systematically uncover such dependencies, we exploited the polypharmacology of kinase inhibitors and combined their pre-existing activity profiles with parasite burden phenotypes to computationally infer host kinases that regulate Toxoplasma gondii replication. This approach, applied across ~300 kinases using 32 broad-spectrum inhibitors, identified ACVR1/ALK2 as a previously unidentified host determinant of Toxoplasma development in the host. We find that Toxoplasma preferentially infects host cells with high basal ACVR1 expression and induces a temporal, selective activation of the ACVR1-SMAD1/5/9 axis. A pronounced increase in phosphorylated SMAD1/5/9 is observed specifically between 6-12 hours post-infection, corresponding to the onset of rapid parasite expansion within the parasitophorous vacuole. Pharmacological inhibition or genetic silencing of ACVR1 suppresses SMAD phosphorylation, disrupts vacuolar architecture, and markedly reduces parasite replication, establishing ACVR1 as a key regulator of intracellular development.

Integration of multiple existing datasets of infected host cells consistently revealed early activation of iron-regulatory genes, a downstream branch of the ACVR1-SMAD pathway. Functionally, iron supplementation fully rescues parasite growth in ACVR1-inhibited or knockdown cells, demonstrating that ACVR1 supports parasite development by maintaining a bioavailable intracellular iron pool essential during the 6-12 h early expansion window. Notably, iron resupply restores the initial phase of parasite growth, corroborating the timing of p-SMAD activation. Importantly, in vivo administration of a selective ACVR1 inhibitor in C57BL/6 mice markedly reduced parasite burden, confirming the requirement of ACVR1 signalling for successful parasite establishment under physiological conditions.

Toxoplasma secretory factors were sufficient to activate the ACVR1-SMAD axis, and interrogation of an existing pooled CRISPR screen with dual host-pathogen single-cell transcriptomic readouts enabled us to shortlist parasite effectors likely responsible for this activation. Ongoing work is focused on identifying the specific effector(s) that regulate this pathway. Together, these findings identify ACVR1 as a mechanistically validated signalling hub hijacked by Toxoplasma gondii to remodel host iron homeostasis, establishing the ACVR1-iron axis as a key host vulnerability that can be targeted to disrupt intracellular parasite development.

Oral Talk 6 & Poster No. 87

Host Genetic and Epigenetic Regulation of MHC-II as a Predictor of Malaria Clinical Outcomes

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Malaria caused by Plasmodium species trigger complex immune regulatory mechanisms that influence disease severity and parasite clearance. This study investigates how promoter polymorphisms in the MHC Class II DRB gene, together with C2TA (Class II Transactivator)–mediated regulation and chromosome 6–specific DNA methylation patterns, shape MHC-II expression in malaria patients. Peripheral blood samples from confirmed Plasmodium infected cases were analyzed to assess DRB promoter variants, C2TA expression levels, and epigenetic modifications at key regulatory regions. Methylation-sensitive assays were used to evaluate DNA methylation signatures across chromosome 6, focusing on loci associated with MHC-II transcriptional control. Gene expression analysis quantified MHC-II (DRB) surface expression on circulating immune cells, correlating these findings with parasitemia and clinical outcomes. Results demonstrated that specific DRB promoter polymorphisms combined with altered C2TA expression significantly influenced MHC-II levels during infection. Hyper-methylation at the DRB promoter and adjacent regulatory regions on chromosome 6 was associated with reduced C2TA activity, leading to diminished antigen-presentation capacity and higher parasite burden. Conversely, patients exhibiting lower methylation and intact C2TA–MHC-II signaling showed better parasite control. These findings indicate that genetic and epigenetic regulation of MHC-II expression, driven by interactions between DRB promoter architecture, C2TA transcriptional activity, and chromosome 6 methylation, collectively modulate host immune competence against P. falciparum. This integrated immunogenetic framework highlights potential biomarkers for susceptibility and therapeutic targets for enhancing antigen presentation in malaria.

Oral Talk 7 & Poster No.88

Profiling Complement Proteins in Pediatric Cases of Tuberculosis and SARS-CoV-2 Seropositivity

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Coinfection with *Mycobacterium tuberculosis (M.tb)* and SARS-CoV-2 poses unique challenges in paediatric population due to overlapping immune pathways and potential immune dysregulation. The complement system, a key arm of innate immunity, plays a dual role in pathogen clearance and inflammation. This study aimed to investigate complement activation in children with Tuberculosis (TB), stratified by SARS-CoV-2 serostatus. Children aged 2–17 years with pulmonary or extrapulmonary TB were enrolled from two tertiary hospitals in South India and grouped by SARS-CoV-2 IgG serostatus: CoV2$^+$ (n=88) and CoV2$^-$ (n=88). Blood was collected at baseline, month 3, and month 6 for complement profiling. Classical, lectin, and alternative pathway proteins and regulatory factors were quantified using multiplex bead assays. Canonical discriminant and Spearman correlation analyses were performed to assess temporal and relational complement patterns. CoV2$^+$ children had significantly elevated levels of classical pathway proteins (C1q, C3, C4, C5a) and regulatory factors (Factor B, H, and I) at baseline, and follow-up in comparison to CoV2- children. However C2 and C5 dimnished in CoV2$^+$ children in comparison to CoV2-CDA revealed distinct, time-resolved complement activity in CoV2$^-$ children, whereas CoV2$^+$ profiles showed overlap, suggesting prolonged immune dysregulation. Correlation analysis identified significant associations between SARS-CoV-2 IgG and complement proteins, with differing patterns in CoV2$^+$ and CoV2$^-$ groups over time. These findings indicate SARS-CoV-2 seropositivity could alter the complement activation trajectory in TB-diseased children, potentially contributing to persistent inflammation. Complement profiling may inform therapeutic strategies and serve as a biomarker of immune recovery in TB– SARS-CoV-2 co-infection.

Oral Talk 8 and Poster No. 81

Hyperactivated CCR5⁺ Neutrophils Drive Gastric Inflammation and Epithelial Injury in Helicobacter pylori Infection

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Helicobacter pylori infects nearly half of the global population and is a major cause of chronic gastritis, peptic ulcers, and gastric cancer. Neutrophils are among the first responders to infection and play a central role in orchestrating the inflammatory microenvironment, yet the mechanisms underlying neutrophil-mediated epithelial injury remain incompletely defined. Here, we investigated the activation and functional heterogeneity of neutrophils during H. pylori infection and examined their impact on gastric epithelial cells using a combination of in vitro and in vivo approaches. In vitro analyses using flow cytometry, immunoblotting, confocal microscopy, and biochemical assays revealed that H. pylori selectively activates a CCR5⁺ neutrophil subset exhibiting a hyperinflammatory phenotype, characterized by enhanced inflammasome activity, degranulation, and elevated expression of NLRP3, caspase-1, gasdermin D, PAD4, MPO, CD66b, and CD35. These CCR5⁺ neutrophils release extracellular vesicles (EVs) that, when applied to gastric epithelial cells in vitro, induce mitochondrial oxidative stress, loss of membrane potential, intrinsic apoptosis, and activation of NF-κB–mediated inflammatory pathways. In mouse models of H. pylori infection, accumulation of CCR5⁺ neutrophils correlated with heightened gastric inflammation and epithelial injury, confirming the in vivo relevance of the hyperinflammatory neutrophil subset. Collectively, these findings identify CCR5⁺ hyperinflammatory neutrophils as central mediators of gastric epithelial stress and provide mechanistic insight into how neutrophil activation contributes to persistent inflammation and tissue injury during H. pylori infection.

POSTER ABSTRACTS

Poster No. 1

Immune Profiling of Monocyte activation markers and Cytokine Responses in Tuberculosis Patients with and without Type 2 Diabetes mellitus

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The converging epidemics of tuberculosis (TB) and type 2 diabetes mellitus (DM) pose a major public health challenge, with diabetes conferring a threefold higher risk of active TB. This study aimed to investigate immunological mechanisms underlying this susceptibility by characterizing monocyte surface markers and antigen-specific cytokine responses in TB patients with and without diabetes. The study cohort (n=80) included adults aged above 30 years: TB patients with DM (TB-DM, n=20), TB patients without DM (TB, n=20), DM-only individuals (n=20), and healthy controls (HC, n=20). PBMCs were collected at baseline and analyzed by multi-color flow cytometry. Classical monocytes (CD14+CD16−) were examined for activation and polarization markers (CD163, PDL-1, PDL-2, CD80, CD86, CD206) and intracellular cytokines (IL-1β, IL-6, TNF-α, IL-12, IL-10) following stimulation with purified protein derivative (PPD), whole cell lysate (WCL), and TLR2/4 ligands. TB-DM monocytes showed significantly increased expression of CD163 ($p<0.01$), PDL-1 ($p<0.05$), and CD206 ($p<0.05$), with decreased CD80 ($p<0.05$) and CD86 ($p<0.01$) compared to TB-only and healthy controls. Upon stimulation with PPD and WCL, these alterations became more pronounced, while under TLR2/4 ligand stimulation, the upregulation of CD163 and CD206 was most prominent ($p<0.01$). Cytokine profiling revealed elevated IL-1β ($p<0.01$), IL-6 ($p<0.05$), and TNF-α ($p<0.01$), along with reduced IL-12 ($p<0.05$) and IL-10 ($p<0.05$) upon PPD and WCL stimulation, with the highest increases in IL-1β and TNF-α observed under TLR2/4 stimulation ($p<0.01$). Overall, TB-DM co-morbidity showed a hyper-activated and dysregulated classical monocyte profile, likely driven by diabetes. These alterations in monocyte subsets may impact both immune and metabolic responses, warranting further investigation.

Poster No. 2

Immunomodulatory Role of Nectin-4 in TAM Activation and Tumor Progression in Oral Cancer

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Oral cancer remains a major global health concern with high mortality driven by its aggressive progression and complex tumor microenvironment. Tumor-associated macrophages (TAMs) are central contributors to this microenvironment, promoting inflammation, metastasis, angiogenesis, and immune dysregulation. Nectin-4, a CAM overexpressed in several epithelial cancers, has recently been recognized for its immunomodulatory functions beyond its classical role in cell adhesion.This study investigates the role of Nectin-4 in shaping macrophage behavior in oral cancer. We demonstrate that Nectin-4 induces hyperactivation of M1 macrophages and subsequently reprograms them into TAM-like cells with strong pro-tumorigenic activity. Using combined in vitro, ex vivo, and in ovo (CAM) models, we show that Nectin-4–activated macrophages significantly enhance tumor cell migration, invasion, and tumor-associated angiogenesis, creating a tumor-supportive microenvironment. Importantly, selective inhibition of iNOS suppressed PI3K–AKT activation and substantially reduced macrophage-mediated tumor progression.As a TAA, Nectin-4 exerts a dual role in the tumor niche by promoting oncogenic immune activation and enabling tumors to exploit macrophage-driven inflammation to support their growth and spread.

Poster No. 3

Phytochemical and GC-MS/LC-MS Profiling of Illicium griffithii Fruits and Their Role in Modulating LPS-Induced Inflammation via SOCS1 and NF-κB/JAK1/STAT1 Pathways

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Endotoxin-induced inflammation remains a major contributor to the progression of various pathological conditions through the excessive activation of pro-inflammatory mediators and dysregulation of intracellular signaling pathways. The present study investigates the therapeutic potential of Illicium griffithii fruit extract against lipopolysaccharide (LPS)-induced inflammatory pathophysiology, with a focus on molecular mechanisms and chemical composition. The anti-inflammatory efficacy of the extract was evaluated through its ability to modulate key signaling hubs involved in innate immune responses. Treatment with I. griffithii extract enhanced the expression of Suppressor of Cytokine Signaling 1 (SOCS1), a crucial negative regulator of the JAK/STAT pathway, thereby attenuating downstream inflammatory cascades. The extract significantly inhibited the phosphorylation of NF-κB, JAK1, and STAT1, indicating suppression of major pro-inflammatory signaling pathways triggered during endotoxin exposure.Phytochemical screening revealed the presence of diverse bioactive constituents, including phenolics, flavonoids, terpenoids, and other secondary metabolites known for their regulatory roles in inflammation. To further characterize the chemical, comprehensive GC-MS and LC-MS analyses were performed, identifying a range of volatile and non-volatile compounds with established or potential biological activity. Correlation of the chemo-profiles with biological outcomes suggests that the synergistic action of these phytochemicals contributes to the observed anti-inflammatory effects. Overall, the findings highlight Illicium griffithii fruit extract as a promising natural therapeutic agent capable of mitigating endotoxin-driven inflammation by activating SOCS1 and inhibiting NF-κB/JAK1/STAT1 signalling pathways. This integrative approach combining mechanistic evaluation and chemical profiling provides a strong foundation for future pharmacological exploration and potential therapeutic development.

Poster No. 4

Immunomodulatory Evaluation of Novel Formulations on CD4⁺ T Cell Polarization and Activation in Rheumatoid Arthritis

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Rheumatoid arthritis (RA) is a chronic autoimmune disorder driven by persistent synovial inflammation, dysregulated cytokine signaling, and aberrant activation of CD4⁺ T cells that shape pathological immune responses. This study aims to evaluate the immunomodulatory impact of three distinct formulations on peripheral blood mononuclear cells (PBMCs) collected from healthy donors and RA patients to determine their influence on CD4⁺ T cell activation and lineage polarization. Functional immune responses were assessed using flow cytometry–based phenotyping of activation markers and polarization-associated cytokines. In addition, a Luminex multiplex cytokine assay was performed to quantify changes in inflammatory mediators under different treatment conditions, enabling a comprehensive assessment of formulation-induced shifts in cytokine expression patterns. Complementing the in vitro work, a bioinformatic analysis of publicly available RA transcriptomic datasets was conducted to identify hub genes, enriched biological pathways, and immune cell infiltration signatures characteristic of active RA. Network and pathway analyses revealed strong enrichment of TNF-α signaling, IL-17 signaling, NF-κB activation, cytokine–cytokine receptor interactions, and the rheumatoid arthritis pathway, highlighting central inflammatory axes relevant to RA pathogenesis. Integration of these computational insights with experimental findings provides mechanistic context for how the formulations may modulate CD4⁺ T cell–driven inflammation. Preliminary observations suggest differential effects of the three formulations on T cell activation thresholds and cytokine modulation, underscoring their potential as immunoregulatory candidates for RA intervention.

Poster No. 5

Reprogramming tumor associated macrophages towards a tumoricidal M1-like phenotype with Genistein loaded mannose coated GMO-MNPs (Gen@Man-GMO-MNPs) for cancer immunotherapy

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Tumor-associated macrophages (TAMs) are the most abundant immunosuppressive cells in the tumor microenvironment (TME) and predominately exhibit pro-tumorigenic M2-like phenotype, involved in tumor progression, drug resistance, metastasis, and poor prognosis. Thus, targeting TAMs has the potential to improve cancer immunotherapy. Consequently, various strategies have been developed to target these cells amongst which repolarization of TAMs towards tumoricidal M1-like phenotype is the most promising option in the field of cancer immunotherapy. Recent reports suggest that iron oxide nanoparticles and several phytochemicals have an important role in TAMs polarization to improve immunosuppressive TME. In this aspect, we investigated the effect of GMO coated superparamagnetic iron oxide nanoparticles (GMO-MNPs) and Phytochemicals (β-Carotene, Genistein, Emodin, Sophoridine) on M2-macrophages polarizing ability. From the preliminary results, we found that Genistein (an isoflavone derived from soybean products) shows better M2 macrophage polarization as compare to other phytochemicals. In this work, we developed a Genistein loaded mannose coated GMO-MNPs (Gen@Man-GMO-MNPs) for specific targeting and polarizing M2-macrophages. We tested these Gen@Man-GMO-MNPs in 4T1 tumor bearing orthotopic mice model and observed that these functionalized nanoparticles mainly accumulated in the tumor site and enhanced the M1 macrophage activity leading to infiltration of cytotoxic T cells in TME, eventually leads to tumor regression. We further elucidated, the polarization effect shown by our magnetic nanoparticles was mainly due to the modulation of IRF5, NF-kB and STAT1 signalling pathways. Over all, these findings suggest that Gen@Man-GMO-MNPs can be a potential candidate for modulating immunosuppressive TME to improve cancer immunotherapy.

Poster No. 6

Desert Medicine Meets Modern Resistance: Camel Milk PGRP-S as a Macromolecular Weapon Against MDR *Staphylococcus aureus*

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The escalating prevalence of multidrug-resistant (MDR) *Staphylococcus aureus* necessitates innovative therapeutic strategies beyond conventional antibiotic frameworks. Camel milk, a traditional bioactive source, contains Peptidoglycan Recognition Protein-S (PGRP-S), an innate immune effector with bactericidal potential. This investigation evaluates the antimicrobial efficacy and immunomodulatory properties of purified camel milk-derived PGRP-S (C-PGRP-S) against MDR S. aureus through multifaceted molecular and cellular approaches. Purified C-PGRP-S was subjected to microbroth dilution assays for minimal inhibitory concentration (MIC) determination, time-kill kinetics for bactericidal assessment, scanning electron microscopy (SEM) for ultrastructural analysis, and intracellular immunoassays measuring reactive oxygen species (ROS) and pro-inflammatory cytokine production in M1 macrophages infected with MDR S. aureus. C-PGRP-S completely inhibited bacterial growth at 40 µg/mL in MIC assays with rapid dose-dependent bactericidal action in time-kill experiments. SEM analysis revealed pronounced membrane perforation and cellular envelope disruption. Immunologically, C-PGRP-S restored ROS generation in activated macrophages while simultaneously attenuating excessive pro-inflammatory cytokine secretion, demonstrating immune-balancing activity. Crystallographic structural data (PDB ID: 3NW3) elucidates the molecular interaction between C-PGRP-S and bacterial peptidoglycan, supporting a dual mechanism encompassing direct bacterial lysis via peptidoglycan engagement and concurrent immunomodulation through innate lymphocyte reprogramming. C-PGRP-S emerges as a promising macromolecular therapeutic candidate with dual-action potential, positioning camel milk-derived proteins as natural alternatives or adjuvant strategies for combating drug-resistant staphylococcal infections.

Poster No. 7

Cisplatin-induced cachexia rescued by Teriflunomide: A pilot study in BALB/c mice

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Chemotherapy-induced cachexia remains a major clinical challenge, contributing to metabolic imbalance, and reduced treatment tolerance. The present study aims to treat the cisplatin-induced mild-to-moderate cachexia with a repurposed therapeutic, Teriflunomide, by targeting TNF-α and IL-6. Adult female BALB/c mice (12-14 weeks) were challenged with intraperitoneal cisplatin (3 mg/kg) on alternate days for five doses. Teriflunomide 10 mg/kg (p.o.) and 5 mg/kg (p.o.) was administered daily for 15 days after 3rd dose of Cisplatin. Behaviour, body weight, food-water intake, serum creatinine and mortality were monitored on days 0, 5, 10, 15, and 20. Gastrocnemius muscles, and spleen were harvested for weight, gross morphology and for histopathology. Muscle tissue homogenates were analysed for TNF-α, IL-6, and NF-κB using ELISA. Transcriptional expression of MyoD, TRIM63 and FOXO3 were evaluated by qPCR. The model was able to induce significant weight changes in the animals without cancer co-infection, indicating to a viable screening technique for anti-cachectic drugs. Teriflunomide (10mg/kg) showed slight improvement in behaviour, weight, and muscle degradation on Day 20, significantly improved creatinine levels, reduced TNF-α ($p=0.002$), IL-6 ($p=0.004$) and upregulated MyoD (2-fold) with no significant changes in TRIM63. FOXO3 was upregulated (3-fold) in spleen and downregulated (0.5-fold) in muscles, consistent with its immune-regulatory and catabolic roles respectively. NF-κB expression remained mildly elevated but was not statistically significant. The low dose Teriflunomide failed to show improvements to the diseased state with one mortality event. Thus, the 10mg/kg dose of Teriflunomide exhibited promising results in halting cisplatin-induced cachexia however long-term studies are warranted.

Poster No. 8

Evaluating the role of the mTOR pathway and protein translation during *Burkholderia* species infection.

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Mammalian target of rapamycin (mTOR) is a central regulatory mechanism controlling many essential pathways in our body. It functions through mTORC1 and mTORC2 complexes. mTORC1 regulates protein translation through the phosphorylation of 4E- binding proteins (4E-BPs). The 4E-BPs bind to the eukaryotic initiation factor 4E and further inhibit the formation of the initiation complex of protein translation. Among many functions of mTOR, its regulation of immune and inflammatory modulators is of interest during bacterial infections. *Burkholderia* species cause various infections and may cause a detrimental outcome for the patients. Burkholderia cepacia causes severe infections in cystic fibrosis (CF) patients and other immunocompromised individuals. It often results in a drastic decline in the patient's condition by causing pneumonia, respiratory distress, and sepsis. There is a research gap in understanding the role of the mTOR pathway and protein translation during the infection of Burkholderia cepacia. We observed significant dysregulation in the mTOR pathway proteins. So, understanding the link between the host mTOR pathway and *Burkholderia cepacia* pathophysiology may uncover vital regulatory proteins for biomarker or drug target identification.

Poster No. 9

Genomic framework and Host Immune Cross-Talk underpinning human gut adaptation of commensals

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The human gut microbiota is a complex ecosystem that plays a crucial role in maintaining host health. Despite extensive information about gut microbiota composition, much less is known about the ecological and immunological competitiveness determining a microbe's fitness within the gut environment. This knowledge would enrich our understanding of the host selection process and gut microbiota establishment, which is essential for the successful implementation of personalized microbiota therapies.

A comprehensive study was designed to assess in vitro ecological fitness (physiological and genomic) and in vivo immunological fitness (expression of pro-inflammatory, anti-inflammatory genes, and cellular integrity genes) of gut isolates. Gut isolates exhibited broad stress tolerance, antioxidant activity, diverse substrate utilization, resistance to bile and lysozyme, and facultative anaerobic growth, while remaining non-pathogenic with no cytotoxic or haemolytic effects. Genomic and immunological analyses confirmed their potential for gut colonization, stress resilience, and balanced modulation of pro- and anti-inflammatory cytokines, highlighting their suitability as safe and functionally fit gut commensals.

Overall, gut isolates demonstrated adaptability in a dynamic gut environment, biosafety, and the ability to balance the host immune system, highlighting key fitness parameters for human gut colonization. Future studies exploring the role of gut microbes under dietary, disease, and microbial interaction contexts could pave the way for personalized microbial therapeutics for human health.

Poster No. 10

Experimental and In Silico Analysis of miR-145-5p identifies Key Regulators of the TGF-β/Smad Pathway in Systemic Lupus Erythematosus

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Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by chronic inflammation and organ damage. MicroRNAs have emerged as key post-transcriptional regulators. Among them, miR-145-5p has been implicated in fibrotic and epithelial–mesenchymal transition (EMT)–related pathways, but its role in SLE remains unclear. The objective of the study is to evaluate the expression of miR-145-5p in SLE patients and to explore its association with fibrosis-related genes and signaling pathways. Plasma miR-145-5p expression was quantified in SLE patients and healthy controls using RTqPCR. Concentration of CTGF, alpha-SMA, and E-cadherin estimated using ELISA. In-silico target prediction was performed using TargetScan, miRDB, and miRTarBase. Functional enrichment (GO and KEGG) analyses were used to identify biological processes and pathways associated with miR-145-5p target genes. We found miR-145-5p expression to be significantly altered between SLE patients and controls ($p = 0.0008$). ROC analysis indicated a moderate diagnostic accuracy. Correlation analysis revealed a significant positive association between miR-145-5p and CTGF and between miR-145-5p and α-SMA, while E-cadherin showed no significant relationship. Target prediction identified 72 common genes in the three databases. GO and KEGG enrichment analyses indicated involvement of miR-145-5p targets in TGF-β signaling, extracellular matrix organization, and cell adhesion processes. miR-145-5p is differentially expressed in SLE and correlates positively with fibrosis-related genes such as CTGF and α-SMA, suggesting its potential role in fibrogenic or EMT-associated mechanisms. These findings highlight miR-145-5p as a potential biomarker and regulatory molecule in SLE-related tissue remodeling.

Poster No. 11

Immunometabolic Profiling of Circulating Follicular T Helper Cells in Dengue

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Dengue is a viral infection caused by the dengue virus (DENV), which belongs to the family Flaviviridae and the genus Flavivirus, and has four serotypes: DENV 1-4. It is transmitted by the female Aedes aegypti mosquito. According to the WHO, dengue is estimated to cause 100-400 million infections annually, with the disease now spreading to previously unaffected areas, including the US and Western Europe. Dengue manifests with or without warning signs and can progress to severe dengue. Follicular T helper cells (Tfh) play a crucial role in the humoral immune response against dengue virus by facilitating the maturation of B cells into plasmablasts, which produce antibodies essential for viral clearance. We hypothesise that metabolic changes in immune cells may determine their response to DENV infection and, consequently, progression to severe dengue. The objective of this study is to identify the differences in metabolic pathway dependencies of Tfh cells between dengue patients and controls. Using a flow cytometry-based metabolic analysis pathway (Met-Flow), we will examine the intracellular expression of enzymes involved in key pathways, including glycolysis (GLUT1 and HK1), fatty acid metabolism (ACAC and CPT1A), oxidative phosphorylation (IDH2 and ATP5A), and redox pathways (PRDX2 and IDH2). With the results obtained, we will decipher the changes that occur in the metabolic signatures of Tfh cells in dengue patients compared to controls, and how these changes may lead to the manifestation of severe dengue. This pilot study will enable us to identify the potential immuno-metabolic adaptations that may occur in dengue patients and influence disease outcomes.

Poster No. 12

Exploring Immunomodulatory and Protective Roles of Vector Salivary Proteins (VSPs) in Experimental Lymphatic Filariasis

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Lymphatic filariasis (LF) is a debilitating vector-borne disease that causes chronic disability and currently lacks effective adulticidal drugs or licensed vaccines, underscoring the need for novel intervention strategies. Because early infection is asymptomatic and chronic pathology is irreversible, transmission-blocking vaccines represent an ideal approach. Vector salivary proteins (VSPs) are known to modulate host immunity and have recently emerged as promising vaccine candidates and biomarkers. In this study, we evaluated the vaccine potential of Aedes aegypti VSPs against LF. VSPs were isolated and characterized for diversity and structural integrity using proteomic and molecular analyses. Co-infection of hosts with L3 larvae and VSPs, mimicking natural transmission, did not provide protection. However, pre-immunization with VSPs yielded significant reductions in parasite burden: ~69% reduction of L3 in BALB/c mice, and in Mastomys, ~76% and ~57% protection against microfilariae and adult worms, respectively. These results prompted investigation of VSP-mediated immunomodulation. Sera from infected animals showed poor cross-reactivity with VSPs, and antibody-dependent cellular cytotoxicity assays indicated minimal humoral involvement. Conversely, ex-vivo analyses of splenocytes and peritoneal macrophages revealed distinct cellular responses. VSPs enhanced lymphocyte proliferation, promoted macrophage-specific phagocytosis, and increased ROS/RNS production. A mixed Th1/Th2 cytokine profile was also observed, indicating an inflammatory yet balanced cellular response. Overall, these findings demonstrate that VSPs elicit robust cellular immunity that correlates with significant protection against filarial infection. This work highlights VSPs as strong and previously underexplored vaccine candidates for lymphatic filariasis.

Poster No. 13

Integration of TLR signalling in Mouse Type I conventional dendritic cells is underpinned by specific gene regulatory networks

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Dendritic cells (DCs) are considered as professional antigen presenting cells which function as a bridge between innate immunity and adaptive immunity. Type 1 conventional dendritic cells (cDC1) are the most efficient cross-presenting cells that induce protective cytotoxic T cell response. During an infection, several molecules i.e. pathogen-associated molecular or damage-associated patterns might be recognized and used to regulate the activated phenotype of the cells. The underlying mechanisms behind functional signal integration in such a microenvironment have not been cohesively addressed. Here, we use single-cell RNA sequencing to explore how individual cDC1s respond when costimulated with CpG, a TLR9 ligand and Poly(I:C), a TLR3 ligand. These costimulated cDC1s display a distinct global transcriptional program. Our data reveal synergistic effects and antagonistic interferences between host responses to the two ligands. Computational approaches that considered co-expression of transcription factors (TFs) and downstream genes and motif enrichment allowed prediction of stimulus-specific TFs and transition of gene regulatory networks during monostimulation to combined stimulation. We use single-cell ATAC sequencing to show that TLR3 and TLR9 signals induce synergy and antagonism in individual cDC1 cells through reprogramming of the chromatin accessibility landscape. Our findings highlight a complex crosstalk between TLR3 and TLR9 signals that is controlled by a balance between a plethora of TFs, specifically Batf and Mxd1, which suggest a mechanism for molecular interaction points during coinfection.

Poster No. 14

Monocyte adhesion and Atherosclerosis during aging

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Atherosclerosis is chronic inflammation of the arteries and is characterised by the formation of aortic plaques as a result of immune cell accumulation, fatty streak formation, inflammation, vascular calcification, and thrombosis. Mechanistically, strong binding of monocytes to aortic intimal endothelial cells, followed by their sub-intimal infiltration and differentiation into foamy macrophages, initiates plaque formation. We aim to investigate the mechanism underlying monocyte adhesion to the endothelial cells during aging that may lead to an increased risk of atherosclerosis. We hypothesise that aging leads to increased signalling via CD74 upon binding to its receptor, Macrophage Migration Inhibitory Factor (MIF), upregulating the expression of monocyte integrins, including VLA-4, LFA-1, and CD11b, which in turn causes stronger monocyte binding to endothelial Vascular Cell Adhesion Molecule-1 (VCAM-1) and Intercellular Adhesion Molecule-1 (ICAM-1). Our data from qRT-PCR and flow cytometry analysis show upregulated expression of Cd74 in THP-1 cells and human blood monocytes following stimulation with oxidized low-density lipoprotein (Ox-LDL). Stimulation of THP1 cells with ox-LDL plus MIF further resulted in increased expression of integrins VLA-4, LFA-1, and CD11b, synergistically implying the monocytes' ability to form stronger adhesion to endothelial cells in the presence of ox-LDL plus MIF. Lastly, we found that Ox-LDL-induced expression of Cd74 in human whole blood cells is higher with increasing age. We are currently studying the mechanism by which CD74 is upregulated by ox-LDL and its impact on monocyte adhesion to vascular endothelial cells during aging

Poster No. 15

Preeclampsia Prediction in Pregnancy by Routine Biochemistry and Immuno-Regulator Profiling

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Preeclampsia continues to be a leading cause of maternal and fetal morbidity, and robust early prediction remains limited in many clinical settings where advanced biomarker assays are not readily available. This study investigated whether a focused panel combining routine biochemical tests with immune-regulator profiling could enhance the stratification of preeclampsia risk using machine-learning methods. Maternal laboratory parameters included Urine Protein (Ur P), Alanine Aminotransferase (ALT), Total protein (TP), interleukin-6 (IL-6), and absolute count of regulatory T-cell marker CD4+FOXP3+ (FOXP3). Random Forest classifiers were trained using 0.632+ bootstrap resampling (B = 1000), and evaluated on the test subset through nested five-fold cross-validation with embedded bootstrap. A composite performance metric was calculated as the fourth-root of F1, Youden Index, Jaccard Index, and absolute Matthews Correlation Coefficient. Three predefined models were compared, with Model B (All Parameters) demonstrating the strongest discrimination (Fourth-root mean ≈ 0.84, SD ≈ 0.06; Sensitivity ≈ 0.88; Specificity ≈ 0.82; F1 ≈ 0.89; MCC ≈ 0.71; Youden Index ≈ 0.70). These findings underscore the importance of endothelial dysfunction, proteinuric renal involvement, and immune tolerance dysregulation in the pathogenesis of preeclampsia. This integrated panel presents a promising, clinically feasible approach for risk prediction that is independent of complex molecular platforms. Further prospective validation may support its use in decision-support tools for maternal health programs.

Poster No. 16

Comparative In Silico Analysis of Antimicrobial Resistance Genes and Virulence Factors in ESKAPE Pathogens

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The rising prevalence of hospital-acquired infections caused by multidrug-resistant (MDR) bacteria has emerged as a serious global health threat. In the post-COVID era, antimicrobial resistance (AMR) is increasingly regarded as a potential next pandemic, particularly among the ESKAPE pathogens (Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa, and Enterobacter spp.), which collectively account for most nosocomial infections worldwide. Resistance to β-lactams, quinolones, and aminoglycosides has become widespread in these organisms, prompting a re-examination of the genomic determinants underlying resistance and virulence.

In this study, we performed a large-scale in silico comparative analysis to explore the relationship between AMR genes and virulence factors (VFs) across 7,762 protein FASTA sequences from GenBank, including 671 A. baumannii, 773 Enterobacter spp., 294 E. faecium, 2,809 K. pneumoniae, 1,088 P. aeruginosa, and 2,127 S. aureus genomes. AMR genes were identified using AMRFinderPlus, and VFs were detected by BLASTp searches against the Virulence Factor Database (VFDB) with ≥90% identity and coverage thresholds. Correlation analyses revealed species-specific trends: S. aureus showed the strongest positive correlation between AMR and VF abundance (Spearman's r = 0.53), followed by E. faecium and A. baumannii (r = 0.34 each), while P. aeruginosa displayed a weak negative association (r = –0.10).

These findings suggest that in most ESKAPE pathogens, increased virulence potential parallels a higher AMR burden. Identifying shared AMR–VF modules provide insights into their co-evolution and offers a foundation for designing broad-spectrum therapeutic strategies targeting both virulence and resistance mechanisms.

Poster No. 17

Screening for Inborn Errors of Immunity (IEI) in a tertiary healthcare centre of Eastern India: Our experience and challenges ahead

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Inborn Errors of Immunity (IEI) are a group of disorders which affects the ability of the body to fight against infection. These disorders include defects in T and B cells, defects in phagocytosis, complement system, defects in cytokines or cytokine signalling pathways, autoimmunity and autoinflammation. Because of inadequate medical awareness, a significant number of patients with IEI goes unrecognized or are diagnosed late. There is a severe dearth of data in terms of incidence or prevalence of IEI in the eastern part of India. To determine the prevalence of the selected IEI in our population and to evaluate the causes and pattern of IEI along with creating awareness amongst parents and health care workers. The study was a prospective, observational hospital-based study. All children up to 18 years of age satisfying the inclusion criteria by Jeffrey Modell Foundation are enrolled. Serum immunoglobulins and complements were estimated using autoanalyzer. The analysis of T-B-NK-cell immunophenotypes and DHR assay in EDTA blood were carried out by flow cytometry. We screened 140 suspected cases of IEI and the preliminary data analysis of the immunophenotyping revealed 2 cases of SCID, 4 cases of CVID, 4 cases of CGD, 2 cases each of XLA and ALPS and 1 of C1q deficiency. We got the prevalence of IEIs from this region as 10.7% after completion of the study along with the dominant pattern of disease as immunoglobulin deficiency in the region. We were also able to raise the awareness about IEIs amongst parents and health care workers while carrying out the study but strengthening the healthcare delivery system for these rare diseases remains a challenge.

Poster No. 18

Role of sex hormones in CD4^{+} T-Cell mediated RA pathology

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The role of T cells has been well documented in the pathogenesis and exacerbation of autoimmune diseases. Interestingly, majority of these autoimmune disorders predominantly affect women. This sexual dimorphism is indicative of the possible role of sex hormones in contributing to the disease pathogenesis. Nevertheless, the specific role of sex hormones, such as Estrogen and Progesterone, in immune-endocrine interactions in RA has not been fully elucidated and remains unclear. In this study, we focus on the critical role of sex hormones in contributing towards the aberrant status of RA T helper subsets, specifically with respect to inflammation and osteoclastogenic potential. Towards this, we evinced a higher expression of ERα and PR on RA Th1 and Th17 subsets, thereby suggesting a link between sex hormones and T cell signalling. To further identify their role in modulating T helper responses, we assessed the inflammatory response of RA CD4+ T cells stimulated under varying concentration(s) of hormone. Interestingly, here our findings revealed two specific patient cohorts; the responders and the non-responders, strongly suggesting towards the differential contribution of sex hormones in modulating inflammatory status of T cells. Along with inflammation, our studies delineated the critical role of these hormones in modulating RANKL expression; a key protein mediating Osteoclastogenesis. Altogether, our results indicate towards diverse processes being regulated by female sex hormones and understand its pivotal significance with respect to modulating TCR responses. Further dissection of TCR signalling pathways with respect to sex hormone stimulation may provide promising targets for therapeutic implications.

Poster No. 19

One Health- Integrated Vaccine Design for Tuberculosis in Humans and Animals

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Tuberculosis (TB) remains a critical One-Health challenge, impacting both human populations and livestock health. In India, the high prevalence of bovine TB within cattle herds results in substantial economic burden through reduced productivity and associated management costs. The increasing detection of zoonotic TB driven by the emerging pathogen M. orygis underscores the need for integrated control strategies beyond conventional surveillance frameworks. The limited efficacy and societal constraints of test-and-cull programs, coupled with the absence of licensed, protective vaccines for bovine TB, highlight an urgent demand for a cross-species, next-generation vaccine platform. Using the conserved genomic architecture and antigenic overlap among M. tuberculosis, M. bovis, and M. orygis, we utilized integrative bioinformatics and immunoinformatics pipelines to map epitopes immunodominant epitopes absent in BCG but conserved across these pathogens as well as experimentally validated common immunodominant antigens. The selected epitope were designed into a chimeric multiepitope vaccine (MEV) construct, rationally optimized for antigenicity, broad population coverage, minimal host homology, and absence of predicted toxicity. Notably, computational immunogenicity analyses indicated potent humoral and cellular responses, characterized by a Th1-skewed immune profile and heightened IFN-γ production. The engineered MEV immunogen was further introduced and overexpressed within M. bovis BCG strain, generating a recombinant BCG based vaccine candidate programmed to augment and prolong BCG induced immunity. This vaccine development strategy advances the development of a unified TB vaccine with potential to curb zoonotic transmission, reduce disease burden, and strengthen One Health-based control efforts.

Poster No. 20

Dissecting the molecular cascade linking AGE exposure to enhanced lipid droplet formation

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Advanced glycation End products or AGEs are chemical aggregates formed when an excess amount of dietary sugars reacts non-enzymatically to proteins, lipids and nucleic acids. AGEs can crosslink with each other, which leads to a plethora of harmful effects. One of such harmful responses is the accumulation of Lipids in the form of lipid droplets in various vital organs, eventually disrupting the natural cellular metabolism, leading to diseases like obesity and T2D. The objective of the study is to identify the cellular pathways taken by the cell for excess lipid droplet storage, i.e. to study the impaired lipolysis and lipophagy pathways induced by AGEs. According to our previous knowledge, lipophagy is completely defective in AGE-treated cells; hence, this study mainly focuses on the impaired lipolysis pathway. The transcriptional regulation of ATGL (cytoplasmic triacylglycerol hydrolase) and its inhibitor protein G0S2 might play a crucial role in understanding this cellular pathway. Using a combination of bioinformatics, molecular biology and biochemical approaches, the regulatory signals of the above-mentioned proteins are identified and analysed. From the initial studies, it was suspected that transcription factors like NFATC1 and HNF4α could be the regulatory proteins involved in this pathway. Collectively, this study highlights the cellular pathways responsible for lipid droplet accumulation on AGE exposure, which will further give us ideas to work on the therapeutic aspects of diseases like obesity and T2D.

Poster No. 21

Unravelling the Immunomodulatory Functions of *Leptospira* Outer Membrane Vesicles

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Leptospirosis, a bacterial zoonotic disease caused by pathogenic *Leptospir*a species, poses a significant public health burden worldwide. Understanding Leptospira host interaction is crucial for identifying critical virulence factors that can be exploited for developing improved Vaccines and Diagnostics. Outer membrane vesicles (OMVs) are nano-sized structures shed by various Gram-negative bacteria, including *Leptospira*, which have garnered increasing attention due to their diverse roles in intercellular communication and host-pathogen interactions. These vesicles are enriched with virulence factors, lipopolysaccharides (LPS), proteins, and nucleic acids and have been implicated in modulating the host immune response. We purified OMVs from pathogenic Leptospira cultured in vitro or under pathogenic conditions (Physiological temperature and Osmolarity) and assessed its immunomodulatory effect on different host macrophages. Our initial result shows that Leptospira OMVs induced proinflammatory effect on mouse macrophages by inducing IL-6 and TNF-a. It also modulated the expression of costimulatory molecules (CD80, CD86) and maturation marker (MHCII) on these macrophages. In conclusion our initial results indicate that Leptospira OMVs can activate the macrophages and may play a crucial role in modulating their function which needs to be explored. Further research into the composition, biogenesis, and immunomodulatory properties of Leptospira OMVs will provide valuable insights into host-pathogen interactions and may lead to the development of OMV-based vaccines or immunotherapies against leptospirosis.

Poster No. 22

Influence of HLA-Bw4 Epitopes with B Leader (-21M/T) Dimorphism on Graft versus Host Disease: A Population Specific Algorithm for Hematopoietic Stem Cell Transplantation

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Human Leukocyte Antigen (HLA) haploidentical hematopoietic stem cell transplants (HSCT) across the globe has elevated the risk of Graft versus Host Disease (GvHD) post-transplant (∼40% cases). Exon 1 of HLA class I gene encodes for a leader peptide sequence largely presented by HLA-E mediating natural killer (NK) cell allo-responses. HLA-B leader encodes for methionine (M) or Threonine (T) at -21 position, influencing post-transplant outcomes particularly GvHD. Similarly, HLA-Bw4 epitopes interact with KIR3DL1 to regulate NK cell mediated cytotoxicity. Evaluation of leader peptide variant and Bw epitopes for any clinical coherence with GvHD could assist selection of most permissible donor among multiple haploidentical donors. Thus, a population-specific algorithm can predict GvHD risk. High resolution HLA-B genotyping data was analyzed in 178 individuals (N=89 HSCT patient-donor pairs) for the leader peptide (-21) variant and Bw4/Bw6 epitopes, using HLA-B leader assessment Tool (BLEAT), and KIR ligand calculator respectively. Higher occurrence of -21T leader allele (~85%) compared to -21M was observed in both HLA-Identical and haploidentical HSCT patients. In HLA-Identical HSCT patients, -21 T leader variant and Bw4 epitopes were significantly over represented (26/48=54.2%) in GvHD (p=0.044) while -21T with Bw6 (35.4%) was significantly lower as compared to other haplotypes (64.6%, p=0.041). Though, lower occurrence of HLA-B leader -21M variant in patient-donor pairs could affect clinical utility of BLEAT algorithm here. Our preliminary data seem translationally supportive towards developing population specific algorithm based on evaluating this dimorphism along with Bw4/Bw6 for GvHD prediction and selection of permissible donors for HSCT here.

Poster No. 23

Neuropilin-1 blockade enhances immunotherapy against melanoma through reduction of peripheral Treg-mediated suppression

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Melanoma is one of the most aggressive forms of skin cancer, with a high mutation rate, and ranks as the fifth most common cancer among Caucasians. Over 40% of patients experience immune-related adverse effects, which limit therapeutic efficacy. Neuropilin-1 (NRP1) has emerged as a critical target in cancer therapy because of its overexpression in multiple malignancies, where it promotes regulatory T cell (Treg) function and tumor progression, often correlating with poor prognosis. This study explored the impact of NRP1 inhibition on B16-F10 melanoma and its role in modulating Treg-mediated immune responses. Inhibiting NRP1 selectively induced apoptosis in melanoma cells without affecting the normal control cells. Moreover, NRP1 inhibition reversed the immunosuppressive effects of B16-F10-conditioned media on splenic T cells. In addition, in tumor-bearing mice, NRP1 inhibition reduced the expression of Tregs and enhanced Th1-associated marker expression in peripheral Tregs. This also led to improved overall survival of mice. Overall, these findings demonstrate that targeting NRP1 can mitigate Treg-mediated immunosuppression, highlighting its potential as an adjunct to immunotherapy.

Poster No. 24

High-Throughput Screening of FDA-Approved Drugs Identifies Potential Inhibitors of TGF-β–Induced Activation of Cancer-Associated Fibroblasts in Pancreatic Cancer

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Transforming growth factor-β (TGF-β) is a central regulator of myofibroblast activation, a key process that drives fibrosis across multiple pathological conditions, including pulmonary fibrosis and cancer-associated fibrosis. Extensive clinical and experimental evidence highlights the pivotal role of TGF-β in promoting fibroblast-to-myofibroblast transition, extracellular matrix accumulation, and tissue remodeling. Consequently, there is considerable interest in identifying novel inhibitors or repurposing existing FDA-approved drugs that can effectively block TGF-β–mediated fibroblast activation. To identify FDA-approved drugs capable of inhibiting TGF-β–induced myofibroblast activation using high-throughput screening, and to validate top candidates in patient-derived cancer-associated fibroblasts through molecular and phenotypic assays. Initially, a high-throughput screening of FDA-approved drugs was performed in TGF-β–induced human lung fibroblast cell lines. Top candidate drugs were subsequently evaluated against TGF-β–induced activation of CAFs isolated from human PDAC tissues. The efficacy of one selected drug was further evaluated on CAFs' fibrogenic properties in a heterotypic co-culture model of cancer cells and CAFs isolated from PDAC patients. Gene expression analysis was conducted to under the mechanisms associated with the selected drug's anti-fibrotic effect. High-throughput screening in TGF-β–induced human lung fibroblasts identified several FDA-approved drugs that significantly reduced myofibroblastic features, including decreased α-SMA expression and altered fibroblast morphology. When tested in patient-derived CAFs, the top candidate compounds similarly suppressed TGF-β–driven activation. Molecular analyses further confirmed downregulation of key fibrotic genes, indicating a consistent anti-myofibrotic effect across both fibroblast models.

Poster No. 25

CODOME-IMMUNOME Entropy Equalization by Attuning Mononucleotide Frequencies Consistent with Chargaff's Second Parity Rule: towards a Robust, Exact HCV-Vaccine Design against HepatoCellular Carcinoma–Liver Cancer

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The absence of an effective prophylactic vaccine against hepatitis C virus (HCV) remains a major barrier in preventing hepatocellular carcinoma (HCC), underscoring the need for fundamentally new principles that unify genome design and immune recognition. Here, we introduce a novel CODOME–IMMUNOME framework grounded in information theory, proposing that coding DNA and the T-cell–visible immunopeptidome are evolutionarily tuned toward entropy equalization through subtle attunement of mononucleotide frequencies while strictly obeying Chargaff's second parity rule. The objective is to exploit this intrinsic genome–immune coupling to rationally design mRNA/DNA vaccine constructs optimized for stable, immunogenic epitope presentation against HCV. We formulate a HyperProteoGenomic mapping that equates nucleotide and proteomic combinatorial capacities, demonstrate that minimal perturbations of base composition can elevate nucleotide entropy without violating strand parity, and computationally propagate these adjustments through codon usage, amino-acid distributions, and nonameric T-cell epitope landscapes relevant to HCV. The results reveal that entropy-balanced codomes preferentially stabilize conserved HCV 9-mer epitopes, reduce background immunological noise, and enhance theoretical compatibility with regulatory T-cell–mediated tolerance, aligning with contemporary insights from immune tolerance biology. We conclude that CODOME–IMMUNOME entropy equalization provides a principled, mechanistic foundation for next-generation nucleic-acid vaccine design, with far-reaching implications for HCV-driven liver cancer prevention and a broader conceptual shift in how genomes are engineered to interface seamlessly with the immune system, for Rational HCV-Vaccine design.

Poster No. 26

Macrophage Polarization Dynamics driving *Leptospira* Pathogenesis

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Leptospira is a gram negative spirochete bacteria causing the zoonotic disease Leptospirosis which has a broad spectrum of clinical outcomes ranging from mild illness to severe hyper-inflammatory disease, yet the early immune mechanisms that shape this variability remain unclear. Because macrophages play a central role in sensing and responding to invading pathogens, understanding how Leptospira influences macrophage polarization may provide important insights into disease progression and host susceptibility. In this study we are focusing on examining polarization dynamics in murine immortalized bone marrow–derived macrophages (iBMDM) at different time post infection. Infection consistently elicited a dominant M1 phenotype, characterized by enhanced expression of pro-inflammatory cytokines and M1-associated genes, and minimal activation of M2-associated genes or cytokines. To explore whether polarization status affects bacterium–host interaction, internalization assays using high-content imaging and flow cytometry are being carried out, indicating that Leptospira can enter into the macrophages across polarization states but M1 macrophages exhibiting a higher internalization rate compared to M0 and M2 although the functional consequences of this uptake still require further investigation. Parallel proteomic analyses of polarized macrophages are in progress to identify molecular pathways involved in Leptospira-driven immune modulation and future studies will extend these findings to in vivo models. Comparative analyses of human and bovine macrophages are also planned to understand species-specific responses. Collectively, this ongoing work aims to elucidate how Leptospira shapes macrophage polarization and how these early interactions might influence bacterial fate and the development of hyper-inflammation potentially revealing targets for therapeutic intervention.

Poster No. 27

A systems biology approach to uncover regulatory control of surface adhesion genes in *Candida glabrata*

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Candida glabrata is an emerging opportunistic fungal pathogen and the second leading cause of candidemia worldwide after Candida albicans. A critical determinant of C. *glabrata* virulence is its ability to adhere to host tissues and form biofilms, processes largely mediated by surface adhesion proteins (adhesins). C. *glabrata* genome encodes large adhesin gene families, including the EPA, AWP, PWP, and AED genes, many of which are positioned within chromosomal subtelomeric regions. In this study, we employ systems biology approaches to investigate the regulatory mechanisms controlling surface adhesion gene expression, with a particular focus on subtelomeric loci. Our analyses identified multiple chromatin remodeling complexes that modulate the transcription of subtelomeric adhesin genes. Functional perturbation of these complexes leads to pronounced changes in adhesin expression and, consequently, alters the surface adhesion properties of the fungus.

Poster No. 28

Integrated Immunometabolic Reprogramming in Rheumatoid Arthritis Reveals Key Metabolic Drivers of Inflammation

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Rheumatoid arthritis (RA) is a chronic autoimmune disorder characterized by persistent synovial inflammation, progressive joint destruction, and systemic involvement. Emerging evidence highlights dysregulated immunometabolism as a key driver of aberrant immune activation in RA, with metabolic shifts influencing immune cell differentiation, effector function, and survival. Understanding these cell-specific metabolic disturbances may reveal novel therapeutic targets and biomarkers for precision medicine. In this study, 58 RA patients and 73 healthy controls were enrolled to investigate immunometabolic alterations. Fresh whole blood samples were analysed using a full spectral flow cytometry platform to characterize metabolic pathway utilization across immune subsets, while serum cytokine and interferon levels were quantified using the Meso Scale Discovery (MSD) platform. Four major metabolic pathways—glycolysis, mitochondrial oxidative phosphorylation (OXPHOS), fatty acid oxidation, and glutaminolysis—were assessed. RA patients exhibited substantial immunometabolic reprogramming across dendritic cells, T-effector cells, and other inflammatory populations, which displayed increased reliance primarily on glycolytic and mitochondrial OXPHOS pathways, reflecting sustained activation. Immunophenotyping further demonstrated an elevated proportion of inflammatory immune cell populations in RA compared to healthy controls. Concordantly, cytokine profiling revealed significantly higher levels of proinflammatory cytokines and interferons, indicating a heightened inflammatory milieu and reinforcing the link between metabolic activation and immune dysregulation. Overall, the study identifies a coordinated enhancement of glycolytic and mitochondrial pathways in key pathogenic immune subsets alongside an amplified proinflammatory cytokine signature. These findings underscore the therapeutic potential of targeting metabolic checkpoints and support the development of metabolism-based biomarkers for disease stratification and personalized therapeutic strategies in RA.

Poster No. 29

Antimicrobial Peptide KU04AMP01 Overcomes Multidrug Resistance and Induces T-Cell Proliferation *in-vitro*

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The rapid emergence of multidrug-resistant (MDR) pathogens, particularly within the ESKAPE group, demands therapeutic strategies that not only combat infections but also engage host immunity. Conventional antibiotics often fall short due to toxicity, lack of immunogenicity, and disruption of commensal microbiota. Antimicrobial peptides (AMPs) offer a promising alternative, with dual roles in direct microbial killing and immune modulation.

This study characterizes KU04AMP01, a novel AMP derived from Pseudomonas bubulae KU04, highlighting its immunomodulatory and antimicrobial capabilities against Acinetobacter baumannii and Pseudomonas aeruginosa. KU04AMP01 retained its antimicrobial potency under variable physiological stress (pH, temperature, and salt), likely due to its stable random coil conformation. It disrupted bacterial membrane integrity, caused DNA damage, and led to nucleic acid leakage, exerting a multifaceted bactericidal effect.

Importantly, KU04AMP01 demonstrated no cytotoxicity in NIH3T3 fibroblast cells. Beyond its antimicrobial action, it significantly promoted T-helper cell proliferation via TLR7 and TLR9 signaling, attributed to its nucleic acid-binding affinity in a concentration-dependent manner. These findings suggest that KU04AMP01 not only eliminates MDR pathogens but also potentiates adaptive immune responses.

Overall, KU04AMP01 represents a promising immunotherapeutic candidate capable of both direct antimicrobial activity and host immune activation. Its dual-function profile offers a strategic advantage in addressing antibiotic resistance while enhancing immune readiness—paving the way for next-generation peptide-based therapies against MDR infections.

Poster No. 30

Itaconic Acid Modulates Tryptophan Metabolism and Suppresses Malignant Behaviour in Glioma Cells

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Glioblastoma (GBM) relies on altered metabolic programming and redox regulation to sustain aggressive growth. Itaconic acid, an immunometabolite produced by activated macrophages, is emerging as a modulator of cancer metabolism. Here, we investigated its effects on U87 glioma cells. Itaconic acid treatment led to marked downregulation of TDO2 at both mRNA and protein levels, indicating suppression of tryptophan–kynurenine metabolism. The upregulation of redox-responsive genes, such as NRF2 and HMOX1, indicates the activation of oxidative stress pathways. Functionally, itaconic acid significantly reduced viability, colony-forming capacity, and cell migration. Gelatin zymography further revealed reduced MMP-2/MMP-9 activity, indicating impaired matrix degradation. Together, these results show that itaconic acid simultaneously disrupts metabolic and functional hallmarks of GBM, highlighting its potential as a metabolic stress-inducing agent with anti-tumor effects.

Poster No. 31

From Cellular Activation to Systemic Immunity: Comprehensive Evaluation of a Multi-Epitope Vaccine against Leptospirosis

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Leptospirosis, a globally prevalent zoonotic disease, continues to pose a significant public health concern, leading to substantial morbidity and mortality in both humans and animals. Although several protective antigens have been identified, existing vaccine candidates have not achieved sterilizing immunity or broad cross-protection against the extensive diversity of *Leptospira* serovars. To address these limitations, we applied a comprehensive approach combining comparative genomics and reverse vaccinology to identify novel potential vaccine candidates (PVCs). Based on the most immunogenic B-cell and T-cell epitopes derived from these PVCs, we designed a multi-epitope vaccine (MEV) construct aimed at eliciting robust and cross-protective immune responses. The designed MEV construct was successfully synthesized, cloned, and expressed as a recombinant protein, which was subsequently purified. The immunostimulatory potential of the purified MEV was evaluated in RAW264.7 macrophages, where it induced the secretion of pro-inflammatory cytokines, upregulation of chemokine expression, indicating activation of the innate immune response. Furthermore, the recombinant MEV exhibited strong seroreactivity with sera from Leptospira patients, showing significantly higher reactivity compared to sera from healthy individuals. In vivo immunization studies in mice demonstrated that the MEV elicited robust immunogenicity, characterized by strong humoral and T cell-mediated responses. In addition, the MEV elicited a diverse humoral response characterized by the induction of IgG subtypes indicating a balanced Th1/Th2 immune response. Sera from MEV immunized mice recognized diverse pathogenic Leptospira serovars, exhibited bactericidal activity, and enhanced phagocytosis by antigen-presenting cells. However, in vivo challenge studies are yet to be performed to confirm the protective efficacy of the vaccine and these studies are ongoing.

Poster No. 32

Effect of all different dengue serotypes virus (DENV1-DENV4) in monocytes THP cell line culture growth

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Dengue virus is still a most important global health problem. Right now four different dengue serotypes are discovered known as serotypes: DENV-1, DENV-2, DENV-3, and DENVE-4. Now it is well established that an infection with one serotypes provides lifetime immunity to that particular serotypes where as subsequent infection with different serotypes can enhance the risk of severe dengue known as dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS). These both are life threatening form of dengue disease due to lack of proper treatment. Dengue viruses are spread to people through the bites of infected Aedes species mosquitoes (Ae. aegypti or Ae. albopictus). Once mosquitoes bite host, this enter into host cells, and starts replication within immune cells and makes their multiples copies of own genome. But unfortunately, how dengue virus serotypes-specific interaction with immune cells and their impact is not fully understood and still a area to discover further. Therefore, in this study we tried to investigate the effect of all four DENV serotypes (DENV-1 to DENV-4) on the growth dynamics, morphological changes, and inflammatory responses of THP-1 monocytes by using in vitro cell line model and our purpose was to proposed study using THP-1 cell line for the reason that dengue virus infects monocytes to replicate, circulate throughout the body also trigger pro-inflammatory responses that can contribute to severe dengue diseases. Dengue infected monocytes release cytokines that damage blood vessels, participates in vascular leakage and stimulate other immune cells like B cells to produce antibodies. In this study we infected THP-1 cell line with different dengue serotypes, and checked different dengue inflammatory cytokines, and cytopathic effect. DENV-2 and DENV-3 induced marked growth suppression and early apoptotic features, whereas DENV-1 showed moderate cytopathic effects and DENV-4 exhibited comparatively milder growth inhibition. These findings highlight critical differences in how each DENV serotype modulates innate immune cell behavior and provide new insights into the early cellular events that shape dengue disease severity. This work advances the understanding of serotype-specific host–virus interactions and supports the development of targeted therapeutic strategies for severe dengue.

Poster No. 33

Development of multiepitope-based Multivalent Vaccine Against Human Papilloma Virus (HPV): An Immunoinformatic Approach

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Human Papilloma Virus (HPV) is a major health concern responsible for genital warts and multiple cancers, including cervical, anal, penile and oropharyngeal malignancies. In India, cervical cancer affects women, with approximately 120,000 HPV-associated cases and 77,000 deaths in 2023. Persistent infection with high-risk HPV strains can integrate into host cells and express the E6 and E7 oncogenes, driving malignant transformation. Current commercial vaccines provide strong prophylactic protection but are limited in strain coverage and lack therapeutic utility. This study generates dual-pronged protection against HPV by developing multi-epitope-based prophylactic and therapeutic vaccines against the seven prevalent high-risk strains in India. Machine learning-guided models were applied to predict B-cell and T-cell epitopes against the L1 and L2 structural proteins, as well as the E6 and E7 oncogenic proteins, for prophylactic and therapeutic vaccines, respectively. These multi-epitopes were non-allergenic, non-toxic, antigenic, and immunogenic and were linked with a commercial adjuvant for the development of the vaccine construct. Further, a novel Enumerative pipeline was developed to generate all possible constructs of the vaccine configurations, enabling optimal candidate selection based on predicted efficacy. Molecular docking and molecular dynamics simulations demonstrated stable interactions between the vaccine constructs and key human Toll-like receptors for immune activation. Furthermore, immune simulation analyses were performed to validate the immunogenic traits of the designed constructs for eliciting robust immune responses. Collectively, these results suggest that the proposed multi-epitope vaccine constructs hold strong promise as next-generation prophylactic and therapeutic candidates for intervening in HPV-associated infections and malignancies.

Poster No. 34

Regulatory Role of PknJ on Pyka and MmaA4 During Mycobacterial Growth and Intracellular Survival

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Protein phosphorylation and dephosphorylation serve as fundamental mechanisms for translating external signals into intracellular responses. These processes regulate immune functions, cell growth, development, and division. Eukaryotic-like serine/threonine protein kinases (STPKs) have also been identified in bacteria, where they play critical roles in growth regulation and virulence. Among the 11 STPKs present in Mycobacterium tuberculosis (MTB) H37Rv, PknJ is notable for being conserved in slow-growing mycobacteria but absent in fast-growing species such as Mycobacterium smegmatis. In this study, we examined the physiological role of PknJ in both slow- and fast-growing mycobacteria. Our findings show that PknJ phosphorylates Pyruvate Kinase A (PykA), a key regulator of the glycolytic pathway. Notably, PknJ expression increases during infection of THP-1 macrophages, indicating its importance in host–pathogen interaction. Recombinant M. smegmatis expressing MTB-specific PknJ exhibited reduced growth and elevated PykA expression compared to the wild type. Furthermore, a pknJ knockout (KO) strain of BCG demonstrated significantly enhanced growth and a marked reduction in the expression of both PykA and MmaA4. We also observed that loss of PknJ increases rifampicin sensitivity, leading to a significant decrease in the minimum inhibitory concentration (MIC) during in vitro assays. Collectively, these results suggest that PknJ regulates critical metabolic and cell-wall-associated enzymes, including PykA and MmaA4, thereby influencing growth, antibiotic sensitivity, and virulence-associated traits in mycobacteria.

Poster No. 35

Energy dynamics in host-pathogen combat: Immune metabolism during *Mycobacterium tuberculosis* infection

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Mycobacterium tuberculosis (Mtb) rewires host cell metabolism for its survival. In lung, Alveolar macrophages (AMs) are the first cell type which interact with the pathogen and play a vital role during pathogenesis. Hence, the interplay between AM metabolism and Mtb burden provides pivotal insights into the metabolic adjustments regulated during infection.

However, little is known about the specific metabolic alterations in AMs that follow the shift in which Mtb uses the host cells metabolic processes to its advantage. Therefore, we developed a hypothesis to control Mtb load in human and murine macrophages by combining transcriptional repression with energy metabolism. We previously showed the negative correlation between NCoR1 (Nuclear Receptor Corepressor 1) expression and autophagic flux-mediated Mtb burden. Currently in C57BL/6 mice, parallel temporal dynamics of Mtb infection revealed unique patterns of ATP level and NCoR1 expression. Hence, we set out to understand the early host pathogen interaction by focusing metabolic modulation at single cell resolution in AMs collected from Broncho-Alveolar Lavage (BAL) fluid. To address these question, both control and NCoR1 KO (KO in macrophages) were challenged with H37Rv followed by Single cell sequencing. Future research will use animal models with rescue studies using metabolites to demonstrate efficient pathogen clearance as well as inhibitor assays to target metabolic pathways.

Poster No. 36

Differential Host Immune Responses to Commensal and Non-Commensal Microbes in an Intestinal Epithelial-Immune Cell Model

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Human gut commensals are known to strengthen intestinal barrier function, whereas pathogenic strains often compromise it, leading to heightened immune activation and tissue damage. Comparative investigations into the immune outcomes induced by commensals and pathobionts are essential to clarify their specific roles in health and disease. Molecular approaches, including amplification and quantification of cytokine and tight junction related genes, provide valuable insights into how different microbes modulate host responses. Therefore, in the present study, we examined the differential host immune responses elicited by gut isolates and free-living microbes using a co-culture model of Caco-2 cells and PBMCs challenged with whole microbial cells.

The results indicated that both types of microbes triggered immune responses; however, free-living microbes induced significantly higher expression of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-8 compared to gut-isolated microbes. In contrast, gut-isolated microbes significantly upregulated the expression of anti-inflammatory cytokines, including IL-4, IL-10, and TGF-β, suggesting that these strains promote a more balanced or tolerant immune profile. Overall, the study indicates that gut isolates stimulate a more immunoregulatory response, whereas free-living or pathogenic microbes provoke a predominantly inflammatory host response.

Poster No. 37

Vitamin D–mediated immune modulation under chronic sunlight UV exposure: Insights into Post Kala-Azar Dermal Leishmaniasis (PKDL) pathogenesis from endemic populations of Bihar, India

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Post Kala-Azar Dermal Leishmaniasis (PKDL), a sequela of Visceral Leishmaniasis (VL), remains a major impediment to Kala-Azar elimination efforts in Indian subcontinent, particularly in Bihar. PKDL patients serve as chronic reservoirs for Leishmania donovani, perpetuating disease transmission. The frequent localization of PKDL lesions on sun-exposed skin indicates a potential role of ultraviolet radiation (UVR) in its pathogenesis. Our study examined the contribution of long-term UVR exposure and Vitamin D receptor (VDR) gene polymorphisms to PKDL development in VL-endemic populations. Analysis of five-year ground and satellite UV indices revealed intense UVR levels (≥12.0–14.5) during summer and pre-monsoon seasons. Among 283 PKDL patients, 38% reported daily sun exposure ≥6–8 hours and 73% exhibited photosensitivity, predominantly among outdoor workers. A significant correlation was observed between UVR exposure and lesion severity. Immunological profiling demonstrated altered cytokine (IL-10, IL-12) and receptor (IFN-γR, TNFRs-1/2, TLRs-2/4) expression, along with dysregulated Vitamin D and iron levels. Experimental validation in THP-1 cells and Balb/c mice confirmed UVR-induced immune modulation mediated through Vitamin D pathways. Genetic analysis of VDR polymorphisms (rs1544410, rs7975232, rs731236) identified a strong association of BsmI (rs1544410) with VL and PKDL ($p < 0.0001$), where AA and GA genotypes conferred higher susceptibility. Expression studies showed up-regulation of VDR and CYP27B1 in pre-treated PKDL and reduced Vitamin D levels in VL ($p < 0.01$). Collectively, these findings highlight the synergistic influence of chronic sunlight UVR exposure and host Vitamin D genetic variations in PKDL pathogenesis, offering new insights for disease prevention and management in endemic regions.

Poster No. 38

Integrative Transcriptomics Analysis Reveals Dysregulation of Autophagy-Related Genes (ARGs) in Systemic Lupus Erythematosus (SLE)

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In autoimmune disorders, the immune system of the body fails to differentiate between self and non-self tissues and attacks the body's own tissues. Systemic Lupus Erythematosus (SLE) is one such autoimmune disorder, which is a chronic inflammatory condition and is characterised by relapsing and remitting flares. Autophagy is a key cellular process that is involved in the recycling of cellular organelles and defective proteins and helps in cellular energy homeostasis. Autophagy has been implicated in the pathogenesis of SLE; however, its transcriptional regulation remains unclear. The current study investigates the involvement of Autophagy Genes (ARGs) in SLE using transcriptomics data analysis. RNA-seq datasets were retrieved from Gene Expression Omnibus (GEO), which included both SLE patients and Healthy Controls. DESeq2 package in RStudio was used to identify the differentially expressed genes (DEGs). A curated list of 570 ARGs was compiled from the Human Autophagy Database (HADb) and GSEA–MSigDB gene sets. SLE-associated differentially expressed genes (DEGs) were intersected with the curated list of ARGs to identify those autophagy genes that are dysregulated in SLE. Protein–protein interaction networks is constructed using STRING and visualized in Cytoscape to identify the hub ARGs. Functional enrichment of the SLE DEGs and the ARGs curated shows the activation of interferon (IFN)-stimulated and antiviral pathways, which suggests an imbalance in the autophagic regulation in the immune cells in SLE patients. The identified ARG hub genes can act as therapeutic targets for treating SLE.

Poster No. 39

Understanding the regulation of protein secretion in Mycobacterium tuberculosis and its impact on bacterial physiology and host-pathogen interaction

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Tuberculosis, caused by Mycobacterium tuberculosis, remains one of the most tenacious infectious diseases of the past and present time, having infected a quarter of the global human population latently. The rise of drug-resistant strains and the limited efficacy of the century old BCG vaccine urgently necessitate the identification of novel therapeutic targets. M. tuberculosis lacks classical exotoxins and instead orchestrates remarkable immune evasive strategies mediated by its phenomenal secretory proteins and immunomodulatory lipids. Unfortunately, the underlying mechanisms of protein secretion machinery remain poorly understood in mycobacteria. In the present study, we have employed a CRISPRi-based, conditional silencing approach to interrogate the function of putative secretion machinery components in the virulent M. tuberculosis H37Rv strain and their impact on M. tuberculosis pathophysiology. Preliminary studies indicate the essential requirement of one such component in the maintenance of cell wall integrity and intracellular survival of the TB pathogen. Efforts are ongoing to elucidate the effect of its knockdown on whole cell proteome profile of the pathogen and disease pathogenesis in mouse model of TB infection.

Poster No. 40

Role of Circulating Immune Cell Signature in The Persistence of Measurable Residual Disease In Pediatric T-Cell Lymphoblastic Leukemia

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Weakened immune-cell surveillance has been a well-known factor responsible for the recurrence/progression of cancer. Studies have demonstrated the association of immune- cell profile with clinical outcomes in solid tumors and hematological malignancy like B-cell acute lymphoblastic leukemia. However, there is no data on Pediatric T-Cell Lymphoblastic Leukemia (T-ALL), which is a poor-risk leukemia. We and others have shown that post-induction measurable residual disease (PI-MRD) predicts relapse-free survival in T-ALL. Herein, we prospectively studied deep immune cell profiles (ICP), including immune checkpoint protein expression in the peripheral blood (PB) of pediatric T-ALL and their association with PI-MRD status. Our objective is to evaluate the role of Circulating Immune Cell Signature in The Persistence of Measurable Residual Disease in Pediatric T-Cell Lymphoblastic Leukemia. A 16-color five-tube antibody panel including antibodies against CD1c, CD3, CD4, CD7, CD8, CD10, CD11c, CD14, CD15, CD16, CD19, CD20, CD25, CD27, CD33, CD38, CD45, CD45RA, CD56, CD83, CD123, CD127, CD141, CD226, CXCR3, CCR4, CCR6, CCR7, CCR10, TCRγδ, LAMP-14-1BB, NKp46, HLADR, and immune checkpoint proteins Tim-3, PD-1, PDL-1, CTLA4, Lag3, OX40, OX40L, and TIGIT was studied for ICP analysis. Data was acquired on BD-LSR-Fortessa and analyzed using Kaluza-v2.0. Percentages & absolute counts of the immune subsets were studied in PB at diagnosis & day 35 post-induction. We studied the levels of 220 immune-cell subsets in peripheral blood from 134 Pediatric T-ALL patients (age, 1-15 years and M: F-4:1). PI-MRD was positive in 69/134 (51.5%) patients (range, 0.005-75%). Cut-off levels for association with positive MRD status were studied using ROC analysis. Analysis of diagnostic samples shown association of 6 subsets with PI-MRD (+) out of which in multivariant analysis PD1+ Th17 and CTLA4 + EM CD8 shown strong association with post induction MRD positivity. Evaluation of PB-ICP at day 35 post-induction showed an association of OX40L+ CM CD8 and TIGIT+ TC2 CD8T with PI- MRD(+) status om multivariant analysis. To our knowledge, this is the first study to evaluate deep immune-cell profiles in patients with pediatric T-ALL. Our data showed the association between exhausted immune cell subsets expressing immune checkpoint proteins such as PD1, CTLA4, and TIGIT with the PI- MRD(+) status. Immune checkpoint proteins play an inhibitory role in response to proinflammatory patterns and significantly contribute to the inhibitory signals regulating immune cell functions against tumor cells. Although limited to a small cohort, our data indicate the role of exhausted T-cell subsets in the immunobiological mechanism of the persistence of residual disease in pediatric T-ALL patients.

Poster No. 41

Propylthiouracil-Induced Hypothyroidism Drives Neuroinflammation and modulate DNA Methylation machinery in the Brain and Spinal Cord of Rats

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Hypothyroidism, marked by reduced T3 and T4 levels, disrupts metabolic functions and contributes to redox imbalance and neuroinflammation. This study investigates the impact of Propylthiouracil (PTU) on the subregions of brain (cerebrum and cerebellum) of Wistar rats focusing on redox imbalance, inflammation, and epigenetic alteration. PTU treatment significantly downregulated antioxidant enzymes (SOD1, SOD2, catalase) and BDNF, suggesting compromised oxidative defense and neuronal support. Elevated 4-hydroxynonenal (4 HNE) levels confirmed enhanced lipid peroxidation. Inflammatory markers, NLRP3, IL 1β, IL 18, Caspase 1, NF κB, and Gasdermin D, were also significantly upregulated, indicating activation of inflammasome cascade and an elevated neuroinflammatory state. Concurrently, proteins associated with DNA methylation such as DNMT1, DNMT3A, DNMT3B, MBD4, and MeCP2 were overexpressed at both transcript and protein levels, pointing to hypermethylation and transcriptional repression of neuroprotective genes. These results highlight that PTU induced hypothyroidism triggers a triad of oxidative stress, inflammation, and epigenetic dysregulation in the brain.

Such findings underscore the need for integrated therapeutic strategies targeting inflammatory, and epigenetic pathways in hypothyroid condition.

Poster No. 42

RBUL3 couple viral RNA sensing and RIG-I signaling for anti-viral defense

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RIG-I-dependent interferon (IFN) response is a key antiviral innate immune program of the host. RNA-binding E3-ubiquitin ligases (RBULs) constitute a sparsely characterized unique family of proteins with exceptional ability to regulate both at RNA and protein level for diverse biological functions. Here, we identify RBUL3 as a viral dsRNA sensing protein that triggers anti-viral interferon response. Mechanistically, we found that the viral dsRNA interaction with RBUL3 enhances its own K63-linked (auto)ubiquitination resulting in its oligomerization and stability. The activated RBUL3 interacts with RIG-I and MAVS over the mitochondria and promotes K63-linked ubiquitination of RIG-I for its enhanced stability leading to upregulation of type 1 IFN anti-viral program. Consistently, RBUL3 restricts the replication of multiple viruses. Further, conditional RBUL3KO mice were deficient in IFN response and anti-viral immunity. Thus, we uncovered an unidentified mechanism of viral sensing-signaling, where a single protein, RBUL3, could sense viral RNA and enhance anti-viral IFN signaling by ubiquitinating RIG-I.

Poster No. 43

Understanding the role of Angiomotin in regulating tumorigenic response: Involvement of Profilin1

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Malignant tumors are marked by their metastatic potential with cell migration being pivotal to tumor invasion. This migration relies on the dynamic remodeling of the actin cytoskeleton, orchestrated by various actin-binding proteins. Profilin1 (PFN1), a 15 kDa G-actin-binding protein is notably under-expressed in several cancers, including breast, pancreatic, and hepatic carcinomas. Our recent mass spectrometry analysis of PFN1 suggested Angiomotin (AMOT) as a probable interacting protein. AMOT is the most recently added component of the Hippo pathway, where it was discovered to inhibit the nuclear localization of Yes-associated protein (YAP) by cytoplasmic retention at tight junctions. YAP is the coactivator of the transcription factor TEAD, crucial in regulating the expression of genes controlling organ size control, tissue regeneration, and angiogenesis (including CTGF). Previous studies reported that PFN1interacts with PTEN and Akt through its PPXY motif binding domain independent of its Acting binding domain, thereby rendering stability to the said proteins and also recognizing the additional roles of PFN1 besides the traditional actin binding activity.

The interaction of PFN1 with AMOT was established by Co-Immunoprecipitation. AMOT+/+ and PFN1+/+ MDA MB-231 cells were generated and also ATRA (All Trans-Retinoic acid- FDA approved), a known inducer of PFN1 was used. Under PFN1 induced conditions of AMOT+/+ cells, YAP was observed to be arrested in the cytoplasm by immunoblotting. This was also reflected at transcript levels of CTGF-TEAD as shown by EMSA. Wound healing and Transwell assays were performed to understand the cell migration status which was observed to be attenuated in PFN1 induced AMOT+/+ cells and were comparable to that of PFN1+/+ cells. Immunoflourescence assay was performed to check the stability of AMOT in the presence and absence of MG132, where AMOT was noticeable in PFN1+/+ cells alone and also in MG132 treated conditions.

Findings till now suggest that, PFN1 interacting with AMOT enables YAP cytoplasmic retention thereby restricting migration of MDA MB-231 cells. PFN1 might be protecting AMOT from proteasomal degradation, which further needs to be validated. PFN1 might play a role in the Hippo pathway.

Poster No. 44

Regulatory T cell Defect Fuels Autoimmunity Through Aberrant Germinal Center-B cell Responses

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Regulatory T cells (Tregs) are essential for maintaining immune homeostasis, and their dysfunction is a hallmark of autoimmune inflammatory disorders. Foxp3 deficiency in Tregs leads to profound Treg impairment and uncontrolled activation of autoreactive T and B lymphocytes. The objective of this study was to elucidate the mechanistic link between Treg dysfunction, metabolic reprogramming, and germinal center (GC)–mediated autoimmunity. Using a Treg-specific deletion of Commd4 (Foxp3-YFPcre;Commd3fl/fl) mouse model, we investigated whether targeting glutamine metabolism could restore suppressive function in Commd3-deficient Tregs and ameliorate autoimmunity. Comprehensive metabolic profiling and immunophenotyping revealed that Commd3 deficiency markedly increased glutaminolysis through GLS1, which in turn fueled hyperactivation of $CD4^+$ T cells and aberrant GC B-cell responses, leading to excessive autoantibody production. To test the impact of metabolic intervention, we generated Tregs-specific depletion of GLS1 (Foxp3-YFPcre;Commd4fl/fl; Gls1fl/fl) and /or pharmacological inhibition of glutaminolysis by 6-diazo-5-oxo-norleucine (DON). Tregs-specific depletion of GLS1 and DON significantly prolonged survival, reduced CD44hiCD62Llo effector T cell activation, and decreased IFN-γ+Teff production, GL7+CD38-GC B cells formation, CTV+B-cell proliferation, and autoantibodies (IgG1 and IgM) generation while enhancing Treg suppressive capacity. In addition, to investigate to intriguing role of B cells and their autoantibodies are responsible for the inflammatory phenotypes and the reduced survival, we generated μMT−/−Foxp3-YFPcre;Commd4fl/fl mice and showed an approximately threefold increased average survival with reduced autoimmune manifestation. In conclusion, these findings demonstrate that glutamine metabolism is a critical regulator of Treg function and highlight metabolic targeting as a promising strategy to restore immune regulation and mitigate autoimmunity arising from Treg dysfunction.

Poster No. 45

A Novel Humanized BCMA CAR-T Therapy with Improved Durable Activity and Low Toxicity for Treatment of Multiple Myeloma

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Chimeric Antigen Receptor T (CAR-T) cell therapy targeting B-cell maturation antigen (BCMA) has transformed treatment options for relapsed and refractory Multiple Myeloma, yet current approaches remain limited by dose-limiting toxicities, T-cell exhaustion, and prohibitive manufacturing costs that restrict broader accessibility. In this study, we indigenously developed several humanized BCMA-targeted CARs and identified a candidate with the best safety and efficacy profile. Four novel constructs (h1-h4BCMA-BBζ) were designed and initially evaluated for vector titer, surface expression on T cells, binding affinity, and in vitro functional performance. Among these, h1BCMA-BBζ and h2BCMA-BBζ, displayed stable surface expression and strong proliferative capacity, with binding affinities comparable to a murine CAR (mBCMA-BBζ). Notably, h2BCMA-BBζ T cells exerted robust, antigen-specific killing of tumor cells and produced markedly lower inflammatory cytokines than murine CAR-T. Supernatants from h2BCMA-BBζ co-cultures induced substantially reduced IL-6 and IL-1β secretion from monocytes, indicating a lower potential for CRS induction. h2BCMA-BBζ T cells also showed superior expansion, a more stable memory phenotype, and reduced exhaustion under repeated antigen stimulation. In a murine myeloma xenograft model, h2BCMA-BBζ achieved complete tumor clearance and conferred a significant survival advantage (>120 days), whereas mBCMA-BBζ-treated mice relapsed and succumbed to disease by day 45. No systemic or organ-specific toxicities were observed post CAR-T administration. Collectively, these findings identify h2BCMA-BBζ as a promising next-generation anti-BCMA CAR with robust anti-tumor efficacy, improved persistence, and a favorable safety profile, supporting its evaluation in an ongoing Phase I clinical trial.

Poster No. 46

Epigenomic control of JunB by NCoR1 and SMRT fine-tunes conventional Dendritic Cell responses

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Epigenetic regulation of immune responses is broadly driven by primed and repressed chromatin states. Coordinated action of TFs, Co-regulators and epigenetic enzymes regulates the outcomes of inflammatory and tolerogenic states. However, the epigenomic control of this differential response in DCs remains elusive. We recently reported that Nuclear Receptors co-repressors NCoR1 and its paralog SMRT (also known as NCoR2) fine-tune immune-tolerance and inflammation in Dendritic Cells (DCs). However, the epigenomic control underlying this differential DC response generation by NCoR1 and SMRT is still unclear. Here, we did an integrative epigenomic analysis of control, NCoR1 and SMRT depleted conventional type-I DCs (cDC1) with or without CpG-B stimulation to understand this mechanism. Clustering analysis of the NCoR1 and SMRT overlapping H3K27ac marked enhancers and global gene expression identified two activation dependent clusters depicting 307 genes / enhancers to be upregulated only in NCoR1 KD condition, and 285 upregulated in both NCoR1 as well as SMRT KD DCs. NCoR1 depletion primarily upregulated both inflammatory and anti-inflammatory pathways, whereas SMRT depletion upregulated only the inflammatory pathway genes like IL-12, IL-6 and IL-23, with a strong down-regulation of anti-inflammatory genes IL-10 and SOCS3. De novo motif analysis identified JunB transcription factor (TF) to be significantly enriched at NCoR1 bound cluster 1 enhancers. Western blotting confirmed the differential expression of pJunB / JunB in NCoR1 and SMRT KD DCs. Moreover, JunB ChIPseq depicted drastically reduced binding on downregulated enhancers (cluster 1) in contrast to a significant increase in upregulated enhancers (cluster 2). Further analysis identified that this differential enrichment of JunB at cluster 1 and cluster 2 genes is controlled by differential TF usage like cRel and STAT1. Interestingly, our analysis also depicted that SMRT binding preferentially shifts to promoter-proximal regions upon CpG activation, correlating with significantly higher RNA Pol-II enrichment at the TSS of SMRT-bound as compared to NCoR1 and NCoR1-SMRT co-bound genes. This strongly suggested control of RNA Pol-II transcriptional activity directly or indirectly by SMRT, which needs further investigation.

Poster No. 47

Exploring Genome Dynamics: From Computational Discovery to Clinical Application

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Our research develops integrative genomics frameworks to understand genome dynamics by analyzing DNA sequence, structure, energy, variation, expression, and epigenetic parameters. Early studies focused on identifying potential drug targets for cancer and pathogens, leading to the development of two bioinformatics tools-OncoRegulon and Pathogen-Specific DNA Drug Finder. This effort evolved toward exploring DNA's energetic landscape, where solvation, stacking, and hydrogen-bonding energies were shown to distinguish functional genomic regions. Building on these insights, we advanced to structural DNA analysis, predicting transcription start sites in prokaryotes and intron–exon boundaries in eukaryotes, resulting in the creation of ChemGenome2.1, a genome-annotation software based on physicochemical DNA properties. Subsequently, my postdoctoral research investigated the relationship between genomic alterations, structural features, and transcriptional regulation in cancer. In metastatic clear cell Renal Cell Carcinoma (RCC), TP53 and SMARCA4 mutations were identified as markers of aggressive disease trajectories. In MiT/TFE translocation RCC, fusion-driven rewiring produced conserved expression modules across species, indicating genome-structure–driven evolution rather than mutation-driven changes. Studies of rapalog resistance revealed that stromal fibroblast reactivation within the tumor microenvironment drives adaptive therapeutic responses, while HIF2α inhibition through RNA-targeting drugs established proof-of-principle for RNA-based therapeutics in RCC. Currently, my Computational Oncology Lab at NIT Rourkela integrates bulk, single-cell, and spatial transcriptomics to investigate tumor-grade progression in RCC and cellular heterogeneity in Adamantinomatous Craniopharyngioma (ACP). Through multi-omics integration, we aim to elucidate immune–stromal interactions, identify therapeutic targets, and advance precision oncology strategies for cancers of national relevance.

Poster No. 48

Zinc transporter (ZIP3) orchestrates zinc-dependent switching between autophagy and apoptosis in *Leishmania donovani*

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Visceral Leishmaniasis (VL) caused by the intracellular protozoan parasite of genus *Leishmania* remains a significant global health crisis as no vaccine is available. Increasing drug resistance presses the need to identify novel molecular targets. Survival of Leishmania critically depends on zinc and its homeostasis. The objective of this study is to investigate the role of zinc transporter (ZIP3) in maintaining zinc homeostasis and survival in *Leishmania donovani*. Chelation of intracellular zinc using TPEN significantly reduced parasite viability by 72 hours, though partial viability was retained at 48 hours, suggesting a biphasic zinc stress response. Fluorescence imaging with FlsuoZin-3 AM and a partial rescue of viability via exogenous zinc supplementation confirmed the critical role of zinc. FACS analysis revealed that no significant apoptosis was observed at 48 hours, but at 72 hrs, the percentage of apoptotic cells was increased. Concurrently, semi-quantitative PCR revealed a time-dependent transcriptional upregulation of LdZIP3, ATG8, and SIR2 till 48 hours post-TPEN exposure. This gene expression profile suggests an early pro-survival mechanism: increased LdZIP3 for enhanced zinc uptake, and activation of autophagic markers, ATG8 and SIR2 for parasite survival under zinc stress. We hypothesize that sustained zinc deprivation triggers a shift from this transient pro-survival autophagic response to a later apoptosis-like death. Establishing LdZIP3 regulated autophagy-to-apoptosis cascade may position it as a novel therapeutic target.

Poster No. 49

Tumor-derived Osteopontin (OPN) mediates macrophage infiltration and polarization and is a potential target in intra-tumoral immunity in melanoma

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Tumor-associated macrophages (TAMs) are greatly appreciated in cancer invasion, migration, tumorigenesis, angiogenesis, therapy resistance, and immune suppression. Osteopontin (OPN), a chemokine-like ECM protein, is identified as a non-collagenous, sialic acid rich and chemokine-like glycosylated phosphoprotein, known to enhance several hallmarks of cancer. Tumor-derived OPN drives monocyte recruitment and macrophage polarization within the tumor microenvironment (TME). The functional diversity of TAMs has versatile roles in tumor events; however, their function in response to OPN remains unanswered. Mouse macrophage (RAW264.7) and human monocyte (THP1) cells were activated by interaction with conditioned media (CM) melanoma cells, and the effect of OPN was examined on TAM polarization using in vitro melanoma models. TAMs were characterized by checking the expression of CD206 and CD163 by flow cytometry. Here, we report that the interaction between cancer cells and macrophages promotes tumor growth. The cancer cells' release osteopontin (OPN), a protein that functions as a chemokine, and initiates monocyte recruitment and macrophage polarization into TAMs. In this study, we report that tumor-derived OPN enriches TAM functional states with a tumor-promoting function. Based on flow cytometry data, we found that there is a decrease in TAM polarization in macrophages treated with OPN Neutralised melanoma cell CM in comparison to CM of parental melanoma cells. Furthermore, we demonstrate that OPN-affected TAMs promote melanoma cell proliferation and migration. Our findings suggest that macrophages, upon interaction with OPN, activate a tumor-promoting function. Thus, OPN may act as a potential intra-tumoral target and OPN-regulated TAM polarized signaling cascades might act as a potential therapeutic target for the management of melanoma growth and metastasis.

Poster No. 50

The Dynamic Role of Mycobacterial Glutamate Decarboxylase in Tuberculosis Pathogenesis

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Intracellular pathogens like *Mycobacterium tuberculosis* (M.tb) employ metabolic adaptations to survive host-derived stresses. Our studies using LC-MRM/MS analysis identified key metabolites and pathways such as the γ-aminobutyrate (GABA) shunt in M.tb's response to abiotic stresses. Given the conserved nature of metabolites across organisms, M.tb-produced metabolites may influence infected macrophages, potentially altering their phenotypic states, affecting infection outcomes. We hypothesized that M.tb Glutamate Decarboxylase (GadB) aids stress adaptation and modifies the host environment. To validate the same, we subjected M.tb to various abiotic stresses and observed increased transcript and protein levels of GadB, alongside elevated intracellular levels of GABA and related enzymes of the shunt pathway. We validated GadB as a functional enzyme using reverse phase HPLC, and observed enhanced catalytic efficiency and substrate binding at acidic pH. Catalytic (GadB_SDM) and C-terminal deletion (GadB_ΔCt) mutants exhibited reduced activity, particularly the latter, which compromised pH-dependent function. Notably, M.tbGadB activity increased in the presence of H_2O_2, conferring its role in oxidative environment. Overexpression of M.tbGadB in M.tuberculosis provided a growth advantage in acidic and oxidative media, with the mitigation of total ROS. Infection of THP-1 macrophages with M.tb::M.tbGadB enhanced bacterial persistence. Cytokine profiling of THP-1 macrophages infected with M.tb::M.tbGadB revealed an M2 polarization, with increased anti-inflammatory cytokines, cell surface markers and other signatures of M1/M2 phenotypes. In summary, M.tbGadB is vital for GABA synthesis, aiding stress reduction and modulating host environment, making it a potential target for anti-M.tb strategies. We are next screening inhibitors against GadB to target metabolic interactions during Mtb pathogenesis.

Poster No. 51

Development of Sensitive Immunoassays to Quantify Human Serum C-Reactive Protein.

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C-reactive protein (CRP) is an acute-phase protein, an inflammatory marker mainly synthesized in the liver. CRP levels in the blood reflect acute or chronic inflammation. Elevated CRP levels are commonly associated with chronic inflammatory diseases. However, sensitive CRP quantification is associated with a significant indication in cardiovascular disease (CVD) risk management. Serum CRP below 2 mg/mL shows no CVD risk, whereas 2-3 mg/L indicates moderate risk and above 3 mg/L indicates a high future risk of CVDs. However, current detection methods are not sufficiently sensitive. Hence, we have optimized a sensitive radioimmunoassay (RIA) and a direct ELISA for CRP. First, a highly sensitive RIA assay (0-200 ng/mL) is developed, which can measure the CRP level in human serum as low as 3 ng/mL. For competitive RIA, CRP was radiolabelled using the chloramine-T method, and the separation phase was based on magnetizable cellulose particles. The optimized assay included two pipetting steps and an incubation time of 1 h at room temperature. In another direct ELISA, the anti-CRP antibody was conjugated with HRP using the sodium periodate oxidation method. The optimized assay protocol is as follows: Add 25 µL standards (0 to 100 ng/mL) to each well and incubate for 2 hrs at RT, followed by plate wash. The blocking was carried out for 30 minutes, followed by washing. Then, add 100 µL of anti-CRP-pAb-HRP to each well and incubate for 1 hr. Afterwards, 50 µL TMB was added to the wells, 0.2M H2SO4 was added to stop the reaction, and the absorbance was measured at 450 nm to plot the standard graph. Optimized assays were analyzed and validated for assay sensitivity, precision, dilution linearity, recovery, and cross-reactivity. The assay covers the clinical range of human CRP analysis for clinical tests.

Poster No. 52

Understanding RANKL-mediated biological responses involving arthritis and it regulation by suitable inhibitor.

Bindi Goradia

Rheumatoid arthritis (RA) is a chronic autoimmune disease characterized by persistent inflammation, joint destruction, and dysregulated cytokine signalling. Central to RA pathophysiology is the aberrant activation of chemotactic cytokines that recruit and polarize immune cells within the synovial microenvironment. The RANKL/RANK signalling axis plays a pivotal role in osteoclast differentiation and bone remodelling, while also modulating NF-κB–regulated cytokine networks that sustain inflammatory responses.

This study investigates RANKL-mediated NF-κB activation and its downstream effects on chemokine expression, macrophage polarization, and osteoclastogenesis. In vitro assays demonstrated that RANKL stimulation upregulated pro-inflammatory cytokines (IL-1β, TNF-α, IL-6) and chemotactic mediators (CCL2, CXCL8), promoting recruitment and M1 polarization of macrophages. RT-PCR and proteolytic enzyme assays confirmed enhanced inflammatory signalling, while TRAP staining validated osteoclast differentiation. Impaired autophagy was observed to prolong osteoclast survival, further contributing to bone resorption.

These findings highlight a mechanistic link between RANKL signalling, chemotactic cytokine regulation, and macrophage polarization in RA pathogenesis. Moreover, Resveratrol emerges as a promising therapeutic candidate capable of disrupting NF-κB–driven chemokine networks, thereby attenuating inflammation and osteoclast-mediated bone loss.

Poster No. 53

Synergistic effect of dual drug loaded PLGA@MnO2 nanoparticles targeting Tumor microenvironment (TME) of Triple negative breast cancer (TNBC) as chemo-immunotherapy through immunogenic cell death(ICD)

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TNBC accounts for 15-20% of breast cancer subtype is the most aggressive with highest relapse and mortality rate due to lack of specific targets. TME is the major hallmark of TNBC as it induces proliferation, immunosuppression, drug-resistance etc through dynamic interaction of its various cellular and acellular components. Chemo-immunotherapy is an emerging treatment option for cancer treatment that combines chemotherapy with immunotherapy to overcome their respective challenges. Phytochemicals are well known for their low toxicity and ability to target cancer cells through multiple mechanisms, so many are in different stages of clinical trials. Initially, we have screened different phytochemicals which are either in clinics or at different stages of clinical trials and can elicit ICD and anti-tumor immune response and found that Berbamine(BM), an alkaloid has shown to induce ICD synergistically in combination with Doxorubicin (Dox) (standard drug for ICD) at a very lower concentration. For this, we formulated BM and Dox co-loaded PLGA@MnO2 nanoparticles(NPs) as drug delivery system and characterized their physio-chemical properties including the O2 generating ability of PLGA@MnO2 in in vitro. Further, we have checked the ICD markers i.e Calreticulin, HMGB1 and eATP of the above nanoformulation in TNBC mouse cell line 4T1 and antitumor immune response in dendritic cells in Mutu DC1940 cell line and found it to be better in inducing ICD and maturation of DCs than the respective individual Dox or BM . Our findings suggest that the above nanoformulation Dox-BM-PLGA@MnO2 NPs can be a promising therapeutic strategy to improve the chemo-immunotherapy for TNBC.

Poster No. 54

Understanding the effect of CCR9-CCL25 chemokine signaling on dendritic cells

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Dendritic cells (DCs) are specialized antigen-presenting cells that initiate adaptive immune responses by capturing antigens and presenting them via MHC molecules to naïve T cells. This process, supported by co-stimulatory molecules and cytokine secretion, drives T cell differentiation. Specific chemokine molecules support the migration of DCs. Thymus expressed Chemokine (TECK) or chemokine CCL25, produced by thymic and intestinal epithelial cells, acts as a chemoattractant for immune cells expressing CC-chemokine Receptor 9 (CCR9), including T cells and DCs. Also, basal expression of CCL25 is maintained, contributing to the thymus and gut homeostasis. It has been found that $CCR9^{+}$ DCs promote regulatory T cell formation and inhibit inflammatory T cell subtypes when stimulated by CCL25 in co-culture with naïve $CD4^{+}$ T cells, indicating a role in peripheral tolerance. However, the origin, functions, and downstream elements of the CCR9-CCL25 axis remain poorly understood. Given the abundance of CCR9+ cells in the thymus, we studied thymic microenvironmental factors in CCR9-/- thymus compared with wild-type thymus using mass spectrometry-based proteomics. Additionally, we studied the immunophenotype of the dendritic cell line DC2.4 upon CCL25 stimulation and during CCR9 upregulation. Finally, we validated the tolerogenic phenotype of CCR9+ DCs on CCL25 stimulation

Poster No. 55

Influence of Membrane Lipid Composition and CD40L Dose on CD40 Domain Localization and Signalosome Formation

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CD40, a costimulatory receptor on macrophages, requires precise membrane partitioning to build its signalosome complex and initiate downstream p38MAPK- or ERK-driven immune responses. However, how membrane lipid composition influences CD40 localization and signalosome assembly remains unclear. This study investigated the CD40 signalosome complex in peritoneal macrophages from BALB/c mice under infection, cholesterol perturbation, and CD40L stimulation. Macrophages were isolated following thioglycolate elicitation, rested, and exposed to Leishmania donovani, β-MCD, or cholesterol supplementation in defined experimental groups. CD40L stimulation was performed for 15 minutes, followed by NP-40 lysis and CD40 co-immunoprecipitation. LC–MS proteomic analysis of the Co-IP complexes revealed clear differences in unique and common proteins between infected and uninfected macrophages, and cholesterol modulation effectively generated mimic conditions that resembled infection-induced or cholesterol-depleted states. Cholesterol depletion by β-MCD produced proteomic profiles similar to infected macrophages, while cholesterol addition restored profiles toward uninfected conditions, confirming the role of membrane cholesterol in regulating CD40 complex composition. In a complementary experiment, membrane microdomain localization was analyzed through HistoDenz density-gradient ultracentrifugation, followed by immunoblotting. CD40 signalosome components were detected in both detergent-resistant (raft) and detergent-soluble (non-raft) fractions; however, increasing CD40L doses (1–6 μg) shifted the signalosome from non-raft regions toward cholesterol-rich raft fractions, indicating ligand-dependent redistribution. Together, these results demonstrate that infection, cholesterol availability, and CD40L concentration dynamically influence CD40 signalosome composition and membrane partitioning, highlighting cholesterol as a key regulator of CD40-mediated signaling.

Poster No. 56

PLPP1-Driven Sphingolipid Remodeling Regulates Terminal CD8⁺ T Cell Dysfunction in CRC: AN INTEGRATED SINGLE-CELL TRANSCRIPTOMIC ANALYSIS

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Colorectal cancer (CRC) remains poorly responsive to immunotherapy, partly due to an immunosuppressive tumor microenvironment (TME) and pervasive T cell dysfunction. To investigate the metabolic and transcriptional mechanisms driving CD8⁺ T cell exhaustion, we performed an integrated single-cell RNA sequencing (scRNA-seq) analysis using three publicly available CRC datasets (GSE146771, GSE161277, and GSE200997). Unsupervised clustering identified 22 distinct cell populations, revealing substantial alterations in stromal and immune cell composition within tumor tissues. Focused re-clustering of CD8⁺ T cells uncovered 13 phenotypically diverse subsets, including naïve-like, GZMK⁺, regulatory, and exhausted (TEX) cells. Tumor-infiltrating CD8⁺ T cells exhibited significantly elevated exhaustion scores ($p < 2.2e{-}16$), accompanied by upregulation of inhibitory receptors such as PDCD1, CTLA4, HAVCR2, ENTPD1, and LAG3. Among the top differentially upregulated genes in TEX cells, PLPP1, a key regulator of sphingolipid metabolism, emerged as a prominent exhaustion-associated molecule. Pseudotime trajectory analysis demonstrated progressive PLPP1 induction from naïve to terminally exhausted states, paralleling exhaustion-related transcription factors including TOX and TIGIT. scMetabolism analysis further revealed selective enrichment of sphingolipid metabolic pathways in TEX cells, suggesting PLPP1-driven metabolic reprogramming. To experimentally validate these findings, we established an in vitro model of CD8⁺ T cell exhaustion via repeated CD3/CD28 stimulation and treated cells with sphingolipid pathway modulators (C6-ceramide, fingolimod, fumonisin B1, and myriocin). Flow cytometry and ELISA analyses showed altered exhaustion marker expression and cytokine profiles upon metabolic inhibition, confirming a functional link between sphingolipid metabolism and exhaustion severity. Together, these in silico and in vitro data identify PLPP1 as a metabolically active checkpoint that reinforces CD8⁺ T cell exhaustion in CRC, highlighting the PLPP1–sphingolipid axis as a promising target to restore T cell function and improve immunotherapy outcomes.

Poster No. 57

Evaluating NCOR1 as a potential prognostic marker for tuberculosis therapy

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Variability in immune responses to infections and inflammation arises from genetic, epigenetic, and environmental factors. Innate immune cells, including dendritic cells (DCs) and macrophages, play crucial roles in regulating inflammation by producing cytokines like IL-12, IL-23, and IL-10. These cytokines determine immune outcomes, influencing T-helper cell differentiation and balancing immune activation and tolerance. Recent studies in mice reveal that NCOR1 and SMRT are central to determining susceptibility to bacterial infections and immune-related diseases. However, their roles in humans remain poorly understood. This study hypothesizes that individual differences in NCOR1 levels influence infection predisposition and inflammatory responses. By comparing healthy individuals with those experiencing infection or inflammation, we aim to uncover pathways controlled by these co-repressors and their impact on innate immune function. Understanding these mechanisms could identify potential biomarkers and therapeutic targets for infectious and inflammatory diseases, providing insights into personalized approaches for immune modulation in clinical applications.

Poster No. 58

Host-Microbe-Phytochemical Interactions Reveal Commensal-Dependent Immunomodulation by *Tinospora cordifolia*

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Host–microbial interactions in the human gut play a critical role in maintaining health. Alterations in these host-microbial immuno-interactions have been associated with various physiological disorders. Factors capable of modulating these immune interactions may therefore serve as potential therapeutic interventions. However, limited information on host immune responses toward gut commensals, as well as the influence of phytochemicals on these interactions, remains a major constraint. Therefore, the present study was designed to elucidate host immune reactions in the presence of a gut commensal and to assess the role of Tinospora cordifolia in modulating these interactions using an intestinal model.

A human intestinal co-culture model comprising intestinal epithelial cells (HT-29) and peripheral blood mononuclear cells was exposed to a gut commensal (Cellulomonas HM71) to evaluate host immune responses. Individual exposure to either the commensal or the plant extract did not induce significant changes in gene expression. However, co-exposure to the commensal plus plant extract, compared with plant extract alone, resulted in substantial transcriptional variation, with 238 differentially expressed genes (81 upregulated and 157 downregulated). Upregulated transcripts were enriched for innate immune and interferon-related pathways, along with complement factors, indicating enhanced chemokine-mediated leukocyte recruitment and activation. Overall, the plant extract alone did not modulate the host immune response, whereas the presence of the commensal bacteria revealed a strong immunomodulatory effect. These findings suggest that the plant extract and gut commensals engage in cross-talk to shape intestinal immune responses, rather than acting independently.

Poster No. 59

A novel role of circCPSF6 regulating antiviral innate immunity via miR-665 and PCBP2-IPS-1 axis

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Innate immunity plays a vital role to subvert virus infection via dynamic regulatory networks that calibrate antiviral response and prevent excessive inflammation. These networks are co-ordinately regulated by coding and non-coding elements of genome. The non-coding elements, particularly circular RNAs (circRNAs) have emerged as a key modulator of various biological processes, yet their precise function in innate immune response during influenza A virus (IAV) infection remains insufficiently defined. Here, we have identified circCPSF6, a conserved circRNA that remains suppressed during IAV infection both in vitro and in vivo. Functional analyses revealed circCPSF6 as an antiviral regulator through dual regulatory mechanism involving RNA-RNA and RNA-protein interaction. CircCPSF6 sponges proviral miR-665, thereby alleviating the repression of key antiviral mediators such as MyD88, STAT2 and IKKε via RNA-RNA interaction. At RNA-protein level, circCPSF6 directly interact with RNA binding protein PCBP2, which is a known negative regulator of IPS-1, thereby sustaining IPS-1 mediated antiviral signaling. These interactions enhance cytokines and interferon-stimulated genes expression which enhance host antiviral response. Our findings report circCPSF6 as a crucial regulator of host innate antiviral immune response and highlight circRNA mediated network as a potential therapeutic target during virus infection.

Poster No. 60

Leptospira-Induced Modulation of Dendritic Cells Across Host Species

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Leptospirosis is a globally neglected zoonotic disease, and the early immune events that determine host susceptibility remain incompletely understood. Dendritic cells (DCs) are central orchestrators of antigen presentation and T cell priming, yet their interaction with pathogenic Leptospira across different host species has not been comprehensively explored. In this study, we generated DCs from mouse bone marrow, human monocytes, and bovine peripheral blood to evaluate how multiple Leptospira serovars influence DC activation and maturation. DCs were stimulated with distinct pathogenic serovars, and their responses were assessed through analysis of key surface markers (CD40, CD80/86, MHC-II) and cytokine profiles (IL-12, TNF-α, IL-6, IL-10) using flow cytometry and immunoassays. Preliminary findings indicate clear differential modulation of DC activation both across species and among serovars, reflected in variable patterns of cytokine secretion and surface marker expression compared with classical TLR-driven activation. These observations suggest that Leptospira may employ species-specific or serovar-dependent strategies to influence DC function, potentially shaping downstream T cell responses and contributing to variation in disease outcomes. This work provides a foundational framework for understanding early innate immune modulation by Leptospira and supports future efforts aimed at developing DC-targeted immunomodulatory tools or vaccine strategies for improved control of leptospirosis.

Poster No. 61

Genetic diversity of *Plasmodium vivax* circumsporozoite protein (PvCSP) gene: a candidate for vaccine development

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Plasmodium vivax is the second most prevalent and the most geographically widespread species. Notably, P. vivax exhibits greater genetic diversity, can form liver-stage hypnozoites, and tends to cause relapses. The P. vivax circumsporozoite protein (PvCSP), is crucial for sporozoite development, invasion of mosquito salivary glands, and entry into liver cells, making it a primary target in malaria vaccine research. While CSP-based vaccines have shown promising results, the extensive genetic variation in the natural population of P. vivax poses a significant concern, as this variation can considerably impact the parasite's traits, treatment responses, and other biological characteristics. In this study, P. vivax isolates were collected from various health centres across Goa. DNA was extracted, and the CSP gene was amplified. The amplified products were then subjected to Sanger sequencing, and the polymorphisms in the CSP gene were analyzed using appropriate software. A total of 110 samples were collected, with 93.6% being VK210, 3.6% VK247, and 2.7% showing mixed infections of VK210 and VK247. The sequence analysis of the central repeat region (CRR) of VK210 revealed two abundant peptide repeat motifs: GDRADGQPA and GDRAAGQPA. Additionally, the sequence analysis identified six subtypes (A1-A6) within the VK210 variant, differentiated by variations in the order and frequency of the peptide repeat motifs. Among these, the A4 subtype was the most prevalent, accounting for 45.31% of the samples. This study highlights the predominance of the VK210 variant and underscores the importance of understanding parasite population genetics. This study also provides valuable insights into the development of a PvCSP-based vaccine that targets multiple strains of P. vivax.

Poster No. 62

TGF-β Mediates Immune Suppression in Visceral Leishmaniasis

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Visceral leishmaniasis persists as a major global health concern due to increasing drug resistance and the absence of effective vaccines, highlighting the need for host-directed immunotherapies. Transforming growth factor-β (TGF-β) plays a dual pro-pathogenic role during Leishmania donovani infection by suppressing macrophage activation and impairing IFN-γ–mediated effector functions. This study aimed to investigate the impact of TGF-β inhibitors on restoring macrophage responsiveness and enhancing anti-leishmanial immunity in both infected BALB/c mice and peritoneal macrophages. Peritoneal macrophages were infected with L. donovani and treated with pathway-specific inhibitors—ITD1, INH9, SIS3, and Salirasib—at multiple doses followed by assessment of nitric oxide production, iNOS gene expression, parasitic load, and cytokine responses. In vivo, infected mice were treated with the same inhibitors alone or in combinations, and parasite burden and spleenocyte cytokine profiles were evaluated. Flow cytometry, qPCR, and Western blot analyses were performed to examine IFNγRα/β expression and STAT1 phosphorylation. TGF-β inhibition significantly enhanced nitric oxide production, upregulated iNOS, reduced intracellular amastigotes, and restored Th1 cytokines (IFN-γ, TNF-α) while suppressing IL-10 and IL-6. Infected macrophages showed marked downregulation of IFNγRα and impaired STAT1 phosphorylation, both of which were reversed by SIS3 and Galunisertib treatment. In vivo treatment similarly reduced hepatic and splenic parasite burden and improved T-cell–mediated immunity. Collectively, these findings demonstrate that targeting TGF-β signaling effectively restores IFN-γ–driven anti-leishmanial responses and offers a promising host-directed therapeutic strategy for visceral leishmaniasis.

Poster No. 63

Integrative Modelling Approach for Accurate T-helper cell Subtype Plasticity Prediction

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The dynamic interplay between Th17 and induced regulatory T (iTreg) cells is central to shaping immune responses, particularly in the context of chronic inflammation and immune resolution. While earlier experimental data has implied a rigid and mutually exclusive commitment to either lineage, more recent findings have revealed that Th17 and iTreg differentiation is, in fact, highly plastic and context-dependent. Notably, even under identical external cues such as TGF-β, naïve CD4+ T cells can diverge into distinct phenotypic populations. To mechanistically explain this heterogeneity, we have developed a mathematical model to simulate the Th17/iTreg differentiation axis. The model exhibits distinct stable states and reflects how subtle variations in precursor cell properties can lead to diverse lineage outcomes. The system is sensitive to modulatory inputs like IL-6, IL-2, IL-21 and TGF-β, which fine-tune differentiation trajectories and guide cells into specific effector roles. Intriguingly, the model also predicts the emergence of a late-stage cell state that co-expresses high levels of both RORγt and Foxp3—which can either be as a transient intermediate or as a stable phenotype arising under defined signaling conditions. These computational insights not only align with a broad range of experimental observations but also propose testable hypotheses about uncharacterized differentiation pathways and potential transitional states, highlighting the utility of modelling in uncovering immune complexity.

Poster No. 64

Development of T cell-guided mRNA vaccine candidates for Japanese encephalitis virus

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Japanese encephalitis (JE), a severe viral encephalitis caused by the Japanese encephalitis virus (JEV). JE impacts more than 3 billion people in Asia and the Western Pacific with a death rate of ~30%. Existing vaccines, such as live attenuated and inactivated virus vaccines, decrease the burden of disease; however, they have limitations, including strain diversity and inadequate immune protection. We proposed a T cell-based mRNA vaccine candidate that incorporates unique and conserved T cell epitopes from virus proteins, which comprise both structural and non-structural proteins. Its purpose is to elicit robust, targeted cell-mediated immunity—specifically, activating T cell responses to confer lifelong protection against JEV infection. The strategy concentrates on highly conserved T-cell antigenic epitopes that enhance memory generation, cross-strain protection, and cellular immunological processes essential for virus clearance. The methodology involves identifying conserved T-cell epitopes, followed by their validation. We have already identified some of the conserved T cell epitopes from both structural and non-structural proteins of the JE virus. The structure of individual peptides was predicted by AlphaFold, which demonstrates the structural integrity of the peptides. Functional validation of these epitopes is currently underway, including assessment of their capacity to induce antigen-specific T cell responses and cytokine production. For downstream development of an mRNA-based vaccine construct, including sequence engineering, codon optimization, and synthesis, we plan to engage a specialized organization with expertise in mRNA platform design and manufacturing to ensure optimal expression and translational efficiency. Overall, this design effectively directs the host's T cell-mediated immunity toward multiple critical viral determinants, ensuring broad, cross-genotypic, and long-lasting protection against JEV infection. By addressing strain variability and the limitations of conventional vaccine platforms, this approach represents a promising advancement in the development of next-generation vaccines.

Poster No. 65

Multi-omics analysis of human patient samples identifies key immune factors in Leptospira infection

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Leptospirosis is a globally neglected zoonotic disease caused by pathogenic Leptospira spp. Despite being known for two centuries, the pathogenesis and host response to Leptospira infection remain poorly understood. To identify critical host factors involved during infection we employ an integrated multi-OMICs approach to investigate the critical host factors during interaction of Leptospira. RNA-seq and LCMS/MS based analysis in PBMCs isolated from patients having febrile illness and a comparative analysis with the proteome of whole blood infection model with Leptospira revealed a complex interplay between gene and protein expression patterns. To address patient heterogeneity, we identified 52 commonly upregulated and 8 downregulated proteins across both RNA and protein levels. Gene ontology analysis suggests these proteins are pivotal in innate immune pathways such as response to stress, response to external stimuli, and defence response. An in vitro model mimicking leptospiral infection validated these findings, revealing 10 proteins consistently upregulated across various serovars infection.7 significant genes were identified after validation, which have a role mainly in immune regulation. Utilizing multi-OMICS approach we elucidated the human host factors which are differentially expressed during Leptospira infection. Further research is needed to elucidate the specific roles of these proteins in Leptospira-host interactions and to develop novel strategies for prevention and treatment of this disease.

Poster No. 66

Meta-inflammation in T2DM: unveiling the role of aberrant CD4+ T cells and pro-inflammatory cytokine networks

Chandrasekhar Pattanaik

Type 2 Diabetes Mellitus (T2DM) is characterized by chronic hyperglycaemia, insulin resistance, and impaired glucose tolerance. Recent
evidence links T2DM to persistent low-grade inflammation driven by elevated inflammatory cytokines such as TNF-α, IFN-γ, and IL-17. This inflammatory state contributes to poor wound healing, cardiovascular complications, and multi-organ damage. Hyperglycaemia is proposed to amplify cytokine production, sustaining immune dysfunction and inflammation. In this study, we aim to investigate how hyperglycaemia and cytokines influence T cell activation in T2DM through TCR signaling, MAPK pathways, and transcriptional regulation, to better understand the role of chronic inflammation in immune dysregulation.

Poster No. 67

Identification of Plasma Immune markers Predictive of Latent Tuberculosis Infection in HIV-Infected Individuals

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Tuberculosis (TB) remains a major cause of morbidity and mortality among people living with HIV (PLH), particularly in high-burden regions. Despite antiretroviral therapy (ART), PLH are predisposed to latent TB infection (LTBI) and its progression to active disease due to immune dysregulation. Identifying immune signatures linked to LTBI progression versus resistance is crucial for developing predictive biomarkers and targeted prevention strategies. Prospective cohort of 80 HIV-infected adults on ART was followed for six months and stratified into four groups based on serial QuantiFERON-TB Gold results: Progressors (12.5%), Sustainers (15.0%), Converters (13.8%) and Resisters (58.8%). Plasma cytokines, chemokines, and growth factors (38 analytes) were quantified using a 45-plex luminex assay. Random forest and principal component analyses identified predictive markers, while protein–protein interaction (PPI) networks and gene set enrichment analysis (GSEA) explored immune pathways. Progressors and sustainers exhibited significantly elevated proinflammatory cytokines (TNF-α, IL-6, IL-17A), chemokines (CXCL10, CCL11), and growth factors (VEGF, PDL-1, Granzyme B) compared to resisters and converters. Dominance analysis identified PDL-1, TNF-α, IL-17A, and FGF2 as top predictors of LTBI progression. Progressors showed minimal longitudinal immune changes, whereas resisters displayed significant modulation of 22 proteins following isoniazid preventive therapy (IPT). PPI networks highlighted IL-10, IL-1β, and TNF-α as key hubs. GSEA revealed enrichment of innate and inflammatory pathways in progressors, while resisters showed adaptive and antiviral pathway dominance. A persistent proinflammatory signature, particularly PD-L1, TNF-α, IL-17A, and FGF2 defines LTBI progression. These markers show strong potential for predicting LTBI risk and informing targeted prevention.

Poster No. 68

Bioengineered, His-Tag and Signal-Peptide Free SPy_2191 Antigen with Alum Provides Safe and Robust Protection against *Streptococcus pyogenes.*

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Group A *Streptococcus (S. pyogenes*) remains a leading cause of invasive and non-invasive infections worldwide, underscoring the urgent need for an effective vaccine. We evaluated a bioengineered SPy_2191 antigen, His-tag and signal-peptide free, formulated with alum, for safety, immunogenicity, and protective efficacy in mice. Immunized mice exhibited excellent safety with no clinical or histopathological abnormalities. Humoral responses were found to be robust, with high titers of cleaved SPy_2191-specific IgG, including both IgG1 and IgG2b, indicating a balanced Th1/Th2 profile. Cellular immunity was confirmed by enhanced antigen-specific $CD4^+$ and $CD8^+$ T cell proliferation and significant production of IFN-γ, IL-4, and IL-17, highlighting activation of Th1, Th2, and Th17 pathways. High levels of chemokines (MCP-1/ CCL2, MIP-1α/ CCL3, MIP-1β/ CCL4 and RANTES/ CCL5) indicate orchestrated a strong but controlled immune response in immunized mice. Functional antibody activity was demonstrated via opsonophagocytic killing of S. pyogenes in vitro. Importantly, immunized mice were found to be protected against lethal dose of S. pyogenes, exhibiting 88% survival and markedly reduced bacterial loads in key organs. Importantly, our in-vitro and in-vivo toxicology study showed that cleaved SPy_2191 protein is effective and safe. These results establish that the His-tag and signal-peptide free SPy_2191antigen, delivered with alum, is safe, highly immunogenic, and capable of inducing coordinated humoral and cellular immune responses that confer functional protection against S. pyogenes. This study supports cleaved SPy_2191 as a promising candidate for further preclinical development and highlights its potential as a universal vaccine strategy against Group A Streptococcal infections.

Poster No. 69

Anti-RNP antibody footprint in patients of mixed connective tissue disease and systemic lupus erythematosus

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Mixed connective tissue disease (MCTD) is a rare autoimmune disease where there is positivity to anti-ribonuceloprotein(RNP) antibodies along with overlapping features of systemic lupus erythematosus, scleroderma and polymyositis. This poses a diagnostic dilemma where there are overlapping features of lupus in MCTD. The aim of this study was to understand the presentation and consequence of patients with anti-RNP antibodies to identify aspects that differentiate them. This was a retrospective cohort study of patients over a period of four years of 2018-2022 with anti-RNP antibodies positive. The immunofluorescence patterns, signs and symptoms were also obtained from lab records. Anti-RNP was positive in 246(35.29%) individuals. Out of these 246 positive patients 21 were diagnosed with MCTD and undifferentiated MCTD. The antibody profile revealed 56% each positivity for anti-Sm and anti-SSA and 47% positivity for anti-Ro-52.2. Out of 246 individuals were 98 anti-RNP positive were diagnosed as per SLE criteria and showed positivity for anti-RNP. Based on the cluster there were differential clinical features which when placed into the SLE criteria and the Kahn and Alarcón-Segovia criteria helped in improving the diagnosis of MCTD. Anti-RNP antibodies are not the uniqueness of MCTD diagnosis as the association is also observed in SLE. However, SLE patients had clustering of antibodies along anti-RNP which also helped in ruling out MCTD from SLE. Further as already known the Kahn and Alarcón-Segovia criteria can be used to rule in the disease.

Poster No. 70

Integrated Oncobiome–Transcriptome Profiling Reveals Host–Microbe Networks in Non-Muscle Invasive Bladder Cancer

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Bladder cancer is characterized by high recurrence, yet the contribution of the tumor-associated microbiome (oncobiome) to disease pathogenesis remains incompletely defined. This study integrates transcriptomic and microbial profiling to elucidate host–microbe interactions in non-muscle invasive bladder cancer (NMIBC).

Meta-transcriptomic analysis was performed on paired tumor and adjacent normal bladder mucosa samples from six patients (n = 12). RNA sequencing was used to identify differentially expressed genes (DEGs) and to profile microbial community composition. Host–microbe interaction networks were constructed using correlation analysis. Fifty-seven DEGs ($\log_2FC \geq 1$) were identified, including 45 downregulated and 12 upregulated genes. Of these, 53% (30/57) were linked to extracellular matrix remodeling pathways, notably TMEM119 and DCN. Tumor tissues exhibited significantly reduced microbial alpha diversity compared with normal mucosa ($p = 0.026$). Distinct microbial signatures distinguished tumor from normal tissue: Propionibacterium acnes was enriched in tumors (23.9% vs. 13%), while Prevotella copri (12.7% vs. 1.7%) and Bifidobacterium longum (3.9% vs. 0.84%) were more abundant in normal mucosa. Network analysis revealed 80 significant host–microbe correlations involving 18 bacterial species and 45 genes. Corynebacterium durum and Veillonella dispar showed strong associations with extracellular matrix genes. TMEM119 emerged as a central hub, negatively correlated with B. longum, Bacillus flexus, and Faecalibacterium prausnitzii, and positively correlated with Prevotella stercorea and Streptococcus luticiae. This integrated multi-omics approach reveals complex host–microbe networks in NMIBC, particularly involving extracellular matrix remodeling. The distinct microbial signatures and extensive gene–microbe correlations suggest that the bladder oncobiome may shape tumor biology through modulation of the microenvironment. These findings lay the groundwork for microbiome-informed diagnostic and therapeutic strategies in bladder cancer.

Poster No. 71

Leptospira-Induced Polarization Dynamics in Human Macrophages

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Leptospirosis is a zoonotic and re-emerging infectious disease of global importance. In recent years, a severe form of the disease known as leptospirosis-associated severe pulmonary hemorrhage syndrome is becoming prevalent due to excessive inflammation of lungs. While antibiotics like doxycycline and penicillin are commonly employed for treatment, challenges such as antibiotic resistance underscore the pressing need for alternative therapeutic approaches. Moreover, due to delayed diagnosis antibiotics are ineffective at later stage of substantial organ damage caused by inflammation. Macrophages are important innate cells which play crucial role in pathogens defense as well as in tissue maintenance. Their remarkable adaptability/plasticity includes a phenomenon known as macrophage polarization, a dynamic process wherein these cells undergo functional specialization into two distinct phenotypes M1 and M2, shaping the immune response and influencing various physiological processes. M1 macrophages exhibit pro-inflammatory responses crucial for pathogen clearance, while M2 macrophages play a role in anti-inflammatory processes, tissue repair, and immune regulation. Several bacterial pathogens are known to subvert macrophage polarization for their own survival, hindering effective immune clearance, however how zoonotic bacterial pathogen Leptospira influences macrophage polarization in disease progression remain poorly understood. In this study, we investigated the temporal dynamics of polarization during Leptospira infection in macrophages of mouse, human and bovine host species. Using flow cytometry to assess surface markers, ELISA for cytokine profiling, RT-PCR for gene expression analysis, and western blotting for protein expression, we elucidated that there is differential response in these macrophages which effects the polarization shifts over time. Further studies using omics (transcriptomics and proteomics) and different bioinformatics tools will identify the molecular markers involved in pathogenesis. These markers may be exploited for developing novel therapeutic intervention for controlling severe inflammation and associated pathology to prevent damage and eventually multi-organ dysfunction during acute Leptospirosis.

Poster No. 72

Outer Membrane Vesicles from *Helicobacter pylori* Reprogram Macrophages to Enhance Survival and Impair Antimicrobial Function

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Helicobacter pylori establishes chronic gastric infection by modulating host immune cells, particularly macrophages. One key mechanism involves the release of outer membrane vesicles (OMVs), nano-sized particles carrying bacterial components that interact with immune cells. While OMVs are known to trigger inflammation, their role in altering macrophage antimicrobial function remains poorly understood. In this study, OMVs were isolated from H. pylori culture supernatants via ultracentrifugation and characterized using nanoparticle tracking analysis (~80 nm), scanning electron microscopy (SEM), and transmission electron microscopy (TEM), confirming spherical morphology. PMA-differentiated THP-1 macrophages efficiently internalized OMVs in an actin-dependent manner, as shown by Cytochalasin D inhibition. Once internalized, OMVs induced robust inflammatory activation, including elevated ROS production and TLR2/NF-κB pathway signaling. Across 72 h, OMVs progressively impaired macrophage autophagy, leading to reduced intracellular killing and enhanced survival, as indicated by MTT, LDH release, Annexin V/PI staining, and autophagy marker analysis (LC3B, p62). These results indicate that H. pylori OMVs generate a pro-inflammatory but functionally compromised macrophage phenotype, supporting immune evasion and persistence during chronic infection. Understanding OMV-mediated macrophage reprogramming provides key insights into host–pathogen interactions and identifies potential targets to limit bacterial survival and associated gastric pathology.

Poster No. 73

Transcriptomic and Clonotypic Landscape of Human Antigen-Specific CD4⁺ T-Cell Responses to Mycobacterium tuberculosis and Dengue Virus

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CD4⁺ T cell immunity is shaped by antigen exposure, even in the absence of overt disease; yet, during immune steady state in the absence of clinical disease remains poorly understood. Common human pathogens such as SARS-CoV-2, Mycobacterium tuberculosis (Mtb), dengue virus (DENV), and cytomegalovirus cause recurring antigen encounters in healthy populations, generating antigen-experienced CD4⁺ T cells, including T follicular helper (Tfh) cells, without clinical manifestation. This study aimed to compare antigen-specific immune responses to these pathogens in healthy adult donors representing a steady-state immune condition, defined by serological evidence of prior exposure but no history of past or ongoing clinical disease, closely approximating asymptomatic immune activation, to assess functional activity, helper potential, and cellular heterogeneity, with a particular focus on Tfh-associated responses. Comparative analyses revealed antigen-dependent differences in CD4⁺ T cell functional profiles, including variation in helper-associated cytokine production and B cell–helping capacity. Antigen-specific CD4⁺ T cells from a subset of donors were further examined to characterise transcriptional and clonal features of responses to DENV and Mtb as representative viral and intracellular bacterial antigens. Dengue-specific central memory-like CD4⁺ T cells, including Tfh populations, exhibited transcriptional programs distinct from those of Mtb-specific counterparts, despite sharing similar phenotypic markers, suggesting divergent functional states shaped by distinct antigens. T cell receptor repertoire analysis revealed differential clonotype expansion. Collectively, these findings demonstrate that antigen type differentially shapes human CD4⁺ T cell organisation even in the absence of clinical disease.

Poster No.74

Inflammatory and Lung-Specific Biomarker Signatures in Tuberculosis–Type 2 Diabetes Mellitus Comorbidity

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The coexistence of Tuberculosis (TB) and type 2 diabetes mellitus (T2DM) results in advanced disease manifestations and hence understanding the inflammatory disturbances and identifying biomarkers is essential for improving treatment strategies and their outcome. This study evaluated the inflammatory profile associated with TB–diabetes comorbidity (TBDM) by analysing both systemic acute phase proteins- C-Reactive Protein (CRP), α2-macroglobulin, Haptoglobin, and Serum Amyloid-A1 (SAA1) and lung-associated inflammatory markers- Indoleamine-2,3-dioxygenase, Chitinase-3-like protein 1 (CHI3L1), S100A9, S100A8, and Granulysin measured to assess systemic inflammatory responses and characterize localized pulmonary inflammation. Three study groups were included: TB-only (n=22), TB with diabetes (TBDM; n=44), and diabetes-only (n=22). Plasma analyte levels were quantified using DuoSet sandwich ELISA kits, and group-wise differences were statistically evaluated. CRP and SAA1 were significantly elevated in the TB-DM group compared to both TB-only and DM-only groups (CRP: $p<0.0001$ vs DM, $p < 0.001$ vs TB; SAA1: $p = 0.0003$ vs DM, $p < 0.001$ vs TB). Lung-associated markers, S100A8 and S100A9 showed significant increases in TBDM (S100A8: TBDM vs TB $p = 0.0021$, TBDM vs DM $p = 0.0352$; S100A9: TBDM vs TB $p = 0.0018$, TBDM vs DM $p < 0.0001$), indicating enhanced neutrophil-mediated pulmonary inflammation in comorbid individuals. These elevations suggest that DM Comorbidities amplifies both systemic and lung-specific inflammatory responses, contributing to greater disease severity. Overall, this study highlights the substantial impact of diabetes on the immune-inflammatory landscape of TB and underscores the need for integrated approaches addressing both metabolic dysregulation and infectious pathology to improve clinical outcomes.

Poster No.75

Monocyte TNF-Superfamily Signalling Dysregulation in Tuberculosis–Type 2 Diabetes Mellitus Comorbidity

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Type 2 diabetes mellitus (T2DM) increases susceptibility to tuberculosis (TB) and contributes to dysregulated immune responses, partly through altered monocyte signalling. Although our earlier work has demonstrated that T2DM alters monocyte subsets and promotes hyperinflammatory responses during TB, the mechanisms underlying dysregulated TNF-superfamily pathways in TB-T2DM comorbidity are underexplored. We aimed to characterise TNF- superfamily receptor and co-stimulatory molecule expression in a cross-sectional cohort of 176 individuals (TB = 44, TB-T2DM = 44, T2DM-only = 44, healthy controls = 44), and T2DM status was confirmed by HbA1c (hyperglycaemia defined as HbA1c $\geq$ 6.5%). Plasma TNF receptors and co-stimulatory molecules were quantified using a Human ProcartaPlex Mix & Match 12-plex immunoassay panel (multiplex ELISA), and the results were correlated with monocyte-associated TNF-α responses following stimulation with Mycobacterium tuberculosis antigens (PPD, whole-cell lysate) and TLR ligands, assessed by flow cytometry. Results demonstrated TB-T2DM individuals exhibited a distinct immune phenotype characterized by reduced expression of CD137, CD40, GITR, TNFR1/2, and TRAIL-R1/R2, with elevated CD27, CD30, and CD95 expression compared to other groups. Spearman correlation analysis revealed a significant negative correlation between TNF receptor expression and monocyte TNF-α responses under unstimulated and TB-antigen conditions ($p < 0.05$), indicating impaired receptor-mediated regulatory feedback in TB-DM. Conversely, TLR-induced TNF-α responses demonstrated strong positive correlations across stimuli ($p < 0.0001$), reflecting preserved innate activation but impaired antigen-specific regulation in hyperglycaemia. Conclusively, these findings emphasize hyperglycaemia-driven monocyte dysfunction alters the TNF signalling, potentially contributing to a delay in effective immune activation and pathogen clearance in TB-DM comorbidity.

Poster No. 76

Understanding the Opposing Effects of Cannabinoid Signalling on the CREB–NF-κB Regulatory Network

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Cannabinoids are the pharmacologically active compounds known to regulate various metabolic and immune-regulatory pathways by binding to a class of G-protein-coupled receptors (GPCRs) called cannabinoid receptors(CBRs). Cannabinoid receptors are increasingly recognized as regulators of immune-cell transcriptional programs, yet their coordinated effects on cAMP-response element–binding protein (CREB) and NF-κB remain insufficiently understood. This study aims to elucidate how cannabinoid receptor engagement influences the balance between CREB-mediated immune-regulatory pathways and NF-κB–driven pro-inflammatory signalling. We observed that stimulation of cannabinoid receptor(CB2) favours CREB activation while concurrently attenuating NF-κB–associated inflammatory genes, reflecting reduced expression of inflammatory mediators and enhanced induction of genes linked to cellular resilience and regulatory function. In contrast, CB1 modulation produced more heterogeneous effects, with context-dependent shifts in CREB phosphorylation and NF-κB activity. Overall, the pattern suggests that CB2 signalling promotes a transcriptional environment biased toward immune regulation rather than inflammation. These results collectively indicate that cannabinoid receptor involvement can differentially shape CREB and NF-κB activity, positioning CB2 as a potential pivot point for modulating immune homeostasis. In conclusion, our findings highlight the CREB–NF-κB regulatory axis as a central mechanism through which cannabinoid receptors influence immune responses and shed light on the therapeutic promise of selectively targeting this axis in inflammatory and immune-mediated conditions.

Poster No. 77

Microbial Tryptophan Metabolites in Gut Immune Regulation

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Tryptophan(Trp) metabolism serves as a critical link between diet, the gut microbiota and the host immune system, making it pivotal to mucosal homeostasis and disorders affecting the intestinal surface. While host metabolism converts Trp into serotonin and kynurenine(Kyn), emerging evidence shows that microbial transformation of dietary Trp into indole-metabolites are immunoregulators. Gut microbes metabolize Trp into derivatives such as indole-3-aldehyde, indole-3-propionic acid and indole-3-acetic acid, which activate AHR and PXR signaling to induce IL-22 and IL-10, increase antimicrobial peptide and mucin gene expression, strengthen epithelial barrier integrity and promote immune tolerance. In contrast, the kyn pathway(KP), catalyzed by IDO1/2 and TDO2, generates metabolites that modulate T-cell differentiation and dendritic cell activity, while the serotonin pathway regulates gastrointestinal motility and neuroimmune communication. Dietary patterns strongly influence the balance among these pathways, fiber-rich diets enhance beneficial indole production. Preclinical studies show that the absence of indole-producing bacteria increases susceptibility to colitis, infection and metabolic inflammation, while supplementation with indole metabolites or high-potency indole-producing strains provides substantial protection. As these pathways strongly control inflammation and barrier repair, modulating them may help treat IBD, reduce infection risk and improve metabolic health. Earlier work using broad-spectrum probiotics supported this concept but produced variable outcomes due to inconsistent microbial survival and limited control over metabolite production. Current evidence emphasizes the need for strategies that identify robust indole-producing taxa, develop targeted probiotic and optimize dietary inputs that reliably restore indole-mediated immune balance and improving mucosal health.

Poster No. 78

Reprogramming Tumor-Associated Macrophages with Nanoparticles to Restore Checkpoint Sensitivity

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Tumor-associated macrophages (TAMs) are abundant immunosuppressive cells within the tumor microenvironment, driving a "cold" phenotype that is resistant to immune checkpoint blockade. Innovative nanoparticle-based immunotherapies are now being designed to reprogram TAMs into a proinflammatory M1-like state and turn cold tumors hot. For example, lipid or polymeric nanoparticles functionalized with macrophage-targeting ligands can deliver Toll-like receptor (TLR) or STING agonists, proinflammatory cytokines, or inhibitory siRNAs directly to TAMs. These cargoes activate NF-κB/IRF signaling and suppress pro-tumor pathways, thereby boosting M1 markers (IL-12, iNOS, TNF-α) and reducing M2 markers (IL-10, Arginase-1). Consequently, chemokines produced by reprogrammed TAMs attract and activate cytotoxic CD8+ T lymphocytes. TAM-targeted nanoparticles efficiently turn immunologically "cold" tumours into "hot" ones by increasing T cell infiltration and IFN-γ expression in preclinical tumour models. Interestingly, combining these nanoparticles with anti-PD-1/PD-L1 therapy results in strong tumour regression and enhanced survival; in certain instances, complete remission is even achieved, suggesting that checkpoint inhibitor sensitivity has been restored. To improve TAM targeting and payload release, recent approaches also make use of biomimetic or stimuli-responsive nanoplatforms (such as cell-membrane-coated or pH-sensitive NPs). Encouraging preclinical research highlights the translational potential of TAM reprogramming using nanoparticles, demonstrating that it can significantly inhibit tumor growth. Overall, by altering the immunological landscape of the tumour and boosting the effectiveness of checkpoint inhibitors, nanoparticle-mediated reprogramming of TAMs shows potential for overcoming immunotherapy resistance.

Poster No. 79

Restoring adaptive immunity of foamy macrophages against Mycobacterium tuberculosis infection by Sphingosine -1 Phosphate in experimental model system

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Foamy macrophages are essential for maintaining Mycobacterium tuberculosis (M.tb) infection because of their altered lipid metabolism and compromised immune systems. The potential of sphingosine-1-phosphate (S1P) to regulate lipid-loaded macrophages and reinstate their ability to promote adaptive immunological responses is assessed in this work. MACS-based positive selection was used to separate $CD14^+$ monocytes from PTB/MDR-TB patients and healthy donors, and M-CSF was used to differentiate them into macrophages. LPS, LDL, fatty acids, and TB sera were used to create foamy macrophages, which were verified by morphological analysis and Oil Red O staining. Lipid accumulation is much greater in MDR-TB patient's macrophages than in healthy controls, suggesting improved metabolic reprogramming. Patient-derived macrophages had higher levels of nitric oxide and enhanced production of IL-6, in contrast to the low-NO, Th2-type foamy phenotype. This profile is consistent with an immunosuppressive, MDSC-like condition, indicating disruption of normal macrophage activation and possible impairment of T-cell priming. S1P decreases IL-6 levels and decreased excessive NO generation, and partially reversed the inflammatory imbalance in foamy macrophages. ApoE, Clec4E, Clec7A, GP96, LDLR1, IL-4, and IL-13 shown to be differentially regulated in MDR-TB macrophages by gene expression analysis, indicating changes in immune signalling and lipid recognition pathways linked to the severity of the illness. Overall, results indicate that the S1P–NO–IL6 axis as a key route regulating foamy macrophage activity during TB. By focussing on this pathogenic foamy macrophages may be reprogrammed into a more immunocompetent state, providing a possible supplementary strategy to enhance host immunity in drug-resistant tuberculosis.

Poster No. 91

Development of Phytocompound-Mediated Therapy Against Oral Submucous Fibrosis

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Oral Submucous Fibrosis (OSMF) is a potentially malignant oral disorder prevalent in Southeast Asia, including India. It is primarily caused by areca-nut chewing, leading to progressive buccal mucosa fibrosis with burning sensation, blanching, stiffness, and limited mouth opening. Long-term exposure to areca-nut and progressive OSMF significantly increases the risk of oral carcinoma. The current treatment regimen with hydrocortisone & hyaluronidase often shows relapse and limited efficacy, underscoring the need for more effective therapeutic options. Phytocompounds are emerging as promising candidates to curb disease progression, with limited detrimental effects. The objective of this study is to explore new targeted therapy against OSMF using phytocompounds to determine their anti-fibrotic properties and efficacy through in-silico, in-vitro and in-vivo methods. Network pharmacology-guided identification of OSMF-associated targets and phytocompound networks was used to determine the phytocompound(s) that can target the disease modules. Furthermore, Molecular docking was employed to identify the active compound(s), following molecular dynamics simulation to validate the atomic-level stability and thermodynamic behaviour of receptor-ligand interactions. The active compound(s) were further subjected to in-vitro validation using both TGF-β and arecoline-mediated fibrosis models of Human Gingival Fibroblast. Our active phytocompound lowered the expression of fibrotic biomarkers in this transformed fibroblasts (myofibroblast). Additionally, our phytocompound selectively mediates the apoptosis of myofibroblasts with no effect on normal fibroblasts. Our integrated network pharmacology-guided experimental validations collectively highlight the phytocompound as a promising therapeutic against OSMF, providing a compelling basis for further preclinical validation and potential translational applications.

Poster No. 92

Immunomodulatory Fate of Nimbolide in Triple negative breast cancer (TNBC)

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Breast cancer remains the most leading cause of death in women worldwide. Amongst all subtypes, TNBC is highly aggressive, accounting for 15%-20% with limited therapeutic options because of the absence of estrogen, progesterone and minimal expression of HER2 receptor. Chemotherapy is the primary choice of the treatment for TNBC, however, the emergence of drug resistance, immunosuppressive microenvironment, metastasis and high rate of recurrence underscores the urgent need for novel therapeutic strategies. Phytochemicals have recently gained increasing attention as novel treatment options in current cancer treatment. Recently, our group has demonstrated nimbolide, a limonoid triterpene derived from Azadirachta indica, have shown anti-cancer activity by targeting cancer cells, cancer stem cells. Also, we are currently working upon nimbolide's role in targeting cancer associated fibroblasts. However, no studies to date have reported the immunomodulatory properties of nimbolide. So, we are highly interested in exploring the immunological potential of nimbolide in the tumor microenvironment of TNBC. To proof the concept, we did a pilot experiment by taking 6-8 weeks female 4T1 bearing orthotopic BALB/c mice for checking the immune cell infiltration to the tumor microenvironment after treating the tumor bearing mice with a dose of 5mg/kg nimbolide and 10mg/kg nimbolide. Our results suggest with nimbolide treatment there is an increase in infiltration of immune cells such as cytotoxic T cells, M1 macrophages and dendritic cells with a reduction in M2 macrophages, regulatory T cells (Tregs) and MDSCs as compared to the control. We anticipate that nimbolide can be a good immunomodulatory candidate capable of reshaping the immune microenvironment, overall promoting antitumor immunity. Further, to enhance its bioavailability and targeted immunomodulatory effects, our future work will be focused upon formulating a nimbolide-based nanoparticle to strengthen the antitumor immunity in TNBC.

Poster No. 93

Potassium Enhances IL-9 Production in CD4 T cells and Boosts Effector Function of CAR T Cells

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Interleukin-9 (IL-9), a pleiotropic cytokine with potent immunomodulatory and anti-tumour functions, is predominantly produced by T helper 9 (Th9) cells, as well as Th2, Th17, and regulatory T cells. Although the transcriptional regulation of IL-9 is well studied, its ionic regulation remains largely unexplored. Using IL-9 GFP reporter mice, we screened physiologically relevant ions (K+, Ca2+, Mg2+, and Zn2+) for their effects on Th9 differentiation and IL-9 production. Remarkably, only extracellular potassium (eK+) significantly enhanced IL-9 expression in an ion channel-dependent manner. Elevated eK+ also boosted IL-9 production in other IL9–producing subsets, while suppressing IL-17 and reprogramming Th17 cells into Th9-like cells via upregulation of Foxo1, a transcription factor we previously identified as essential for Th9 biology. Importantly, potassium supplementation markedly improved the effector function of CD19-directed Th9 CAR T cells in B cell co-culture assays. These findings reveal an ionic mechanism regulating T cell plasticity and suggest a novel immunometabolic strategy to enhance CAR T cell therapy.

Poster No. 94

The Infectious Origins of Autoimmune Disorders: Psoriasis and Sarcoidosis as Models

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One of the most important and well supported contributors to autoimmune disease is infection. Various pathogens have been linked to numerous autoimmune diseases, for example Epstein-Barr virus (EBV) is linked to Multiple sclerosis, SARS-CoV-2 to Lupus, oral bacteria such as P.gingivalis to periodontal disease to name a few. This study focuses on understanding how commonly found environmental microbes trigger autoimmune disease, namely Psoriasis (a skin inflammation) and Sarcoidosis (Lung autoimmune disease). By analysing blood and tissue samples from patients with Psoriasis and Sarcoidosis, and applying single-cell RNA sequencing, immunological techniques, and clinical parameters, we show how microbial triggers shapes the immune responses and contributes towards disease development. We found that immune responses targeted against Group A Streptococcus (GAS) and airborne fungi *Apsergillus nidulans* trigger autoimmune led inflammation at distant sites. GAS bacteria were found to stimulate neutrophils in skin to exhibit antigen presenting properties that presented and activated T-cells in skin leading to inflammation. Similarly, A.nidulans was found to trigger T-cells in BAL that lead to autoimmune responses in the lungs of HLA-DR3+ sarcoidosis patients. These data highlight the importance of understanding the infectious agents as central to immune dysfunction in autoimmunity so that clinicians and researchers can move toward more precise, preventive, and personalized approaches to autoimmune care.

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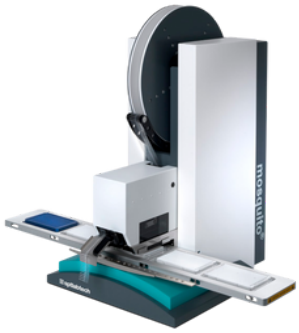
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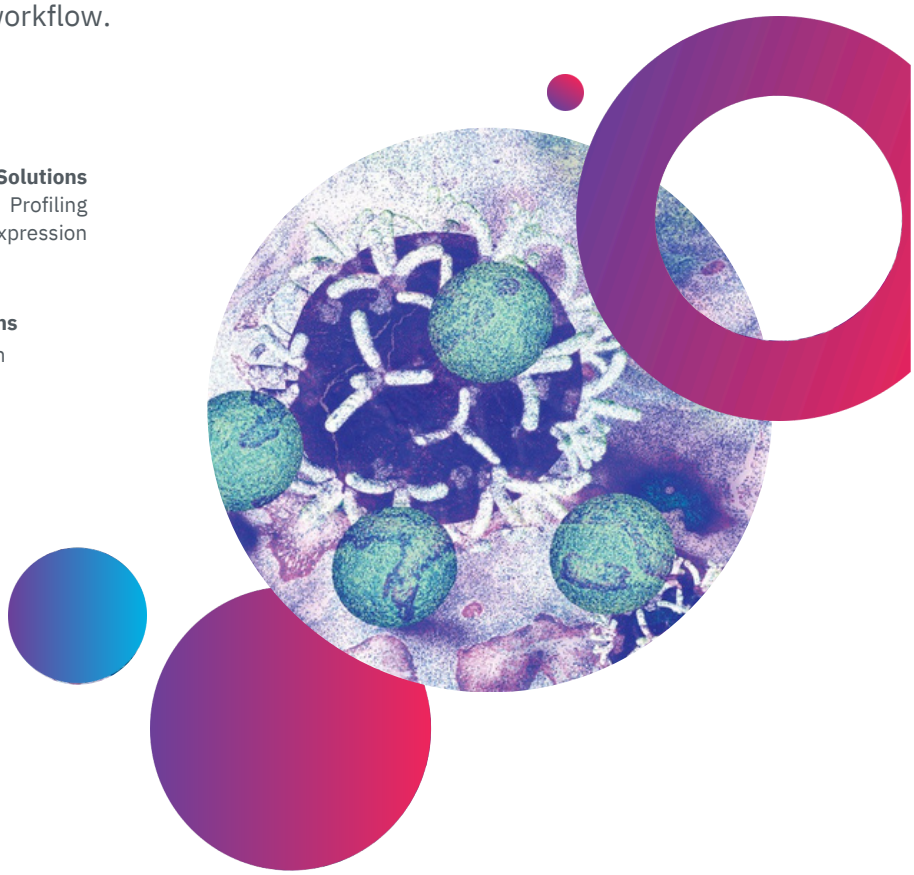
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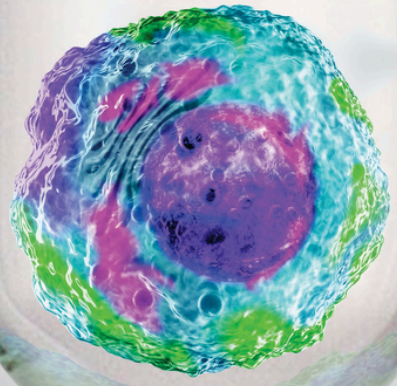
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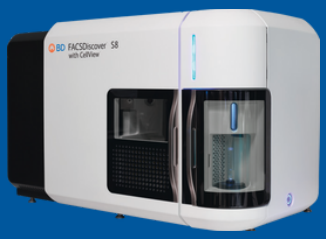


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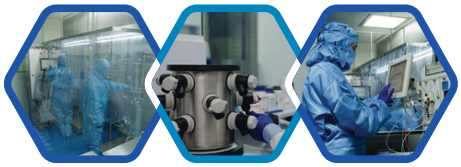
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