ABSTRACTS

33rd National Congress of Parasitology

26-28 November, 2025

CSIR-Central Drug Research Institute, Lucknow

This Souvenir and Abstract Book is published on the occasion of the "33rd National Congress of Parasitology on Recent Advances in Parasite Biology and Drug Discovery, November 26-28, 2025, Jointly organized by the CSIR-Central Drug Research Institute and The Indian Society for Parasitology at CSIR-Central Drug Research Institute, Sector 10, Jankipuram Extension, Lucknow, Uttar Pradesh 226031.

Editorial Board

33rd National Congress of Parasitology

on

Recent Advances in Parasite Biology and Drug Discovery

November 26-28, 2025

Jointly organized by

CSIR-Central Drug Research Institute
and
The Indian Society for Parasitology

Venue

CSIR-Central Drug Research Institute

Sector 10, Jankipuram Extension, Lucknow,
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The Indian Society for Parasitology

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Dr. Radha Rangarajan
Chairperson 33rd NCP and
Director, CSIR-CDRI

Message

It gives me immense pleasure to welcome all the delegates participating in the 33rd National Congress of Parasitology (NCP-2025), being organized by CSIR-Central Drug Research Institute (CSIR-CDRI), Lucknow in collaboration with the Indian Society of Parasitology (ISP). This year marks 75 years since CSIR-CDRI was established and 50 years since ISP was founded. In fact, the ISP itself traces its origins to CSIR-CDRI. Therefore, the 33rd NCP is a befitting occasion to mark both milestone anniversaries.

NCP-2025 brings together leading minds in the field to deliberate on the pressing challenges, emerging trends and innovative approaches in parasite biology and drug discovery. As parasitic diseases continue to pose significant global health concerns, such scientific congregations play a key role in the acceleration of product ideas and control strategies.

We are honoured to have over 300 delegates, including distinguished experts from India and abroad, participating in NCP-2025. The event will feature more than 41 plenary/invited lectures, 24 flash talks, and over 150 poster presentations, offering an excellent platform for learning, networking and discussion.

I am confident that the deliberations during this congress will not only enhance our collective understanding of parasitic diseases but also inspire new partnerships and translational outcomes contributing to global health priorities.

I congratulate the organizing committee for their dedicated efforts in bringing this event to fruition and wish all the participants a productive and enriching learning experience. I thank all the sponsors, CSIR laboratories and funding agencies for their generous support of NCP-2025.

(Radha Rangarajan)

Dr A M Khan,
President, ISP

Message

It is a matter of great pride and privilege to extend my warm greetings to all members, researchers, and well-wishers of the Indian Society for Parasitology (ISP) as we celebrate our Golden Jubilee Year. Established in 1973, the Society has, over the past five decades, emerged and served as a leading platform for promoting excellence in parasitological research, education, and public health in India. I extend my appreciations to the Director of CSIR-Central Drug Research Institute (CDRI), Lucknow and her team of scientists for joining us in the Golden Jubilee Celebration of ISP by organising 33rd National Congress of Parasitology at CDRI Lucknow which is also coinciding with Platinum Jubilee Celebration of CSIR-CDRI, Lucknow. The ISP recognizes that parasitology is well fitted in to the One Health framework, encompassing the health of humans, animals and environment, and our collective efforts will contribute to improving public health, poultry and livestock productivity. Contributions of ISP member parasitologists in the areas of research and intervention/ elimination of diseases like Malaria, Lymphatic Filariasis and Kala-azar are immense. Our mission also extends beyond laboratory walls. The ISP member-researchers actively support and conduct community-level initiatives through disease investigations, epidemiological studies, awareness campaigns, and health education programs pertaining to parasitic diseases. We are committed to translating scientific discoveries into practical solutions that directly benefit the people, their animals, and environment, while also generating basic knowledge of parasites and diseases caused by them.

In alignment with the Government of India's health and agricultural priorities and policies, the Society continues to support national strategies aimed at controlling neglected tropical diseases, zoonotic infections, enhancing veterinary health, and safeguarding environment. These efforts reflect our deep commitment to serving the nation through conducting studies on parasitic diseases. As we step into our next 50 years, we envision a future driven by collaboration, innovation, and compassion. Let us encourage young scientists, embrace interdisciplinary research, and harness modern tools—especially AI-based analytics, molecular diagnostics, and predictive modelling—to confront the evolving challenges of parasitic infections. During three days of conference (26-28 November 2025), scientists and researchers of diverse group will be interacting and sharing their experiences of research in the area of parasitology and I am confident that deliberations in this meeting will come up with innovative ideas and leads in conceptualising newer strategies for tackling of emerging/ re-emerging parasitic infections of national and international importance. On this historic occasion, I express my heartfelt appreciation to all past Presidents, Member-Secretaries, Office bearers, and members whose dedication and scholarship have strengthened the foundations of the Society. Together, let us continue to advance the frontiers of Parasitology and contribute meaningfully to a healthier, parasite-free world.

(A. M. Khan)

Dr. Satish Mishra
Secretary, ISP and Convener, 33rd NCP

Message

It is with great pleasure that I welcome all delegates to the 33rd National Congress of Parasitology (NCP). This year's congress holds special significance as we commemorate 75 years of CSIR-Central Drug Research Institute (CSIR-CDRI) and 50 years of the Indian Society for Parasitology (ISP). These dual milestones make this year's congress particularly momentous. This year's theme, "Recent Advances in Parasite Biology and Drug Discovery," reflects our focus on addressing critical challenges in parasitic diseases through cutting-edge research and collaboration. The congress is organized in association with the ISP, established in 1973. The Society has played a pivotal role in advancing parasitology research in India and fostering meaningful collaboration between academia and industry. Through its annual meetings and outreach, the ISP promotes innovation, dialogue, and knowledge-sharing across a wide spectrum of parasitological sciences. This event brings together eminent scientists, academicians, clinicians, young researchers, and students from across the country. It provides a platform for exchanging ideas, sharing research findings, and forging new collaborations. The scientific program includes keynote lectures, oral presentations, and poster sessions covering various topics such as host-parasite interactions, disease pathogenesis, immune evasion, drug discovery, vaccine development, biochemical pathways, drug resistance, and more. These sessions are designed to stimulate discussions that will shape the future of parasitology in India and beyond.

One of the key highlights of the congress is the recognition of outstanding contributions to the field. The ISP will confer several prestigious awards, including the Dr. B.N. Singh Memorial Oration Award, Dr. B.P. Pandey Memorial Oration Award, and Prof. Kona Hanumantha Rao Memorial Oration Award. To encourage emerging talent, awards like the Young Scientist Award, Prof. M.B. Mirza Award, and best oral/poster presentation awards will also be presented. Parasitic diseases continue to be a significant burden, particularly in resource-limited regions. With the emergence of drug resistance and evolving disease patterns, there is an urgent need for advanced diagnostic tools, innovative treatments, and collaborative research efforts. This congress provides an ideal forum to address these issues through meaningful dialogue, scientific exchange, and the strengthening of partnerships. I am confident that the interactions and deliberations during the congress will inspire new ideas, foster collaborations, and contribute significantly to the advancement of parasitology research. I extend my best wishes for a successful and enriching conference and thank all participants for their valuable contributions.

(Satish Mishra)

The organizing committee is grateful for financial and logistic support to 33rd NCP 2025 from

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33rd National Congress of Parasitology

on

Recent Advances in Parasite Biology and Drug Discovery

November 26-28, 2025

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Lucknow

Lucknow's history began in the Ramayana era as a city named Lakshmanpur, founded by Lord Rama's brother, Lakshman. It grew under various empires including the Mauryas, Guptas, and Mughals before becoming the capital of the Nawabs of Awadh in the 18th century, leading to a golden age of arts and culture. After being annexed by the British in 1856, it was a major site of the 1857 rebellion and later modernized under British rule. Following Indian independence in 1947, it became the capital of Uttar Pradesh.

Ancient origins and medieval rule

- According to legend, the city was founded by Lakshmana, brother of Lord Rama, and was originally known as Lakshmanpur or Lakshmanavati.
- During this time, it was part of the Koshal kingdom.
- The region later fell under the control of major empires such as the Mauryas, Shungas, Kushans, and Guptas.
- From the 14th to the 16th century, it was ruled by the Delhi Sultanate and the Sharki Sultanate of Jaunpur.

Mughal era and the Nawabs of Awadh

- Under the Mughal Empire, Lucknow grew as an important administrative and cultural center.
- The decline of the Mughal Empire led to the rise of the Nawabs of Awadh, who shifted their capital to Lucknow in 1775.
- This period is considered a golden age, with patronage for art, architecture (like the Bara Imambara), music, and a refined court culture.

British rule and the 1857 rebellion

- British influence increased in the late 18th century, leading to the annexation of Awadh in 1856, which caused widespread resentment.
- Lucknow was a focal point of the Indian Rebellion of 1857 and was site of a major siege.
- Following the rebellion, the British Crown took direct control, and the city was modernized with new infrastructure, railways, and roads.

Modern era

- Lucknow played a significant role in India's independence movement, including the Lucknow Pact of 1916 and the Kakori Conspiracy trials.

After India gained independence in 1947, Lucknow became the capital of Uttar Pradesh.

CSIR-CDRI, Lucknow

The Central Drug Research Institute (CDRI) is a constituent laboratory of the Council of Scientific and Industrial Research (CSIR) under the Department of Scientific & Industrial Research, Government of India, Ministry of Science and Technology, Government of India. The Institute is engaged in the development of drugs, diagnostics and process technologies in disease areas of national relevance to enable affordable healthcare. Since its inception in 1951, CDRI has made significant advancements in its mission, with 13 drugs and more than 80 process technologies licensed to industry. CSIR-CDRI is a unique biomedical research centre with strengths in basic and applied research. There are eight therapeutic areas in the Institute: Microbial Infections, Parasitic Infections, Viral Infections, Cancer, Metabolic Disorders, Bone and musculoskeletal disorders, Neurological Disorders and Reproductive Health. Scientists are engaged in cutting edge fundamental research, aimed at dissecting cellular, molecular and chemical processes underlying pathological conditions. In addition, scientists have access to the full gamut of expertise in drug discovery and early clinical development, including medicinal chemistry, pharmacology, pharmacokinetics and toxicology. This allows for a seamless translation from basic research into application. The Institute's sizeable publication and patent portfolio, reflects this blend of research capabilities.

Indian Society for Parasitology

The Indian Society for Parasitology, a registered scientific society established in 1973 is affiliated with World Federation of Parasitologists and has its headquarters at the Central Drug Research Institute (CDRI), Lucknow, India. The Society comprises of parasitologists, epidemiologists, clinicians, biologists and academicians working in various areas of Parasitology related to humans and other animals. The Society facilitates exchange and dissemination of knowledge as well as promotes their research and development activities in parasitology and tropical medicine research. The Society publishes the Journal of Parasitic Disease in association with Springer, a leading publisher of scientific, technical and medical research work. It also organises scientific meetings, which act a platform for exchange of ideas on various areas of parasitology and allied areas.

33rd National Congress of Parasitology

On

"Recent Advances in Parasite Biology and Drug Discovery"

26-28 November, 2025

Program

Day 1 - Wednesday, 26 November, 2025					
07:30	Breakfast	Venue: CSIR-CDRI Lawn			
08:00	Registration	Venue: CSIR-CDRI Auditorium Complex			
Session Co-ordinator: Dr. Mrigank Srivastava					
09:00	Inauguration and Release of Abstract Book				
09:30	Welcome Address	Dr. Radha Rangarajan, Chairperson 33rd NCP and Director, CSIR-CDRI			
09:40	Address	Dr. A.M. Khan, President, ISP			
09:45	Address	Dr. Satish Mishra, Secretary, ISP and Convener 33rd NCP			
Inaugural Session Venue: Auditorium **Chair: Dr. Radha Rangarajan, Co-Chair: Dr. Saman Habib**					
09:50	Inaugural Lecture	Prof. NK Ganguly, Former DG, ICMR			
10:30	Special Talk	Dr. Bhupendra Tripathi, Gates Foundation, New Delhi			
10:45	Tea break	Venue: CSIR-CDRI Lawn			
Session I Venue: Auditorium **Biochemistry & Cell Biology of Parasites** **Chair: Dr. Pawan Malhotra, Co-Chair: Dr. Swati Patankar** **Session Co-ordinator: Ms. Ayushi Singh**					
11:20	IL 1	Dr. Hemalatha Balaram, JNCASR, Bangalore	12:20	IL 3	Prof. Mrinal Bhattacharya, University of Hyderabad, Hyderabad
11:50	IL 2	Prof. Suman Kumar Dhar, TERI, New Delhi	12:40	IL 4	Dr. Kalyaneswar Mandal, TIFR, Hyderabad
13:00	Lunch	Venue: CSIR-CDRI Lawn			
13:00	**Poster Session**	Venue: CSIR-CDRI Lawn			
	EC meeting	Venue: Hall 3			

Session II

Venue: Auditorium

Drug Discovery & Translational Research for Parasitic Diseases

Chair: Dr. Hemalatha Balaram, Co-Chair: Prof. Suman Kumar Dhar, Session Co-ordinator: Mr. Suryansh Rajput

15:00	IL 5	Dr. Pawan Malhotra, ICGEB, New Delhi	16:20	IL 8	Dr. Puran Sijwali, CCMB, Hyderabad
15:30	IL 6	Dr. Swati Patankar, IIT, Bombay	16:40	IL 9	Dr. Shailja Singh, JNU, New Delhi
16:00	IL 7	Dr. V. Arun Nagaraj, ILS, Bhubaneswar			
17:00	Tea break	Venue: CSIR-CDRI Lawn			

Session III			**Session IV (Parallel)**		
Immunobiology of Parasitic Infections			**Biology of Parasitic Infections**		
Venue: Auditorium			Venue: Hall-2		
Chair: Prof. Mrinal Bhattacharya, Co-Chair: Dr. Kalyaneswar			**Chair: Prof. Sukhbir Kaur, Co-Chair: Dr. Amit Prasad**		
Session Co-ordinator: Ms. Pragya Mehra			**Session Co-ordinator: Ms. Shikha Yadav**		
17:20	IL 10	Prof. Syamal Roy, IACS, Kolkata	17:20	IL 14	Dr. Tanmay Majumdar, NII, New Delhi
17:50	IL 11	Dr. Susanta Kar, IICB, Kolkata	17:40	IL 15	Dr. Abhisheka Bansal, JNU, New Delhi
18:10	IL 12	Dr. Pankaj Sharma, NCCS, Pune	18:00	PT 2	Dr. Souvik Bhattacharjee, JNU, New Delhi
18:30	IL 13	Dr. Suprabhat Mukherjee, KNU, Asansol	18:20	PT 3	Dr. Sandeep Kumar Malhotra, AU, Prayagraj
18:50	PT 1	Dr. Mradul Mohan, NIMR, New Delhi	18:40	PT 4	Prof. Biplob Kumar Modak, SKBU, Purulia

Session V

Session Co-ordinator: Ms. Ayushi Singh

19:00 **12X5 Min Flash Talk Series-1 by Selected Young Scientists**

Venue: Auditorium

20:00 Dinner — Venue: CSIR-CDRI Lawn

Day 2 - Thursday, 27 November, 2025

07:30 Breakfast — Venue: CSIR-CDRI Lawn

Session VI

Venue: Auditorium

Drug Discovery & Translational Research for Parasitic Diseases

Chair: Prof. Syamal Roy Co-Chair: Dr. Krishna Pandey, Session Co-ordinator: Ms. Eisha Pandey

09:00	Plenary Lecture 1	Dr. Jeremy Burrows, MMV, Genève	10:00	IL 17	Dr. Pushkar Sharma, NII, New Delhi
09:30	IL 16	Dr. Saman Habib, CSIR-CDRI, Lucknow	10:30	IL 18	Dr. Asif Mohammed, ICGEB, New Delhi

11:00	Tea break	Venue: CSIR-CDRI Lawn			
11:20	IL 19	Prof. Vishal Trivedi, IIT, Guwahati	11:40	IL 20	Dr. Sunanda Bhattacharyya, University of Hyderabad, Hyderabad

Session VII

Session Co-ordinator: Ms. Ayushi Singh

12:00	**12X5 Min Flash Talk Series-2 by Selected Young Scientists**	
	Venue: Auditorium	
13:00	Lunch	Venue: CSIR-CDRI Lawn
13:00	Poster Session	Venue: CSIR-CDRI Lawn

Session Co-ordinator: Mr. Nirdosh & Ms. Samata Chowdhury

14:00	Parallel Session Young Scientist Award	Venue: Hall 2

Session VIII			**Session IX (Parallel)**		
Fundamental and Translational Parasite Biology			**Biochemistry & Cell Biology of Parasites**		
Venue: Auditorium			Venue: Hall-2		
Chair: Dr. Asif Mohammed, Co-Chair: Prof. Vishal Trivedi			**Chair: Dr. Puran Sijwali, Co-Chair: Dr. Sunanda Bhattacharyya**		
Session Co-ordinator: Ms. Samata Chowdhury			**Session Co-ordinator: Ms. Shweta Shinde**		
15:00	IL 21	Dr Krishna Pandey, RMRIMS, Patna	15:00	IL 25	Dr. Pradeep Das, NICED, Kolkata
15:30	IL 22	Dr. Dinesh Gupta, ICGEB, New Delhi	15:20	IL 26	Dr. Ambak Kumar Rai, MNNIT, Prayagraj
15:50	IL 23	Dr. Saraboji Kadhirvel, CUP, Punjab	15:40	IL 27	Dr. Arumugam Rajavelu, IIT Madras
16:10	IL 24	Dr. Abhik Sen, RMRIMS, Patna	16:00	PT 6	Dr. Anchal Singh, BHU, Varanasi
16:30	PT 5	Dr. Ankit Gupta, AIIMS, Raebareli	16:20	PT 7	Dr. Tarun Kumar Bhatt, Central University of Rajasthan
			16:30	PT 8	Dr. Shruthi S Vembar, IBAB, Bengaluru
16:40	Tea break	Venue: CSIR-CDRI Lawn			

Session X		**Session XI (Parallel)**		
Oration Award		**Techno-Commercial Presentation**		
Venue: Hall-2		Venue: Hall-3		
(Moderator: Dr. Satish Mishra)		**(Moderator: Ms. Eisha Pandey)**		
17:00	Dr. B.N. Singh Memorial Oration Award Dr. Krishanpal Karmodiya, IISER Pune	17:00	TC 1	Premas Life Sciences
		17:20	TC 2	Strand Life Sciences
17:30	Dr B.P. Pandey Memorial Oration Award Prof. Sukhbir Kaur, Panjab University, Chandigarh	17:40	TC 3	New England Biolabs
		18:00	TC 4	Merck
18:00	Dr. K. Hanumantha Rao Memorial Oration Award Prof. Harpreet Kaur, Panjab University, Chandigarh	18:10	TC 5	DSS Takara
		18:20	TC6	Corning
18:30	General Body Meeting	Venue: Hall 2		

19:00	**Cultural Program by Mr. Abhishek Borkar and Mr. Yashwant Vaishnav** Venue: Auditorium	

20:30	Gala Dinner	Venue: CSIR-CDRI Lawn

Day 3 – Friday, 28 November, 2025

07:30	Breakfast	Venue: CSIR-CDRI Lawn

Parasite, Vectors and Veterinary Parasitology

09:00	Plenary Lecture 2	Dr. Anup Anvikar NIMR, New Delhi

Session XII Venue: Auditorium **Chair: Dr. Anuradha Dube** **Co-Chair: Dr. Sanjay Batra** **Session Co-ordinator: Ms. Pragya Mehra**			Session XIII (Parallel) Venue: Hall-2 **Chair: Dr. A.M. Khan** **Co-Chair: Shailja Bhattacharya** **Session Co-ordinator: Ms. Payel Acherjee**		
09:40	IL 28	Dr. Aparup Das RMRC, Port Blair	09:40	IL 31	Dr. Anand Srivastava NIAB, Hyderabad
10:05	IL 29	Dr. S.L. Hoti VCRC, Puducherry	10:00	IL 32	Dr. Dhanasekaran Shanmugam NCL, Pune
10:30	IL 30	Dr. Amit Prasad IIT, Mandi	10:20	IL 33	Dr. Abhijit S. Deshmukh NIAB, Hyderabad
10:55	PT 9	Dr. Vahab Ali RMRIMS, Patna	10:40	PT 10	Dr. Somnath Waghmare NWC, Pune

11:15	Tea Break	Venue: CSIR-CDRI Lawn
11:40	**Panel discussion** **Theme: Exploring new technologies in combating parasitic diseases.** **Dr. Saman Habib (Moderator), Dr. Swati Patankar, Prof. Syamal Roy, Dr. Anup Anvikar, Dr. Jeremy Burrows, Dr. Bhupendra Tripathi** **Session Co-ordinator: Ms. Ayushi Singh**	
12:40	**Celebrating 75 Years of Parasitology Research at CSIR-CDRI** Moderator: Dr. Saman Habib	
13:20	**Valedictory Function** Venue: Auditorium **Session Co-ordinator: Dr. Aamir Nazir**	
14:00	Lunch and Adieu!	Venue: CSIR-CDRI Lawn

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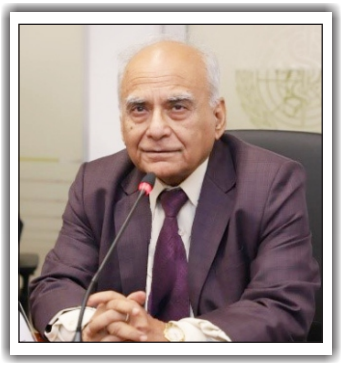

Prof. Nirmal Kumar Ganguly
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Parasitic Diseases Which Have Reached Elimination And Science Behind That

Prof. Nirmal Kumar Ganguly, MD is a distinguished Indian medical scientist and global health expert with extensive contributions to research, public health, and biotechnology. He has held several prominent positions in India, including Director-General of the Indian Council of Medical Research (ICMR), Director of PGIMER (Chandigarh), and Director of the National Institute of Biologicals (Noida). He has also served as a Distinguished Biotechnology Research Professor with the Department of Biotechnology, Government of India, and as President of JIPMER and the Asian Institute of Public Health, Odisha. Prof. Ganguly has been actively involved in numerous international advisory and scientific bodies. He is a member of the Cholera Vaccine Investment Case Advisory Group at the International Vaccine Institute (South Korea), and serves on advisory boards and scientific committees of institutions like the University of Oxford, Boston University, University of Minnesota, WHO, CDC (USA), and Sanofi Pasteur. He is also associated with global initiatives such as Grand Challenges (Canada and Gates Foundation) and the Worldwide Antimalarial Resistance Network. Academically, he is an adjunct professor at Boston University and the University of Minnesota, and serves on the editorial board of Molecular and Cellular Biochemistry. His research focuses on tropical diseases, cardiovascular and diarrhoeal diseases, immunology, and public health. He has published over 775 papers and supervised 130 Ph.D. theses. Prof. Ganguly is a fellow of several prestigious academies including the Royal College of Pathologists (London), Imperial College Faculty of Medicine (London), TWAS (Italy), and the Indian National Science Academy. He has also led numerous Indian medical and scientific societies. His career reflects a deep commitment to advancing global health, biomedical research, and capacity-building in public health systems.

Plenary Lecture 1

Dr. Jeremy Burrows
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Antimalarial Drug Discovery: Next Generation Antimalarials

Falciparum malaria, a devastating parasitic disease affecting millions of people each year, is treated using fixed dose combination treatments, the Artemisinin Combination Therapies (ACTs) over 3 days of dosing. The constant threat of resistance and the need to deliver alternative options to treat patients in the event of all ACTs failing, as well as the need for improved drugs against other human-infecting *Plasmodium* parasites, has led to Medicines for Malaria Venture (MMV) and its partners, in collaboration, to build up a portfolio of projects and compounds focused on the treatment and prevention of malaria. MMV's mission is to reduce the burden of malaria in disease-endemic countries by discovering, developing and facilitating delivery of new, effective and affordable antimalarial drugs in collaboration with international partners.

MMV manages a significant antimalarial pipeline, and this has been strengthened in recent years with the delivery of new products, new clinical candidates and early-stage discovery projects. The talk will outline the progress made, against the ever present challenge of resistance emergence to frontline therapies, and the complexities required to deliver impactful products for control and elimination. In particular, our focus is on delivering differentiated candidate drugs with long human half-lives and duration of cover either from oral or intra-muscular dosing. In this context, new thinking and modes of working, such as optimizing specifically to reduce the resistance risk, utilizing unbound pharmacokinetic and pharmacodynamic parameters to better design and estimate dose and machine learning opportunities will also be highlighted along with specific candidate drug case studies.

Plenary Lecture 2

Dr. Anup Anvikar
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Next-Generation Tools for Malaria Control: From Parasite Biology to Precision Public Health

The fight against malaria is at a critical crossroads, with progress threatened by evolving drug resistance, insecticide tolerant vectors, and persistent transmission in endemic regions. To overcome these challenges, the next generation of malaria control demands a transformative toolbox built on fundamental discoveries in parasite biology, advanced technologies, and precision public health strategies.

Key insights into the biology of *Plasmodium* species, including liver-stage dormancy, gametocyte maturation, and epigenetic regulation are shaping new therapeutic and transmission blocking interventions. Genomic surveillance and multi-omics platforms are enabling real-time tracking of drug resistance, vector evolution, and parasite population structure. Tools such as CRISPR-based functional genomics, artificial intelligence driven drug discovery, and high throughput phenotypic screening are accelerating the development of novel antimalarials. Special emphasis should be placed on the mechanisms of artemisinin and partner drug resistance, genomic surveillance frameworks, and innovative strategies to outpace parasite evolution, including triple drug therapies, and host-directed therapeutics.

Beyond chemotherapy, next-generation tools also include gene-drive mosquitoes, long-acting monoclonal antibodies, mRNA and multistage vaccines, digital disease surveillance, and AI-supported early warning systems. The integration of these innovations into public health systems through precision targeting, regional collaboration, and policy alignment will be essential for sustainable malaria elimination. By bridging molecular science with field-ready innovation, this session presents a roadmap for future malaria control that is smarter, faster, and resistant to emerging threats.

Special Talk
Dr. Bhupendra Tripathi
Deputy Director
Gates Foundation – India Country Office, New Delhi
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India's Progress Towards Elimination of Visceral Leishmaniasis and Lymphatic Filariasis – Last Mile Challenges

Dr. Bhupendra Tripathi leads the foundation's infectious disease and vaccine delivery efforts in India and is based in the India Country Office. He and his team work toward the elimination of infectious diseases such as tuberculosis, visceral leishmaniasis, lymphatic filariasis, malaria, and measles in India and work to improve immunization coverage and equity. Before joining the foundation in 2014, Bhupendra held positions at the World Health Organization, UNICEF, and JSI, working on polio eradication, measles elimination campaigns, routine immunization, and programs to improve reproductive, maternal, newborn, child, and adolescent health and nutrition. Bhupendra earned his medical degree and a post-graduate degree in public health in India. He also completed advanced courses at the London School of Hygiene & Tropical Medicine and the University of Geneva. He is a board member of the American Society of Tropical Medicine and Hygiene and a lifetime member of Indian Association of Preventive and Social Medicine.

Invited Lectures

IL 1

Dr. Hemalatha Balaram
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Unraveling TCA Cycle – Linked Metabolism in the Malaria Parasite *Plasmodium*

Malaria, caused by Plasmodium spp., remains a public health burden in many developing countries. This challenge is further exacerbated by the emergence of drug-resistant parasite strains and the lack of a fully effective vaccine. The Plasmodium parasite requires two distinct hosts—an invertebrate mosquito and a vertebrate—to complete its complex life cycle. As the parasite transitions through different developmental stages within these hosts, it encounters varying nutrient environments, necessitating metabolic adaptations. Metabolism, comprising interconnected anabolic and catabolic pathways, is a fundamental and evolutionarily conserved feature of all life forms. Our understanding of parasite metabolism has evolved from studying isolated enzymes and metabolites to examining the dynamic networks that sustain parasite survival and development. In my talk, I will present our recent findings on the role of tricarboxylic acid (TCA) cycle anaplerosis in *P. berghei*, a rodent model of malaria. Using a combination of genetic knockouts and isotope tracing through LC-MS, we have examined the essentiality of specific enzymes and transporters involved in TCA cycle function across asexual erythrocytic stages and early sexual development. These insights contribute to a broader understanding of metabolic variability across stages in Plasmodium and could highlight new possibilities for therapeutic intervention.

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Studies on Non-Replication Functional Role of *Plasmodium falciparum* Proliferating Cell Nuclear Antigen 2 (*Pf*PCNA2)

In its intricate life cycle alternating between humans and mosquitoes, *Plasmodium* parasites must go through at least four rounds of replication. DNA replication is a highly coordinated process involving several proteins and enzymes, one of which is proliferating cell nuclear antigen (PCNA). In most eukaryotes, there is only one gene that encodes PCNA although there are exceptions. Archaeans like *Sulfolobus solfataricus, Arabidopsis thaliana, Drosophila melanogaster, Toxoplasma gondii* and *Plasmodium sp,* all have a second copy of PCNA (PCNA2). We have shown earlier that *Plasmodium falciparum* PCNA1 (PfPCNA1) is mostly nuclear with its function in DNA replication DNA damage response while PfPCNA2 is both nuclear and cytoplasmic. While nuclear PfPCNA2 is involved in DNA damage response, the role of cytoplasmic PfPCNA2 is largely unknown. Interestingly, LC/MS analysis of PfPCNA2 immunoprecipitated proteins show specific interaction of PfPCNA2 but not PfPCNA1 with a unique bifunctional enzyme called GluPho (glucose-6-phosphate dehydrogenase-6-phosphogluconolactonase), a fusion protein consisting first two enzymes of Pentose Phosphate Pathway as mentioned above. The details of interaction of PfPCNA2 with GluPho leading to enhancement of G6PD activity and its implication for parasite biology will be discussed. Since PPP generates NADPH required for neutralizing oxidative stress encountered by the parasites during its life cycle in different hosts, the interaction of PfPCNA2 with GluPho and modulation of the activity of the key enzyme of PPP not only highlights the cytoplasmic function of PfPCNA2, it offers excellent opportunity to establish PfPCNA2 as a potential drug target that is absent in the human host.

IL 3

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The Yin and Yang of *Plasmodium* Recombination

Homologous recombination between two unrelated genomic loci must be prohibited in order to maintaining genomic integrity. On the other hand, recombination between the same allele coming form the two parents must be allowed despite the variations in their sequences. Thus, the cellular recombination machinery must be regulated at different levels in order to allow or disallow recombination between two DNA substrates. Here we describe two such levels of regulations: one that negatively regulates recombination and the other that inhibits such negative-regulator and thus ultimately promoting recombination. The consequences of such intricate interplay between the positive and negative regulators on the outcome of mitotic and meiotic recombination will be elucidated in the context of *Plasmodium* biology.

IL 4

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Engineering Biomolecules to Outsmart Malaria

Malaria parasites claim over half a million lives annually. A critical step in their invasion of human red blood cells is the interaction between two parasite proteins, AMA1 and RON2, which drives moving junction formation. Disrupting this interaction offers a powerful strategy to block invasion. In this talk, I will present how we are engineering biomolecules to outsmart malaria by combining chemical peptide and protein engineering with mirror-image biological display. Using these approaches, we design peptides that inhibit AMA1–RON2 binding and identify mirror-image protein (D-protein) inhibitors that resist proteolysis and show reduced immunogenicity. Such engineered D-proteins hold strong promise as next-generation antimalarial therapeutics.

IL 5

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Plasmodium falciparum Secretome/Surfactome at Asexual Blood Stages – A Hidden Treasure for Understanding Parasite Survival, Development, Invasion and Cytoadherence

Cells talk to each other and to their respective host cells in case of pathogens by cell surface/secretory molecules. Secretory proteins from all cells make up a complex subset of molecules referred as "secretome" and cell surface molecules are referred as surfactome. These secretory/surface proteins play important roles in cell to cell communication, host cell invasion and in case of vertebrates these proteins help in keeping a delicate balance between protective immunity of the host and pathogenic immune evasion responses thereby mainitaining homeostasis and eliminating the the infectious probes. Here, we describe the proteomic approaches to illustrate *Plasmodium falciparum* "secretome" as well as surfactome in an *"in vitro"* cultured parasites as well as from isolated merozoites as well as trophozoites . In total 33 proteins were identified from *P. falciparum* secretome and sequence analysis of many of these putative *Plasmodium* secretory antigens revealed that they possessed important extracellular binding/signalling modules required for parasite establishment, pathogenesis and immunomodulation. Important among these proteins are the proteins that share homology with the *Caenorhabiditis elegans* Sel 1 protein, an extracellular protein shown to be shown to be a negative regulator of Notch Pathway *genes ;lin-12* and *glp-1* referred here as *Pf*Sel1 & *Pf*Sel 2 proteins, . Another set of proteins that we identified are the Complement control protein (CCP1) that modulates complement activation, a putative serine protease (DegP), a protein kinase, a tyrosine phosphatase and GBP-130 that are involved in pathogenesis as well virulence. Likewise, we could identify more than 400 proteins on merozoite surface including the 20S proteosome machinery, M17 Leucyl aminopeptidase, M18 aspartyl aminopeptidase and most of the RBCs invasion related proteins. In addition, we have also illustrated the surfactome of infected erythrocytes at trophozoite stage of mild and severe malaria parasites to identify the proteins that are overexpressed in severe malaria parasites. Many of these aspects will be discussed in detail in my talk

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Targeting Nuclear Import for Therapeutics Against Multiple Stages of Apicomplexan Parasites

Apicomplexan parasites, *Plasmodium falciparum* and *Toxoplasma gondii*, cause widespread human disease. Although treatments exist, these drugs do not target multiple stages of the two parasites and are succumbing to drug resistance. New therapeutic pipelines are essential. We have shown proof-of-concept that the nuclear transporter, importin alpha, is a novel drug target. We identified small molecules that inhibit importin alpha function *in vitro* and in parasite cultures. We also found that a short peptide that contains a viral nuclear localization signal (NLS) greatly reduces parasite viability by targeting importin alpha. These biomolecules act upon multiple stages of the parasites, bringing hope for novel therapeutic strategies in the future.

IL 7

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Metabolic Adaptations in the Malaria Parasite – The Paradigm of Take it or Make it

Malaria parasite has evolved with extensive metabolic adaptations to successfully replicate and survive in the human and mosquito hosts. Such adaptations help the parasite to counter the nutrient scarcity, and modulate the disease outcome and transmission. The typical examples include the switching between glycolysis and oxidative phosphorylation, the existence of *de novo* and salvage pathways for key metabolites, and the presence of various transport mechanisms to exploit the host nutrient resources. A detailed understanding of such metabolic flexibilities would offer insights on the adaptation of parasites to diverse host niches, their fitness and transmission potential, and the mechanisms underlying disease pathogenesis. In this talk, I will address the paradigm of "take it" or "make it" that exists in the malaria parasite. The two examples of heme and glutamine synthesis, and their physiological relevance in disease pathogenesis and parasite survival will be discussed. The importance of comprehending such dynamic aspects of metabolic adaptations will be emphasized in the context of developing new therapeutic intervention strategies for malaria.

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An ER-Resident Lipid Scramblase is Crucial for Biogenesis and Function of Apicomplexan Parasite Secretory Organelles

DedA superfamily proteins are implicated in lipid distribution and organelle biogenesis, and has members in all the life forms. The vacuole membrane protein 1 (VMP1) is *the best* studied DedA superfamily member. Apicomplexan parasites, including *Plasmodium falciparum* and *Toxoplasma gondii*, contain specialized secretory organelles such as micronemes, rhoptries, and dense granules, which are essential for parasite motility, host cell invasion, development, and egress. *T. gondii* and *P. falciparum* contain multiple DedA superfamily proteins. Since DedA superfamily proteins are yet to be reported in parasitic protozoans and VMP1 homologs have crucial roles in several processes, including organelle biogenesis, we identified and investigated the vacuole membrane protein 1 (VMP1) of *P. falciparum* (PfVMP1) and *T. gondii* (TgVMP1). PfVMP1 and TgVMP1 are ER-localized lipid scramblases. TgVMP1 depletion adversely affected parasite development, motility, host cell invasion, and egress. These phenotypes were consistent with impaired rhoptry and dense granule biogenesis, and decreased secretion of micronemes and rhoptries in TgVMP1-depleted parasites, indicating a crucial role for TgVMP1 in the biogenesis and function of these organelles. TgVMP1 depletion impaired lipid droplet homeostasis, ER organization, and intravacuolar network formation. Restoration of the ER-localized lipid scramblase by complementing TgVMP1-depleted parasites with PfVMP1 or a homolog as distant as human VMP1 rescued the depleted parasites, indicating their functional conservation and a crucial role for ER-resident lipid scramblase activity in the biogenesis and function of secretory organelles. The essentiality of TgVMP1 for parasite development and likely functional conservation of apicomplexan VMP1 proteins highlight their drug-target potential.

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Chemical Degradation Identified Human P38-MAPK as a Host Target to Combat Parasitic Infection

The interplay between host and parasite determines parasite burden and disease outcome. Parasite exploits host signaling pathways like p38-MAPK for its survival and pathogenesis. NR-7h, a proteolysis-targeting chimera (PROTAC) targeting human p38-MAPK was used to assess p38-MAPK role in *Leishmania donovani* and *Plasmodium falciparum* infection in their respective hosts. NR-7h degraded host p38-MAPK in a time- and dose-dependent manner. Degradation of host p38-MAPK by NR-7h reduced parasite load in host cells dose-dependently, implicating the role of p38-MAPK in parasite survival. The modulation of cytokine profiling and oxidative burst upon NR-7h mediated degradation of host p38-MAPK was further correlated with parasite death. The synergistic effect of host p38-MAPK degradation by NR-7h with Amphotericin B enhances the efficacy of parasite-directed therapy. This study underscores the importance of host p38-MAPK for *L. donovani* and *P. falciparum* progression and highlights NR-7hpotential in antiparasitic therapy by targeting this pathway.

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Kinetoplastid Membrane Protein-11 (KMP-11) as a New 'Virulence Factor' for *Leishmania* Infection

The protozoan parasite, *Leishmania donovani* (LD) causes visceral leishmaniasis (VL) or kalaazar. LD replicates with in the phagocytic cells or macrophages (MØ) and causes wide variety of pathetical consequences. Unless patients are treated appropriately, it is always fatal. The mechanism of LD entry into MØ is still far from clear. The LD surface is studded with a protein, Kinetoplastid membrane protein-11 (KMP-11) which through its high affinity with LD-ergosterol maintains bilayer pressure of leishmania parasites. Oddly enough, KMP-11 showed very high affinity for cholesterol which is rich in the mammalian cells. Taking together led us to believe that LD-KMP-11 may favor their docking onto MØ surface. Indeed, we showed MØ lacking cholesterol failed to support LD entry. Similarly, LD-KMP-11$^{-/-}$ failed to gain access into MØ. We showed that KMP-11 forms oligomer that created a bridge between LD and MØ, causing membrane phase transition and facilitate LD entry. Further mapping of KMP-11 spanning the sequence, we showed that N-terminal sequence is critical for the entry process. To our knowledge, this is yet another important role of KMP-11 "as a new virulence factor" in VL pathogenesis.

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Dissecting the Role of Protein Arginine Methyltransferases (PRMTs) in the Alternative Activation Mediated Immunosuppression During Experimental Visceral Leishmaniasis

In mammalian cells, arginine methylation is a frequent post-translational modification that occurs on both histone and non-histone proteins and bears implications in the pathophysiological regulation of several biological processes, including DNA damage response, RNA splicing, metabolism and more recently, inflammation and immunity. The key enzymes that catalyse the synthesis of methylarginines in proteins are known as protein arginine methyltransferases (PRMTs). In a disease like leishmaniasis, where pathogenesis heavily banks on immune diversion, the role of PRMTs in the impairment of *Leishmania*-induced macrophage function remains obscure. In the same vein, given that chromatin remodelling by PRMTs is a mechanistic prerequisite for the acquisition of M2/anti-inflammatory phenotype, however its relevance in context of experimental VL is poorly understood. We observe preferential upregulation of PRMT1 amongst the type I PRMTs and functional knockdown of PRMT1 is met with removal of activatory H4R3me2a marks along with a concomitant decrease in expression of key M2 marker genes (MRC1, PPAR-γ and ARG1) in THP-1 derived macrophages. Furthermore, investigation of upstream signalling events leading to PRMT1 induction during *L. donovani* infection unravels the involvement of cAMP/p-PKA/p-CREB axis at the early phase and TGF-β signalling at the late phase, which culminates with an increase in C/EBPβ expression and its nuclear translocation, which then binds onto the PRMT1 promoter, facilitating its expression in host. We further uncover a role for *L. donovani* LPG in promoting PGE2 release from infected macrophages, thereby leading to CREB-mediated induction of PRMT1 in the early-phase. Besides epigenetic modifications, PRMT1 is also found to confer activatory ADMA marks on two M2-associated transcription factors- c-Myc and STAT6, thereby increasing their DNA binding activity on the promoter regions of their cognate genes- MRC1 and ARG1, respectively. Collectively, our findings uncover a dual role for PRMT1 in promoting transcriptionally permissive chromatin by deposition of H4R3me2a marks on promoter regions of M2 marker genes, as well as activatory post-translational methylations on transcription factors which may ultimately contribute to an immunosuppressive milieu and disease progression in the long run.

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Globotriaosylceramide (Gb3) Functions as an Intrinsic Adjuvant to Elicit Broadly Protective Antibody Responses Against Pathogens

Developing vaccines against parasitic pathogens remains a major challenge due to their complex life cycles, extensive antigenic diversity, stage-specific antigen expression, and sophisticated mechanisms of immune evasion. Protective humoral immunity must therefore generate antibodies that are both high in affinity and broad in reactivity across distinct parasite stages. This refinement of antibody responses occurs within germinal centers (GCs), yet the molecular determinants that govern B cell selection remain incompletely understood. Our work reveals that the glycosphingolipid globotriaosylceramide (Gb3) regulates multiple layers of GC B cell function. We show that Gb3 disengages CD19 from its chaperone CD81, enabling CD19 to associate with the B cell receptor (BCR) complex, thereby amplifying downstream signaling essential for effective clonal selection. Furthermore, Gb3 increases MHC class II expression on GC B cells, augmenting their antigen-presenting capacity and expanding the spectrum of peptides recognized by T follicular helper (Tfh) cells. This facilitates the expansion of diverse Tfh populations and enhances support for B cells specific to subdominant epitopes. As a result, selected antibodies exhibit increased diversity and longevity. Moreover, exogenous Gb3 acts as a vaccine adjuvant in mouse models and elicits cross-protective immunity. Thus, this work identifies a previously unrecognized function of Gb3 in coordinating B cell signaling and antigen presentation, thereby shaping the depth and breadth of humoral immunity, with direct relevance for engineering vaccines that elicit durable, high-affinity, and broadly reactive antibody responses against parasitic diseases.

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Exploring New Targets for Therapeutic Intervention of Lymphatic Filariasis

Lymphatic filariasis (LF) caused by the nematode parasite *Wuchereria bancrofti*, *Brugia malayi* and *B. timori*, is considered to be the world's second most leading cause of long-term disability. India remains a major contributor to the global burden of this disease and increasing new cases of LF is being reported till date, especially after COVID-19 pandemic. Especially a cross-sectional study undertaken by my research group between 2021-2024 revealed a notably high prevalence 10.63% of LF in and around the four coal-mining districts of West Bengal, India. In this context, my research throughout the last decade aimed at identifying new therapeutic target(s) from the parasite as well as the host to facilitate the adoption of accurate parasite-directed and/or host-directed therapeutic strategy. Bestrophin-9 homolog (an immunoreactive surface protein) and intracellular steroid hormone receptor (ISHBR, a regulator of worm molting) were purified from *W. bancrofti* microfilariae (larval stage) through bioactivity guided chromatographic purification and sequenced through MALDI-ToF-MS/MS. Efficacy of Betrophin-9 and ISHBR as therapeutic targets for developing anti-filarial agents were examined by enzyme-linked immunosorbent assay, immunoblotting, flow cytometry, and surface plasmon resonance analysis. It was found Bestrophin-9 as a 70 kDa phosphorylcholine-binding antigen that is present on the sheath (outer covering) of *W. bancrofti* and it binds to the human Toll-like receptor 4 (TLR4) of both macrophages and dendritic cells to release the pro-inflammatory cytokines. Whilst ISHBR was purified from the somatic fraction that appeared as a 43 kDa protein and it was found to interact with the dafachronic acid. The studies attested the candidacy of these two filarial proteins as targets developing antifilarial agents using the chemical library compounds as well as synthetic nature-inspired fused scaffolds. Additionally, anti-MfP serum/antibody was also examined for its protective as well as efficacy as both protective and prophylactic agent. More recently, host TNFR1 (both soluble and membrane bound) has been found as another possible mediators of immunopathogenesis in LF and hence development of chemotherapeutics from the natural sources has been emphasized. Given their immense impact in filarial biology and immunopathogenesis and life cycle, Bestrophin-9, TLR4, ISHBR and TNFR1 represent as efficacious targets for developing appropriate intervention strategies against LF.

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Host-Directed Therapeutic Strategy: Antagonizing B-Catenin Limits Parasites Growth

Recent studies highlight the critical role of innate immunity in *Toxoplasma gondii* infection, though the β-catenin pathway's involvement remains unexplored. We found that AKT-mediated phosphorylated β-catenin translocates to the nucleus, promoting *T. gondii* replication by activating the IRF3 promoter via the β-catenin-TCF4 complex. In the absence of β-catenin, parasite replication was inhibited. AKT also induced STING-TICAM2-dependent phosphorylation of IRF3, enhancing IDO1 transcription, which was essential for parasite growth. Interestingly, IDO1 degradation favored parasite replication by diverting tryptophan to melatonin, reducing ROS, and promoting growth. Stable IDO1 under IFN-γ catabolized tryptophan to kynurenine, suppressing AKT and β-catenin, leading to apoptosis and reduced replication. Kynurenine and its analogue, teriflunomide, inhibited AKT and β-catenin, triggering apoptosis to limit parasite growth. Our findings suggest targeting the IRF3-IDO1 pathway and its metabolites as potential immunotherapies to control *T. gondii* by disrupting the AKT-β-catenin axis.

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Reduced Activity of Mutant CDPK5 is Compensated by the Malaria Parasite by Increased Expression of Other Kinases Involved in PKG-Mediated Signaling Cascade

Plasmodium falciparum Calcium Dependent Protein Kinases (PfCDPKs) are important antimalarial drug targets since they are not present in humans. CDPK5 is critical for merozoite exit from the infected red blood cells. However, artificial activation of protein kinase G (PKG) in CDPK5-knockdown parasites can rescue the egress defect, suggesting existence of alternate signaling pathway/s. To decipher the components of the putative alternate signaling cascade, here we have employed a genetics approach to generate a mutant parasite harboring a hypomorphic allele of *pfcdpk5*. To this end, various mutants of CDPK5 were designed by substituting the gatekeeper residue (L199) with amino acids of different sizes. The purified WT and the mutant proteins were purified and used in an *in vitro* kinase assay to test the effect of gatekeeper substitution on their kinase activity. The threonine and methionine gatekeeper substitutions lead to ~ 89 % and ~ 35 % reduction in the transphosphorylation activity of the enzymes, respectively, compared to the WT. The methionine gatekeeper mutant best mimics the activity of the wild-type enzyme compared to all other mutant enzymes. Serine and alanine mutations completely abrogate the transphosphorylation of the mutant enzymes. Using CRISPR-Cas9, we successfully introduced the L199M mutation in the endogenous cdpk5 locus and generated Pf::cdpk5l199m parasite; however, repeated attempts to generate Pf::cdpk5l199s parasites failed. Interestingly, the Pf::cdpk5l199m parasite shows increased sensitivity towards compound 2, a potent and specific inhibitor of protein kinase G (PKG). Since PfCDPK5 is essential for the egress of merozoites, the reduced activity of CDPK5L199M may be compensated directly/indirectly through PKG. Interestingly, the transcript and protein expression of PfCDPK1 and PfPI3K are upregulated in the Pf::cdpk5l199m parasite compared to the control. Elevated expression of PfCDPK1 and PfPI3K in Pf::cdpk5l199m parasite shows less sensitivity towards specific inhibitors of PfCDPK1 and PfPI3K. Our results suggest that the increased expression of PfCDPK1 and PfPI3K may compensate for the function of the mutant PfCDPK5 in the Pf::cdpk5l199m parasites. Repeated attempts to completely knock out cdpk5 in the WT and the Pf::cdpk5l199m parasites failed, suggesting that the basal activity of CDPK5 is critical for the merozoite egress. Targeting a single kinase may allow the parasite to develop cross-resistance towards drugs that target other compensatory kinases. Therefore, targeting two or more kinases simultaneously may avoid the development of drug resistance against a single kinase. Furthermore, understanding the development of compensatory mechanisms by the parasite may serve as an important guide in the co-selection of drug targets.

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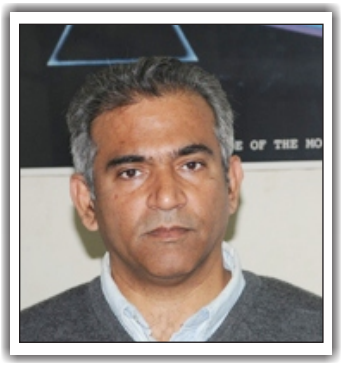
Organelle-Targeted Nucleases for *Plasmodium* Genome Maintenance and Role of an LCR in DNA Substrate Repertoire Expansion

Organelle genome stability is essential for normal cellular function and growth. In the malaria parasite, it also has implications in development of resistance to drugs which target organelle-encoded protein(s). To examine DNA repair mechanisms in *Plasmodium*, we identified apicoplast and mitochondrial repair enzymes and investigated their biochemical function and substrate specificities, with genetic knockout/knockdown conducted in collaboration with Dr. Satish Mishra at CDRI. Targeting of major base excision repair (BER) proteins to the *P. falciparum* mitochondrion indicated that the organelle is the major site for BER. Although BER proteins were not clearly identifiable in the apicoplast, a multifunctional *Pf*Exo/FEN with unique cleavage properties, not reported in any known exonuclease across organisms, was targeted to the organelle. *Pf*Exo differed from its ortholog in *P. berghei* in carrying [4Fe-4S] and containing a ~150 residue low complexity region (LCR) insertion. LCR-deletion and -insertion mutagenesis showed that the sequence expanded the substrate repertoire of *Pf*Exo, imparting it properties that would allow it to function in a range of critical DNA transactions. Two other 'essential' mitochondrion-targeted exonucleases, *Pf*Exo$_{mit1}$ and *Pf*Exo$_{mit2}$, are highly diverged proteins conserved only in certain alveolates. They differed from each other in their DNA substrate specificities. *Pf*Exo$_{mit2}$ cleaved dsDNA bidirectionally; pull-down assays indicated its possible role in DSBR and MMR pathways. The specificity of *Pf*Exo$_{mit1}$ for ssDNA suggested its role in clearance of ssDNA “puddles” reported to accumulate during mtDNA rolling circle replication. Our results map the limited set of *P. falciparum* organellar exonucleases to specific DNA transactions in the apicoplast and mitochondrion.



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Regulation of Plasmodium falciparum Division by Aurora Related Kinases

The malaria parasite exhibits “atypical" and unusual features of cell division. During blood stage schizogony, it undergoes endomitosis, which is accompanied with asynchronous nuclear division that precedes cytokinesis. Aurora kinases are highly conserved eukaryotic protein kinases that regulate division in various organisms. Present studies relate to PfArk1 and PfArk2, two of the three Aurora related kinases (Arks) present in *Plasmodium falciparum*, which are indispensable for the parasite but their precise role in the asexual development of the parasite remains unclear. We demonstrate that conditional knockout of PfArk1 and PfArk2 impaired asexual division of the parasite. UExM analysis of PfArk1/2-depleted parasites revealed that severe defects in spindle formation and karyokinesis. As a result, cytokinesis of these parasites was impaired and these parasites. Quantitative phosphoproteomics was used to identify targets for PfArk1/2, which revealed several kinetocohore proteins which may be targeted by these kinases, which explains their role in above mentioned processes.

IL 18

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Organelle Dynamics in Malaria Parasite and Identification of Key Proteins for Cellular Homeostasis as Drug Targets

Malaria remains a global threat with millions of deaths annually. Emergence of parasites strains resistant to widely used antimalarials, including the Artemisinin Combination Therapy (ACT), and absence of an effective vaccine, make it essential to identify new drug-targets and develop new pharmacaphores against the parasite. Our group is studying organelle dynamics and organelle associated metabolic pathways to understand cell biology of *Plasmodium* during blood stage cycle. Using detailed gene knock-down studies and omics analyses, we have deciphered functional role of key organellar proteins in homeostasis, parasite survival and drug resistance. Phospholipid (PL) biosynthesis is crucial to maintain membrane biogenesis and lipid homeostasis in the rapidly growing malaria parasite in the human host cell. We have functionally characterized an ER-localized Phosphatidyl-serine synthase (PSS) enzyme in *Plasmodium falciparum* to reveal its key role in phospholipid homeostasis, organelle development and parasite survival. GFP targeting approach confirmed it to be localized in the parasite ER-protrusions which form ER-mitochondria contact sites (ERMC) and associate with the mitochondria surface throughout the parasite growth cycle. Understanding organelle interaction and crosstalk can help us to design novel antimalarial strategies.

IL 19

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Hunting Down Malaria Parasite with Newcastle Disease Virus

Merozoites utilizes sialic acids on RBC cell surface to rapidly adhere to and invade the RBCs. Newcastle disease virus (NDV) displays a strong affinity towards membrane bound sialic acids. Incubation of NDV with the malaria parasites dose-dependently reduces its cellular viability. The anti-plasmodial activity of NDV is specific as incubation with JEV, DEV, IBV and Influenza virus did not affect the parasite propagation. Interestingly, NDV is reducing more than 80% invasion when RBCs are pre-treated with the virus. Removal of the RBC surface proteins or the NDV coat proteins results in disruption of the virus binding to RBC. It suggests involvement of specific protein: ligand interaction in virus binding. We established that the virus engages with the PRBCs through its HN protein by recognizing sialic acid-containing glycoproteins on the cell surface. Blocking of the HN protein with free sialic acid or anti-HN antibodies abolished the virus binding as well as its ability to reduce parasite growth. Surprisingly, purified HN from the virus alone could inhibit the parasite's growth in a dose-dependent manner. NDV is preferentially targeting the PRBCs compared to normal erythrocytes. Immunolocalization studies reveal that NDV is localized on the plasma membrane membrane as well as weakly inside the PRBC. NDV neither cause any infection or causes aggregation of the human RBCs. This is the first report to exploit virus as an anti-malarial agent and our findings suggest that NDV is a potential candidate for developing targeted drug delivery platforms for malaria.

IL 20

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Cross Talk Between *Plasmodium* Hsp70 And Hsp90 Chaperone Systems in Client Maturation

Plasmodium experiences significant temperature changes during febrile episodes within human host. Heat shock proteins play a crucial role in maintaining protein folding under temperature stress and thus essential for parasite survival and infectivity. The cytoplasmic chaperone systems of *Plasmodium* majorly consist of PfHsp70-1 and PfHsp90 which collaborate with their cochaperones in a client specific manner to establish the homeostasis of client proteins within the parasite. We have deciphered the molecular mechanisms behind the stabilization of one of the important replication proteins ribonucleotide reductases (RNR) and identified important J-domain protein that regulates the early folding intermediates of PfR2 (ribonucleotide reductase subunit-2). We find that the perturbation of association between the J-domain protein and Hsp70 destabilizes endogenous PfR2 with concomitant reduction in the cellular pool of dNTPs resulting in replication arrest in the parasite. Additionally, we have identified the Hsp90 cochaperone which plays a critical role in maintaining homeostasis of PfR2 by regulating its later stage of folding. We show that knockout of that cochaperone channels PfR2 to proteasomal degradation. Our study shows that inhibition of catalytic function of RNR along with the inhibition of respective chaperone-cochaperone interaction display profound synergism and sensitivity during the growth of malaria parasite. Sequence diversity of *Plasmodium* cochaperones compared to human orthologs promises important avenues to be explored for development of anti-malaria compounds.

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Recent Advances in Kala-azar Management

Kala-azar (visceral leishmaniasis, VL), caused by *Leishmania donovani* and transmitted by female Phlebotomus sandflies, remains a major neglected tropical disease in India and other endemic regions. Despite substantial progress, challenges such as drug resistance, treatment failure, and PKDL persistence hinder elimination efforts. A comprehensive review of evolving management strategies for VL was undertaken, emphasizing therapeutic advancements, clinical trials, and national programmatic adaptations. Key developments in drug regimens, combination therapies, and vaccine research were analyzed in the context of global and national elimination targets. Traditional regimens using antimonials and amphotericin B have been progressively replaced by safer and more effective options. Liposomal amphotericin B (AmBisome) has become the firstline treatment in the National Kala-azar Elimination Programme, offering high cure rates with minimal toxicity. Paromomycin and oral miltefosine have expanded therapeutic accessibility, while combination therapies (e.g., miltefosine–paromomycin, AmBisome–miltefosine) show promise against emerging resistance and relapse, including VL–HIV co-infections and PKDL. Novel compounds such as LXE408 (phase II trials) represent the next generation of oral therapeutics. Parallel vaccine research, including Leish-F1 + MPL-SE and DNA/protein-based candidates, is advancing toward preventive interventions. Recent pharmacological and translational advances have markedly improved Kala-azar management. However, sustained surveillance, treatment adherence, and research investment remain critical to achieving the WHO 2030 elimination goal. Collaborative national and global efforts are essential for transitioning from disease control to long-term eradication.

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Generative Machine Learning–Driven design of Novel Inhibitors Targeting *Plasmodium falciparum* Lactate Dehydrogenase

The increasing prevalence of multidrug-resistant Plasmodium falciparum strains underscores the urgent need for antimalarial agents with novel mechanisms of action. Here, we present a generative machine learning–based approach to design structurally diverse inhibitors of P. falciparum lactate dehydrogenase (PfLDH)—a key enzyme in the parasite's glycolytic pathway and an attractive therapeutic target. Using the deep learning package mollib, pre-trained on ~365,000 ChEMBL bioactive molecules, we applied transfer learning starting from five potent PfLDH inhibitors to generate a focused library of ~3000 novel compounds. These were prioritised using molecular similarity analysis, molecular docking, and 100 ns molecular dynamics (MD) simulations. Top-ranked compound 203, featuring a unique dioxatetracyclo chemotype, demonstrated a docking score of −12.739 kcal/mol—superior to parent inhibitors—and exhibited stable interactions with key PfLDH residues (ILE31, ASN140, ARG109, ARG171). MD analyses confirmed complex stability with low ligand RMSD and consistent protein–ligand hydrogen bonding. The compound's low Tanimoto similarity (<0.5) to known inhibitors highlights its structural novelty. This integrative workflow demonstrates the potential of generative deep learning combined with structure-based virtual screening to expand the chemical space for antimalarial discovery and identify new chemotypes for PfLDH inhibition, which may be applied to other drug targets too.

IL 23

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Subtractive Genomics and Computational Structural Studies of *Wuchereria bancrofti* Targets for Developing Novel Antifilarial Therapeutics

Lymphatic filariasis, or elephantiasis, is one of the most debilitating mosquito-borne neglected tropical diseases, severely impacting public health in many developing nations, including India. Although three nematode species are known to cause elephantiasis, *Wuchereria bancrofti* accounts for nearly 90% of infections. Current therapeutic options, including diethylcarbamazine, albendazole, and ivermectin, used in double- or triple-drug therapy, are increasingly limited by the emergence of drug resistance due to SNPs, poor specificity for filarial targets, and limited efficacy across all larval stages of the parasite. Our study aims to identify and validate novel druggable targets in *W. bancrofti* and discover potent antifilarial lead compounds.

We employed a subtractive genomics approach to identify putative drug targets, focusing on major antioxidant enzymes. The structures of these targets were modelled computationally, and their stability was evaluated through large-scale molecular dynamics simulations. Structure-based drug design using the small molecule library prioritized non-toxic lead candidates with high affinity and specificity compared to human homologs. The top-ranked leads demonstrated better *in vitro* activity, with improved IC_{50} values relative to reference drugs, using the cattle filarial nematode *Setaria digitata* as a surrogate model. Pharmacophore features derived from these ligands will be exploited to design new derivatives with enhanced efficacy for subsequent *in vivo* testing using filarial-infected rodent models. Further, the recombinant expression of target proteins in and their crystallization are underway, yielding microcrystals of varying morphology. Optimization for X-ray diffraction and structure determination of native and ligand-bound complexes are in progress. This integrative computational and experimental approach provides promising leads and structural insights for the development of novel, stage-effective antifilarial therapeutics. Detailed findings will be presented during the conference.

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Unveiling the Immunoregulatory Role of Dermal Fibroblasts in Visceral and Post-Kala-Azar Dermal Leishmaniasis

Although the immunological transition from visceral leishmaniasis (VL) to post-kala-azar dermal leishmaniasis (PKDL) is still poorly understood, PKDL develops in a proportion of VL patients following apparent recovery. The function of dermal fibroblasts in influencing immunological responses during *Leishmania donovani* infection is investigated in this work. After culturing and RNA sequencing dermal fibroblasts from VL, PKDL, and healthy individuals, 516 genes that were differentially expressed by PKDL and VL fibroblasts were discovered. Important pro-inflammatory mediators (NFKB1A, MMP2, CXCL8, IL6, IL1β, and CXCL8) were markedly downregulated in PKDL fibroblasts. When fibroblasts and THP-1 macrophages were co-cultured, it was discovered that VL fibroblasts strongly expressed cytokines (IL-6, IL-1β, IL-8, IFN-γ, and TNF-α) and chemokines (CXCL1–12), which in turn promoted NF-κB activation and immune effector cell recruitment. Conversely, cytokine and chemokine output was decreased by Leishmania donovani infection, indicating immunological fatigue. After infection, PKDL fibroblast-stimulated THP-1 cells showed increased levels of IL-10 and IL-12, indicating a balanced inflammatory and regulatory state that supports parasite persistence with little tissue damage. Mechanistically, THP-1 cells' NF-κB p50/p65 nuclear translocation was triggered by fibroblast-derived IL-6, but NF-κB activation was stopped by IL-6 neutralization. These results show that PKDL fibroblasts use IL-10 and IL-12 to support regulated immunological reactivation, while VL fibroblasts create a pro-inflammatory environment driven by IL-6 and NF-κB. In leishmaniasis, fibroblasts are important modulators of cutaneous immune responses that affect the chronicity and outcome of the illness. Modifying host immunity in VL and PKDL may be possible through the use of innovative treatment approaches that target fibroblast-mediated mechanisms.

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Studies on Mevalonate Kinase of *Leishmania Donovani*: Role in Host Invasion, Pathogenesis and Parasite Survival

Leishmania secretes over 151 proteins during *in vitro* cultivation. Cellular functions of one such novel protein: mevalonate kinase is discussed here; signifying its importance in *Leishmania* infection. Visceral Leishmaniasis is a persistent infection, caused by *Leishmania donovani* in Indian subcontinent. This persistence is partly due to phagocytosis and evasion of host immune response. The underlying mechanism involves secretory proteins of *Leishmania* parasite; however, related studies are meagre. We have identified a novel secretory *Leishmania donovani* glycoprotein, Mevalonate kinase (MVK), and shown its importance in parasite internalization and immuno-modulation. We have shown that MVK is essential for the parasite to protect it from oxidative stress by regulating ergosterol biosynthesis. MVK was found to be secreted maximum after 1 h temperature stress at 37°C. Its secretion was increased by 6.5-fold in phagolysosomelike condition (pH ~5.5, 37°C) than at pH ~7.4 and 25°C. Treatment with MVK modulated host immune system by inducing interleukin-10 and interleukin-4 secretion, suppressing host's ability to kill the parasite. Peripheral blood mononuclear cell (PBMC)-derived macrophages infected with mevalonate kinase-overexpressing parasites showed an increase in intracellular parasite burden in comparison to infection with vector control parasites. Mechanism behind the increase in phagocytosis and immunosuppression was found to be phosphorylation of mitogen-activated protein (MAP) kinase pathway protein, Extracellular signal-regulated kinases-1/2, and actin scaffold protein, cortactin. Thus, we conclude that *Leishmania donovani* Mevalonate kinase aids in parasite engulfment and subvert the immune system by interfering with signal transduction pathways in host cells, which causes suppression of the protective response and facilitates their persistence in the host. Our work elucidates the involvement of Leishmania in the process of phagocytosis which is thought to be dependent largely on macrophages and contributes towards better understanding of host pathogen interactions.

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Protective Role of Linoleic Acid, an Essential Fatty Acid, in the Containment of *L. Donovani* Infections in Hamsters

A major risk factor for visceral leishmaniasis (VL) is malnourishment and malnourished people show altered levels of linoleic acid (LA, 18:2; u-6 fatty acid). Derivatives of LA i.e. arachidonic acid and its metabolites possess immune-modulatory properties. However, its role in VL is not yet investigated. Our present study establishes the importance of LA in VL infection. *L. donovani* infected hamsters showed increased concentrations of LA after 60 days of infection. On the contrary, a decline in LA concentrations was observed in the serum samples of VL patients, indicating malnutrition among these patients. In our *in vitro* study, supplementation of LA before the infection promoted the protective type of immune response (↑ IL-12 and iNOS expression) and significantly reduced the parasite load within infected macrophages. This protective response of LA was accomplished via 5-lipoxygenase pathway. Further, 15-day LA pre-treatment in hamsters demonstrated its preventive efficacy by containing parasite burden and enhancing Th-1 type immune response (IL-12, iNOS, IFNg, TNF-α) during initial phase of infection across various visceral organs. The evidence of disease containment was confirmed through splenic biopsies. Comparable responses were also observed upon therapeutic application of LA to *L. donovani* infected macrophages (↑ Th-1 and ↓ Th-2 type response). Thus, we conclusively demonstrated the protective role of LA in restricting the parasite load. The people belonging to endemic regions of India are heavily relying on LA-poor mustard oil for the dietary usage, in terms of the proportion of its constituents. Hence, incorporation of LA-rich oil (sunflower oils, sesame oil, etc.) in daily food habits could be a significant movement towards the elimination of the disease from India.

IL 27

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Epigenetically Regulated RIFINs Linked to Severe Malaria Pathogenesis

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

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Genomic Evidence for Host-Switch of the Malaria Parasite, *Plasmodium falciparum* in India: Implication on Targeted Elimination

Malaria is a mosquito-borne parasitic disease and endemic in India. The government of India has set up a National Framework for Malaria Elimination (NFME) that aims to interrupt indigenous transmission by the year 2030. Although remarkable achievements have been made to achieve the goal, but malaria persists in populations living in ecological and social margins, (*e.g.* rural, island and tribal populations) as these populations live at the interface of human-mosquito vectors-and wildlife. Accordingly, in India about 80% of the malaria burden of the country comes from populations living in such conditions. The deadliest form of malaria parasite, *Plasmodium falciparum* is known to have switched it's host to human from the non-human primates (NHP), but exactly from which NHP, was not known. Since many NHPs inhabit in rural, forested, hilly and island populations in India, we made an effort to understand host-switching of *P. falciparum* parasite in India. For this, we have sequenced a bunch of whole mitochondria genomes of Indian *P. falciparum* sampled from different human populations in India, and also two sequences of the *P. falciparum*-like malaria parasites (*P. coatneyi* and *P. fragile*) infecting Indian rhesus macaque. Firstly, different population genetic parameters of Indian mitochondrial genomes compared with world-wide data indicated the ancestral nature of *P. falciparum* in India. Secondly, not only we found evidences for host-switch of *P. falciparum* in India, but interestingly found a new 'genetic type' of *P. falciparum* (*PfIndia**) circulating in Indians. With genomic analyses, *PfIndia** was found to be intermediate between *P. falciparum* and P. falciparum-like parasites (*P. fragile* and *P. coatneyi)*, and possibly had served as a 'missing link' during the host-switch. A parallel study describing infection of *P. falciparum* in Indian bonnet and rhesus macaque suggests that zoonotic cycle of malaria parasites is maintained in India. Therefore, targeted malaria elimination will highly depend on the way the program will deal with the ongoing zoonotic cycle of malaria parasite.

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Molecular Xenomonitoring for Monitoring Lymphatic Filariasis Elimination Programme

Tools to monitor the programme to eliminate lymphatic filariasis (LF) are essential components of the strategy and WHO has developed monitoring and evaluation guidelines for stopping decisions. However, there are some challenges with the current human infection-based surveillance and a number of countries are reporting failures in the assessment. Therefore, there is a need to develop supplementary surveillance for stronger evidence for interruption of transmission so that the risk of resurgence of infection after stopping MDA can be prevented. Transmission of lymphatic filariasis has two components: transmission from man to mosquito vector (infection) and transmission from the mosquito vector to the human host (infectivity). Direct detection of microfilariae of the parasite in the vector is indicative of both the presence of patent (circulating microfilariae) infections in humans and transmission of the infection from humans to the vector. Thus, the transmission is a key indicator of transmission intensity and can be monitored by measuring changes in infection status of vectors (called xenomonitoring), apart from that of humans. Vector infection and infectivity rates are conventionally determined by dissection and microscopic examination of vector mosquitoes for filarial parasites and their stages. However, this method is cumbersome, subjective, has low through-put and not applicable in areas with ultra-low parasite prevalence in vectors. These attributes make this method unsuitable for use in assessing large scale programmes such as GPELF. Hence, there is a need for alternative, simple, faster, less expensive and mass screening method(s). Efforts were made during the last two and half-decade, and highly sensitive and specific PCR based Molecular Xenomonitoring (MX) methods for the detection of infection and infective (L3) stage larvae of *Wuchereria bancrofti* and *Brugia malayi* in vectors have been developed. A simple and inexpensive technique for isolation of DNA from infected mosquitoes has been developed and found to be useful in xenomonitoring of LF. Since pools of 25-100 mosquitoes can be subjected to MX, a technician with experience in MX can process about 1000s of mosquitoes per day, which is 10-40 times the output of conventional method. Further, the molecular xenomonitoring (MX) for *W. bancrofti* developed at the VCRC has been validated in several geographic locations in India, which showed that it is 2-3 times more sensitive than mf survey when compared with other parameters (microfilaria and circulating filarial antigen). Currently, studies on the performance of MX in field settings, in comparison with Mf and serological (filarial antigens and antibodies) surveys are going-on. Status of Molecular xenomonitoring in the global LF elimination efforts will be discussed.

IL 30

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Investigating the *Taenia solium* Fatty Acid Binding Protein Superfamily for Their Immunological Outlook and Prospect for Therapeutic Targets

Neurocysticercosis is a major neurological threat, accounting for over 30% of cases in endemic areas. *Taenia solium*, the causative agent, and its other helminthic counterparts lack key components of the cellular machinery required for endogenous lipid biosynthesis. This deficiency enables the parasite to obtain all required lipids from its host organism. To facilitate effective lipid transport, the cestode parasite employs Fatty Acid Binding Proteins (FABPs), which bind to lipid ligands and allow lipid transport across membranes and into the cytosol. *T. solium* expresses an abundance of FABPs. Apart from transporting ligands, FABPs interacts with the host immune system, however the functional aspect of *T. solium* FABP is still unknown. Elucidating the functional outcome of FABP on host immune system will contribute to understand the detailed immunopathology of cysticercosis infection. TsFABP1 is one of the members of *T. solium* FABP family, which is secretory in nature, interacts with neighbouring cells, potentially modulating their functions. We expressed TsFABP1 in the pET23a vector, purified it with Ni-NTA affinity chromatography, and measured the molecular weight at 15 kDA. TsFABP1 purified form induced anti-inflammatory gene expression in THP-1-derived macrophages in a dose-dependent manner. TsFABP1 inhibits the CD14-TLR4 pathway by binding to CD14 at the LPS binding site, reducing ROS and cleaved IL-1β production. Interestingly macrophages readily internalize the cyanine5-labelled TsFABP1 and in the cytosol the protein may play significant role in immunomodulation. Here for the first time, we report that the TsFABP1 play role in PPAR-γ pathway, which was previously unknown. TsFABP1 suppresses IRE-1α in a PPAR-γ-dependent manner. In conclusion, TsFABP1 is an anti-inflammatory molecule that also downregulate the endoplasmic stress response associated molecule and apart from that TsFABP1 can be explored for therapeutic potential.

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Deciphering the *Theileria annulata* Kinome to Unravel the Cyclin–CDK Interactome

Theileria parasites are the only eukaryotes that have the ability to transform another eukaryotic cell, specifically the leukocytes of cattle. The transformed cells exhibit phenotypes like those of cancer cells. Targeting protein kinases is a cornerstone of modern cancer therapy. Given that *Theileria*-infected cells display cancer-like phenotypes; the selective inhibition of key kinases may offer a promising strategy for eliminating *T. annulata*-infected cells. Our group has previously reannotated the kinome of *T. annulata* and identified 54 protein kinases in its proteome, which were classified into eight kinase groups: AGC, CAMK, CK1, CMGC, NEK, OPK, and aPK. In this study, we focused on the CMGC family of protein kinases, particularly the cyclin-dependent kinases (CDKs). CDKs execute their functions through interactions with cognate cyclins and play essential roles in regulating cell cycle progression, mitotic division, and transcription, as extensively documented in mammals and other apicomplexan parasites like *Plasmodium* and *Toxoplasma*. The findings from the yeast two-hybrid demonstrate that a single cyclin can interact with several TaCDKs. TaCDK7 specifically interacts with TaCyclin H as well as TaMAT1 and is involved in transcription. Western blot analysis employing lysates from infected and control cells verified the specificity of the *in-house* generated antibodies against all TaCDKs and TaCyclins, which showed different localization patterns in schizont and merozoite phases. Their relationships were further confirmed by colocalization investigations in schizonts and during synchronized S-phase and G2/M phases, suggesting that a single cyclin can associate with multiple TaCDKs. The interaction of a single TaCyclin with multiple TaCDKs suggests its involvement in distinct regulatory pathways during the parasite's life cycle.

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Noncoding but Noteworthy: A Novel, Stage Regulated lncRNA Governs the Developmental Fate in *Toxoplasma gondii*

Stage differentiation is central to the survival and transmission of Toxoplasma gondii, yet the molecular mechanisms driving this process remain poorly understood. In our study, we identified a novel long non-coding RNA (lncRNA), that exhibits stage-specific expression during parasite development. Deletion of this lncRNA resulted in a significant slowdown of parasite replication and aberrant vacuolar organization. Transcriptomic profiling further revealed extensive gene expression changes, including a shift toward sexual stage commitment. Together, these findings suggest that the lncRNA serves as a key regulatory molecule orchestrating developmental transitions in T. gondii. Our work uncovers a previously unexplored mechanism of post-transcriptional regulation involving lncRNA in parasite differentiation and life cycle stage transition and complex nature of the process.

IL 33

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Toxoplasma gondii RNA Triphosphatase (TgCet), the First mRNA Capping Enzyme: Potential Drug Target for Toxoplasmosis

Toxoplasma gondii, an apicomplexan parasite, exhibits a complex life cycle that involves propagation in multiple hosts and major changes in gene expression across lifecycle stages. RNA modifications play an important role in regulating gene expression in eukaryotes; however, the role of 5' 7-methylguanosine (m^7G) cap, the first modification of mRNA, remains unknown in *T. gondii*. We recently demonstrated that the mRNA capping machinery of *Toxoplasma comprises* three distinct enzymes: RNA triphosphatase, guanylyltransferase, and guanine-N7-methyltransferase, which collectively add a functional m^7G cap to RNA. Biochemical and genetic analyses reveal that among three capping enzymes, RNA triphosphatase (TgCet) is unique and a member of the tunnel family of metal-dependent phosphohydrolases, which is structurally and mechanistically distinct from the human RNA triphosphatase. Knockdown studies show that TgCet is essential for mRNA capping, and its depletion leads to widespread changes in m^7G-capped transcripts, resulting in the complete arrest of parasite replication in culture and in the mouse host, thereby protecting the host from lethal infection. Furthermore, the therapeutic potential of TgCet was evaluated, showing that Myricetin selectively inhibits TgCet activity and effectively blocks parasite replication in culture. Overall, these findings highlight the essential role of TgCet-mediated mRNA capping, establishing RNA triphosphatase as a potential drug target for toxoplasmosis.

Oration Awards

Dr. B. N. Singh Memorial Oration Award

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Transcriptional and Metabolic Basis of Artemisinin Tolerance

The emergence of resistance to antimalarial drugs, including artemisinin-based combination therapies (ACTs), remains a major obstacle to malaria control. Mutations in *Plasmodium falciparum* Kelch13 (K13), a key molecular marker of artemisinin (ART) resistance, are associated with multiple adaptive mechanisms such as reduced hemoglobin endocytosis and consequent lower drug activation, activation of the unfolded protein response, elevated phosphatidylinositol-3-phosphate (PI3P) levels, and induction of autophagy. However, K13 mutations alone do not confer complete resistance. A small fraction of ring-stage parasites can enter a quiescent, drug-tolerant state, leading to recrudescence after treatment. This phenomenon parallels stress-induced transcriptional heterogeneity in P. falciparum, suggesting the existence of persister subpopulations capable of metabolic rewiring to survive drug pressure. Our work aims to dissect the interplay between transcriptional plasticity, lipid metabolism, and hypoxia-driven responses that together define the metabolic basis of artemisinin tolerance in *P. falciparum*.

Dr. B. P. Pandey Memorial Oration Award

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Integrative Anti-Leishmanial Strategies: Harnessing Phytochemicals, Therapeutics, and Vaccines"

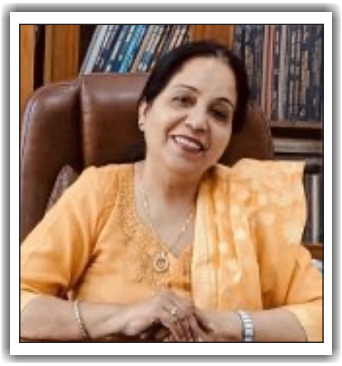
Visceral leishmaniasis (VL), or kala-azar, is a neglected tropical vector borne disease caused by *Leishmania donovani*. Current management strategies are limited by the scarcity of effective drugs, toxicity, drug resistance, and the lack of a vaccine. These challenges underscore the need for safer and more effective integrative strategies that combine plant-derived bio-actives, improved chemotherapeutic formulations, and rationally designed vaccine candidates.

To address these limitations, our research focused on exploring natural compounds and their active metabolites with anti-leishmanial potential that are both safe and immunomodulatory. An ***in silico***, ***in vitro***, ***in vivo*** approach is used to identify drug targets and repurposed candidates from FDA-approved libraries, focusing on *Trypanothione Reductase* (TRYR) and *Pteridine Reductase 1* (PTR1). Parallel studies with *Leishmania*-derived nosodes and homeopathic preparations revealed safe, immunoprotective potential.

Recognizing the limitations of chemotherapy alone, we expanded our efforts toward vaccine development. Vaccine formulations using first and second generation antigens, combined with adjuvants such as MPL-A, saponin, tomatine, quercetin and novel synthetic TLR7/8/NOD2 agonists (HYBRID2, dhBBIQ, PAM-BBIQ and DMP), induced robust Th1-skewed immune responses, elevated IFN-γ and TNF-α, and generated long-lived memory $CD4^+$ and $CD8^+$ T cells.

Collectively, these findings advocate a synergistic, holistic paradigm-integrating phytochemicals, repurposed drugs, and next-generation vaccines - to achieve durable immunity and move closer to the ultimate goal of visceral leishmaniasis eradication.

Prof. Kona Hanumantha Rao Memorial Oration Award



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Unveiling Hidden Diversity: Taxonomy of Parasites in Fish and Veterinary Animals

It is a moment of great pride to receive award of Prof. K. Hanumantha Rao Memorial Oration Lecture bestowed on me by the prestigious Indian Society of Parasitology, India. Prof Hanumantha Rao was a great helminthologist of the country and he contributed immensely in the field of taxonomy of trematode parasites. I feel, it is a momentous moment to mention that Prof. Rao was one of the examiner for the evaluation of my PhD thesis in 1995 wherein I worked on reproductive biology of four species of nematode parasites exhibiting oviparity, ovoviviparity and viviparity. My journey in the field of parasitology started very early in MSc Hons wherein I worked on taxonomy of fish parasites and published my first paper in Rivista De Parasitologica in 1987. During the whole journey as a parasitologist, I was involved in the field collections and identification of various groups of parasites infecting fishes and domestic animals. Indian subcontinent due to its tropical and subtropical climate, dense population, varied ecosystems, and socio-economic diversity, is a hotspot for a wide range of indigenous parasitic diseases. These diseases are caused by protozoa, helminths, and ectoparasites, many of which are endemic to the Indian subcontinent. My major contribution has been on diversity of myxozoan species infecting gills, fins, scales, skin and other vital organs such as kidney, liver and heart of freshwater fishes inhabiting culture ponds and wetlands of Panjab & major lakes of Jammu and Kashmir. Besides contributing various synopses, new species records, new methodologies for easy detection in tissues and infection indices have been proposed and published in journals of repute. There is much contribution from my lab on the diversity and molecular identification of acanthocephalan and cestode parasites of siluroid fishes; anoplocephalids infecting small ruminants; management of root knot nematode and molecular characterization of tick species etc. Other aspects being worked out are host-parasite interactions disease patterns and pathogenesis in fish host; alternate hosts involved in cultured system and biochemical & immunological alterations in the fish host. More recently, the focus is on the evaluation of GI parasites particularly in cestodes (anoplocephalid) and amphistomes (*Paramphistomum* spp. / *Cotylophoran* spp.) infecting small ruminants for the accumulation of heavy metals. Some helminths accumulate heavy metals (Cd) and (Pb) at levels thousands of times higher than in the host's tissues. This makes them more sensitive and reliable for monitoring contamination than host tissues alone, furthermore the presence of helminths can also influence heavy metal levels within the host animal, potentially offering a protective effect by detoxification.

Being a hardcore taxonomist, I encourage my students to maintain a spirit of discovery and analyze data responsibly and publish in quality journals.

Proffered Talks

Molecular Analysis of Anti-Malarial Drug Resistance Markers in Nancowry Group of Islands of Andaman & Nicobar Archipelago

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Anti-malarial drug resistance poses a major challenge to malaria treatment and elimination efforts worldwide. *Plasmodium falciparum* isolates have developed resistance to several key antimalarial agents, including sulfadoxine-pyrimethamine (SP) and artemisinin. Genetic mutations in *pfdhfr*, *pfdhps*, *pfmdr1*, and *pfkelch13* genes are known molecular markers of such resistance. The Andaman and Nicobar Archipelago, historically malaria-endemic, remains a region of strategic importance for India's malaria elimination goals.

Genetic profiling is one way to asses/detect the drug resistance associated mutations present among *Plasmodium falciparum* isolates. In this study, mutations linked with SP drug failure were found among majority of collected *Plasmodium falciparum* isolates. The quadruple mutations in the *pfdhfr* gene and triple mutations in the *pfdhps* genes were found among majority of isolates. However, no validated mutation(s) linked with artemisinin resistance was detected among *Plasmodium faclciparum* isolates.

Based on the findings, it can be concluded that these mutations have been fixed in the parasite population locally. The fixation of SP resistance-associated mutations among *P. falciparum* isolates highlights the need for continuous molecular surveillance in the Andaman and Nicobar Islands. Given the high prevalence of multidrug-resistant genotypes and the geographic remoteness of the islands, policymakers should consider revising local anti-malarial treatment strategies to support India's malaria elimination objectives.

A *Plasmodium falciparum* Molecular Mechanism of Heme Binding and Sensitivity to Artemisinins

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Heme is critical to activating artemisinins (ARTs), drugs essential for malaria control. *Plasmodium falciparum* Kelch13 (K13) protein binds heme *in vitro*, but its *in vivo* functions remain unknown. As precise regulation of free-heme levels is not feasible in infected erythrocytes, we developed mammalian cell model to study molecular properties of K13 at biological heme concentrations. We show that K13 levels are exquisitely responsive to nanomolar and micromolar amounts of heme. Heme stabilizes K13 with chemical and molecular specificity, raises its oxidative-stress responses and association with autophagic-endosomes. Targeted disruption of lysosomal autophagy further increases K13 levels to fuel ART-induced redox-cell death that is proportional to K13 intensities at physiological heme levels. K13's kelch-domain confers both heme and ART responsiveness to its mammalian orthologue KEAP1. These data suggest a novel molecular mechanism for K13 heme-binding in regulating ART-sensitization and the power of models to study frontiers of pro-oxidant stress.

How Nemic Zoonoses Evolved

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Literature records that the nematodes with Pre-cambrian origin evolved independently several times over the period of time. After originating from Tethys Ocean (Moravec, 2010), the genera like *Rhabdochona nemacheli* (Rautela and Malhotra, 1982) flourished in the semi-temperate zone of Garhwal Himalayas. From thereonwards the conditions in Gangetic plains influenced roundworms to result into emergence of characters of Camallanidae *viz.* buccal capsule with tridents anteriorly in *Paracamallanus tridenti* which instantly merged with characters of Anisakidae, to initiate zoonoses outbreak as exemplified by *Indospinezia mulstispinatum* (Jaiswal and Malhotra, 2017). Certain advanced characteristics of typically bifid mucron admixed with physalopterid characteristics of heavily papillated structures emerged in camallanoid worms. One of the prominent features that contemplated implications of environmental influence in a unique fashion encompassed Gangetic water quality triggered morphology of sunflower papillae that appeared in *Indospinezia* and *Rostellascaris.* Consistently ahead of emergence of anisakid characteristics in *Rostellascaris spinicaudatum* interlabia were seen devoid of buccal tooth. These worms transformed into typically advanced anisakid *Pronakid goai* (Malhotra and Yadav, 2025) that had spicules in males with spoon-shaped distal extremity. In the higherarchy *Anisakis typica* apparently evolved next to *Rotundocollarette capoori* (Yadav, Kapoor and Malhotra, 2022) exhibited buccal tooth atop cephalic complex and the 3 anisakid features.

Gregarines: An Understudied Parasitic Group with Huge Potential to Mankind

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Gregarines are a diverse and largely understudied group of apicomplexan parasites that primarily infect the digestive tracts of invertebrates, especially insects. Unlike their pathogenic relatives *Plasmodium* and *Toxoplasma*, gregarines are typically non-lethal and mildly pathogenic, which has contributed to their neglect in research. However, their unique biology and evolutionary position offer vast untapped potential for applications in biotechnology, agriculture, and medicine. Gregarines are promising candidates for environmentally friendly biopesticides, as infections can reduce host fitness and enhance susceptibility to other control measures. Similarly, they harm aquaculture, particularly shrimp and crab farming. Their close evolutionary relationship to pathogenic apicomplexans also makes them valuable models for studying parasite biology, host interaction, and novel mechanisms of gliding motility. Insights from such studies could inform the development of new drugs and vaccines against major parasitic diseases. Additionally, gregarines are important for understanding gut microbiota in edible insects—a growing area in sustainable food systems. It serves as an excellent model for studying cell polarity, organelle differentiation, motility, and understanding the mechanisms of host attachment and nutrient uptake. Last, but not least, students can see live parasites moving, dividing, and interacting with hosts — making them curious and attracting them to study biology/ parasitology.

Multigene Families at Work: Orchestrating Metal-Ion Transport in Apicoplast and Mitochondria of *Plasmodium falciparum*.

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A significant elevation in serum magnesium (Mg) levels has been observed during *Plasmodium falciparum* infections, suggesting a possible interplay between the parasite's life cycle and host magnesium metabolism. A multigene family of metal-ion transporters, primarily of prokaryotic origin, is known to regulate Mg homeostasis, and three functional homologs of these transporter family have been identified in *P. falciparum*. To investigate their roles, we performed computational and phylogenetic analyses to predict the subcellular localization of the homologs. Transgenic parasites were generated through transient transfection which revealed that two homologs are trafficked individually to distinct organelles, the apicoplast and mitochondria, while a third homolog exhibited dual localization in both organelles. Based on these findings, we hypothesize that two homologs interact to form a heteropentameric transporter, thereby providing functional compensation upon the loss of an individual homolog. Importantly, cation hexaammines demonstrated potent parasiticidal activity by selectively inhibiting the parasite's Mg^{2+} transport system through direct interaction with the transporter pore. Moreover, the selection of Co(III)Hex-resistant parasite lines provided molecular insights into the mechanisms of Mg^{2+} transport mediated by these transporters.

Designing of a Novel Multi-Epitope Cocktail Vaccine Candidate for Lymphatic Filariasis Using Immunomics, HRAMS, Computational Biology, and Molecular Dynamics Approaches

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Lymphatic Filariasis is a common neglected tropical disease (NTD) affecting millions of people globally and imposing huge burden on world economy. The current anti-filarial drugs lack adulticidal activity, have low bio-availability and are only microfilaricidal. An effective multi-epitope LF vaccine may provide the necessary boost to the LF elimination programs. A multi-epitope cocktail vaccine candidate (CVC) was developed in this study through the application of immunoproteomics and immune-informatics. The antigenic proteins were identified by immune-blotting against various categories of *W. bancrofti* infected LF sera. The primary antigenic proteins were 14-3-3 zeta, Enolase, Galectin, Tubulin beta chain, and Heat shock protein 70. After predicting the linear B-cell and T-cell epitopes of individual antigens, the five antigens were combined to create a multi-epitope CVC. A three-dimensional model of the candidate vaccine was predicted, refined, and validated using RAMPAGE and PROCHECK servers. In order to improve the vaccine's immunogenicity, the 50S ribosomal subunit of Mycobacterium tuberculosis, a Toll-like receptor (TLR) agonist, was incorporated into the candidate vaccine. The docking of the chimeric peptide vaccine against the TLR5 yielded a docked complex with a high binding efficiency. The *in-silico* immune simulation resulted in a substantial increase in the populations of CD4+ T-cells and CD8+ T-cells. In conclusion, the recombinant putative vaccine demonstrated a high level of immunogenicity, which would be experimentally demonstrated in the future to facilitate the development of a potent LF vaccine.

Identification of Novel Malaria Diagnostic Markers

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A protozoan parasite from the family Plasmodium causes malaria, a vector-borne illness that is spread by the female mosquitoes. The World Health Organization (WHO) estimates that 250 million cases are recorded annually. To lessen the complications of the illness, it is crucial to diagnose the parasite and its causative species as soon as possible. Various techniques (RDT, PCR, microscopic analysis, etc.) are accessible and regularly used to identify the malaria parasite. However, each technique has advantages and disadvantages of its own. Rapid Diagnostic Tests (RDTs) based on HRPII protein approach is used most commonly. False-negative and False-positive reports, which are both connected to HRPII protein, are among the major RDT drawbacks. As a result, HRPII protein replacement is urgently required for the prediction of *P. falciparum* infection. We have chosen malaria proteins with a very high abundance but a very short half-life for the proposed research. With this approach, a diagnostic marker with extremely high specificity and no false positives would be developed for the first time. Through precise pre- and post-treatment detection, the chosen marker(s) will eliminate both the issues of false negatives and false positives and will contribute to the 2023 goal of 'Accelerating the fight against malaria'.

Expanding the Genome and Transcriptome Engineering Toolkit *for Plasmodium* Functional Screens

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Available CRISPR/Cas genome-editing tools fail to precisely target all *Plasmodium falciparum* genomic regions and are hence not suitable for genome-wide phenotypic screens. This prompted us to adapt and optimize a diverse set of CRISPR/Cas systems to *P. falciparum*, including DNA-targeting Cas9 and Cas12 platforms for gene knockout and RNA-targeting Cas13 systems for transcript knockdown. Thus far, we have tested 12 new DNA-targeting systems and identified two that work in *P. falciparum* asexual blood stages. Further, we have established CRISPR/Cas-based mRNA knockdown in asexual stages using a compact Cas13b, which, to the best of our knowledge, is the first such study. Beyond repurposing established systems, we performed large-scale bioinformatic mining of protein databases and identified compact, previously uncharacterized Cas13 variants with significant potential for RNA manipulation in parasites. Collectively, these approaches expand the molecular toolbox available for *Plasmodium* research and provide powerful strategies to dissect unique aspects of parasite biology such as virulence gene expression and stage transitions.

PT 9

Prolonged Sunlight or UV Radiations Exposure Contributes to the Pathogenesis of PKDL in VL-Endemic Populations of Bihar

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Post Kala-Azar Dermal Leishmaniasis (PKDL) is a post-treatment complication of Visceral Leishmaniasis (VL) and posing a major challenge to Kala-Azar elimination efforts in Indian subcontinent. PKDL patients act as reservoirs for *Leishmania donovani*, sustaining disease transmission. The PKDL lesions are generally occurred on the sun-exposed areas of the body suggests a possible role for sunlight ultraviolet radiation (UVR) in this disease development. In the present study, we have assessed both the ground and satellite-based UV radiation in VL-endemic areas, which revealed intense UVR levels (≥12.0–14.5) during summer and pre-monsoon seasons, but lower indices (<5-6) in winter. We have analyzed >250 PKDL patients' data which showed 38% (n=108) and 22% (n=63) of PKDL patients were exposed daily for ≥6–8 and 4-6 hrs, respectively and 73% experienced photosensitivity symptoms on their skin. Our studies showed that the mainly PKDL patients were labourers (33%) and field workers (20%). UVR-induced immune modulation was also validated in THP-1 cells found in alteration in release of cytokines level. Biochemical and immunological profiling revealed altered cytokine levels (IL-10, IL-12), IFN-γR, Vitamin D, and iron in VL and PKDL patients compared to healthy controls. PKDL cases showed upregulation of TLR-2/4 and TNFR-2 and downregulation of TNFR-1. We have also examined Vitamin D receptor (VDR) gene polymorphisms (rs1544410, rs7975232, rs731236), and found a significant association of Bsm1 (rs1544410) with VL and PKDL ($p<0.0001$). Individuals with AA and GA genotypes had a higher risk of disease. The expression of VDR and CYP27B1 genes were upregulated in pretreated PKDL but down-regulated in VL patients. Vitamin D levels in VL patients were found lower than PKDL ($p<0.01$) patients. In conclusion, our results demonstrated that prolonged sun-light (UVR) exposure is a risk factor for the development of PKDL in VL treated/endemic populations.

Molecular Docking and Dynamics Based Discovery of Novel Anthelmintic Inhibitors Against *Haemonchus contortus* O-Acyltransferase

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Haemonchosis caused by the parasitic worm *Haemonchus contortus* is a major threat to the cattle and other ruminants and imposes significant economic losses in the livestock industry. Different medications have been reported; however, these are not reliable now due to mass drug resistance. Increasing drug resistance has prompted the search for novel anthelmintic candidates through *in silico* approaches. In this study, O- acyltransferase (OAT), a key enzyme involved in lipid metabolism and membrane biosynthesis, was selected as a potential drug target. The Physiochemical properties, Secondary structure details were analysed. Then the OAT structure was modelled and validated for reliability prior to virtual screening of selected natural compounds using molecular docking approach. Among the screened molecules, luteolin (–7.890 kcal/mol) and quercetin (–7.861 kcal/mol) exhibited the highest binding affinities, followed by kaempferol (–6.618 kcal/mol), epigallocatechin (–6.231 kcal/mol), resveratrol (–6.164 kcal/mol), curcumin (–5.686 kcal/mol), and berberine (–5.400 kcal/mol). These top-ranking compounds showed strong hydrogen bonding and hydrophobic interactions with essential catalytic residues of OAT. Molecular dynamics (MD) simulations confirmed the structural stability of the luteolin–OAT and quercetin–OAT complexes with minimal RMSD fluctuations, suggesting stable binding throughout the simulation period. ADMET profiling further indicated favorable pharmacokinetic and safety properties for the top compounds. Overall, the results highlight luteolin and quercetin as promising natural inhibitors of O-acyltransferase, providing valuable leads for developing next-generation anthelmintic agents against pathogenic *H. contortus*.

Flash Talks

FT 1

Investigating the Role of H3K36 Methylation-Mediated Epigenetic Regulation in the Malarial Parasite *Plasmodium falciparum*

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A closer look at *Plasmodium falciparum* reveals some peculiar adaptations: an unusually AT-rich genome, a relative paucity of specific transcription factors and limited heterochromatin. Distinct transcription profiles mark the parasite's different stages. Epigenetic control has emerged as an important contributor to its survival and virulence. *Plasmodium falciparum* histones are marked dynamically with a vast array of post translational modifications. Activating histone marks are present in abundance. Among the repressive marks, H3K27 methylation is absent and H3K9me3 is present in the limited heterochromatic regions. H3K36 trimethylation, a histone mark typically associated with active transcription, is also enriched at the heterochromatin regions and regulates transcriptional repression of a group of variant surface antigen genes in *Plasmodium falciparum*, which play a key role in immune evasion and host-pathogen interactions. H3K36 dimethylation, which shows a much broader distribution across the genome, has been implicated to play a role in global transcriptional repression. The histone methylation landscape is governed, in part, by a group of writer proteins, the functions and substrate and product specificities for many of which remain to be determined. Our work involves identifying *Plasmodium falciparum* SET domain proteins involved in H3K36 methylation and to understand their role in transcriptional regulation.

FT 2

Understanding the Role of Tyrosine Kinase-Like Protein (TKL3) in the *Plasmodium berghei* Life Cycle

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The *Plasmodium* parasite, responsible for malaria, has a complex life cycle that involves both a vertebrate host and a mosquito vector. Transmission of malaria parasites to mosquitoes is initiated by sexual stage maturation, zygote formation, ookinete differentiation, and oocyst formation that occurs in the mosquito midgut 1. These processes are highly regulated by protein phosphorylation. Here, we found that *Plasmodium berghei* tyrosine kinase-like (TKL3) is expressed in gametocytes and ookinete and localizes to the cytoplasm. The targeted deletion of the TKL3 gene impacts the fitness of blood stage parasites, impairs the parasite's ability to form fertile male gametes, and, consequently, affects ookinete differentiation. Oocyst production is reduced by 70–90%; however, sporozoites that are produced exhibit morphological features identical to those of the wild type, infect hepatocytes normally, and mature into hepatic merozoites. TKL3 KO sporozoites exhibit a delay in prepatency, attributed to a slower blood growth phenotype. Our findings indicate that TKL3 regulates asexual blood stage propagation and facilitates the transmission of parasites between their insect and mammalian hosts.

Novel PA1-Like Protein Homologue from *Taenia solium* Drives Cellular Rewiring of Glucose Metabolism and Immune Responses Underlying Anti-Inflammatory Effects

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Taenia solium, the pork tapeworm, is the causative agent of Neurocysticercosis (NCC), responsible for over 70% of acquired epilepsy cases in endemic regions. Its excretory-secretory proteome (ESP) interacts directly with the host immune cells, yet a vast plethora of molecules still remain uncharacterised. To delve into the molecular complexity of ESPs, we performed proteomic analysis of ESPs, identifying 247 proteins with both pro- and anti-inflammatory potential, ranging from 4–88 kDa. Among these, we focused on a novel PA1 like protein homologue. We investigated the role of PA1 in macrophage metabolism and immune modulation. The quantification of mRNA transcripts and corresponding protein levels was determined using qRT-PCR and immunoblot analysis. The internalization assay revealed efficient uptake and uniform distribution of PA1 in differentiated THP-1 macrophages. Notably, PA1 decreased glucose uptake by reducing GLUT1 expression, disrupting cellular glucose homeostasis followed by the induction of M2 phenotype in macrophages and impairment of autophagic flux. Furthermore, thermal stability of the protein was assessed through MD simulations and temperature sensitivity assays. Collectively, these findings identify PA1 as a stable ESP effector capable of rewiring macrophage metabolism and modulating immune responses, providing novel insights into host–parasite interactions during *T. solium* infection.

Molecular Surveillance of *pfhrp2* and *pfhrp3* Deletions in *Plasmodium falciparum* from Nine Malaria-Endemic States in India in 2023–2024

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Plasmodium falciparum rapid diagnostic tests (RDTs), widely used in India, rely on detection of histidine-rich protein 2 (HRP2). Deletions in *pfhrp2* and *pfhrp3* genes can lead to false-negative RDT results, undermining malaria control strategies. Continuous surveillance of these deletions is essential for early detection of emerging "diagnostic resistance". This study aimed to assess extent of *pfhrp2/pfhrp3* deletions across nine malaria-endemic regions in India. A cross-sectional study (Sept 2023–April 2024) was conducted at 12 sites representing varied transmission settings. Of 10,290 participants screened by HRP2-based RDTs and microscopy, RDT-negative but microscopy-confirmed *P. falciparum* cases were tested for *pfhrp2/ pfhrp3* deletions. A literature review and meta-analysis of Indian studies was then undertaken. Among 1,025 microscopy-confirmed *P. falciparum* mono-infections, 19 (1.9%; 95%CI: 0.2–3.5%) were RDT-negative and were confirmed to harbor dual pfhrp2/pfhrp3 deletions. *Pfhrp2* deletion was highest in Lawngtlai-Mizoram reported in 5.1% (9/176) [95%CI: 2.7%–9.4%] followed by South Tripura-Tripura [4.6% (1/26), 95%CI: 0.8%–21.8%], West Singhbhum-Jharkhand [3.2% (8/248), 95%CI: 1.6%–6.2%], Kothagudem-Telangana [0.96% (1/104), 95%CI: 0.2%–5.3%], and no deletions were reported in the remaining eight sites. The literature review identified eight studies from India; pooled meta-analysis estimated a *pfhrp2* deletion prevalence of 3.0% (95%CI: 1–6%), *pfhrp3* at 2% (95%CI: 1–7%), and dual deletions at 1% (95%CI: 1–3%). *Pfhrp2* deletion in *P. falciparum* from Lawngtlai-Mizoram exceeded the WHO's policy threshold of 5%, while all other sites remained below it. Continued surveillance is essential to track and manage the impact on malaria diagnosis.

FT 5

Cardiolipin Synthase is Required for Maintaining Mitochondrial Function During Blood and Liver Stage Development of *Plasmodium*

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Cardiolipin synthase (Cls) is a crucial enzyme in the biosynthetic pathway of cardiolipin, a distinctive dimeric phospholipid essential for the structural and functional integrity of mitochondrial membranes. In most eukaryotes, including humans, Cls is classified as a eukaryotic-type enzyme that catalyzes cardiolipin formation via the condensation of CDP-diacylglycerol and phosphatidylglycerol (PG). In contrast, *Plasmodium* species encode a bacterial-type Cls, structurally and mechanistically distinct, which localizes to the inner mitochondrial membrane and mediates the reversible condensation of two PG molecules, yielding cardiolipin and glycerol. Previous studies have implicated bacterial-type CLS as essential for mitochondrial function and parasite survival; however, its role across the *Plasmodium* life cycle remains largely uncharacterized. In this study, we investigate the role of Cls in the rodent malaria parasite *Plasmodium berghei* and demonstrate its essential role in parasite mitochondrial function and life cycle progression. *Pb*Cls is expressed in both blood and liver stages and localises to the mitochondria. Genetic disruption of *Pb*Cls significantly impairs asexual blood-stage proliferation. While *Pb*Cls-deficient parasites complete development within the mosquito and generate sporozoites capable of hepatocyte invasion, they exhibit a severe defect in liver-stage maturation and a significant delay in the prepatent period, highlighting a stage-specific requirement for cardiolipin synthesis during hepatic development. Further, mitochondrial analysis of *Pb*Cls KO parasites reveals a marked reduction in mitochondrial membrane potential and a significant reduction in mitochondrial branching. These results establish *Pb*Cls as a vital component of malaria parasite development and highlight the critical role of Cls in maintaining mitochondrial function.

Isolation and Taxonomic Characterization of Some *Trichodinid* Ciliated Parasites (Protozoa: *Ciliophora*) from Some Freshwater Edible Fishes of West Bengal

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Trichodinid ciliates are the organisms belonging to Subkingdom Protozoa, which can act as parasites or symbionts of both freshwater and marine fishes (Van As and Basson, 1989). They can be identified by their unique ciliary structures. In order to explore the diversity of trichodinid ciliated parasites among the freshwater edible fishes from some selected regions of West Bengal, several species of freshwater fishes belonging to different family have been examined. The survey was conducted from November, 2023 to January, 2025. Among the several species of fishes, five species were found to host the trichodinid ciliates belonging to genera *Trichodina* Ehrenberg, 1830; *Tripartiella* Lom, 1959; *Trichodinella* Sramek-Husek, 1953 and *Paratrichodina* Lom, 1963. This study involves the re-description of the taxonomy of the parasites highlighting the morphological variation observed. This study also provides the new records of geographical distribution and the new host fish for the parasites and also gives a comprehensive analysis to unveil the diversity of the parasite.

Mitochondrial Genome Variability and Potential Drug Resistance in *Theileria annulata* from Indian Cattle

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Bovine tropical theileriosis, caused by *Theileria annulata*, is a major constraint to cattle health and productivity in India. Buparvaquone (BPQ) remains the primary therapeutic option, yet increasing reports of treatment failure raise concerns about the emergence of drug resistance. To explore the genetic basis of persistent infections, we undertook a longitudinal field study in a small-holding farm in Pune district, Maharashtra, where cattle had exhibited chronic clinical signs of theileriosis for 6–8 months despite repeated BPQ treatment. Blood samples were collected from affected animals in two phases, separated by approximately one year, and subjected to molecular analysis. Gene-specific amplification and sequencing of *cytb*, *dhodh*, and *pin1* confirmed *T. annulata* as the causative agent. While the *dhodh* and *pin1* sequences showed limited variation relative to the reference genome, the *cytb* gene revealed an unusually high level of polymorphism, with >50 mutations detected in several animals. Notably, the persistence of these variants upon repeat sampling and their apparent transmission within the herd underscores the potential spread of mutant genotypes. In parallel, the nearly complete mitochondrial genome (~5.9 kb) was successfully amplified in overlapping fragments and sequenced, revealing that genetic variation was not restricted to *cytb* but extended to other loci within the mitochondrial genome. These findings suggest that mutations in the *cytb* gene may be linked to reduced BPQ efficacy, as has been reported in related apicomplexan parasites, and raise the possibility of novel mitochondrial adaptations emerging in field populations of *T. annulata*. Ongoing work is directed at characterising the functional relevance of these mutations and understanding their role in the persistence and transmission of drug-refractory infections. Insights from this study will be critical for informing surveillance strategies and guiding sustainable control of theileriosis in Indian livestock.

Masters of Manipulation: Unmasking the *Theileria* Secretory Proteins in Host Cell Transformation

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Theileriosis is a tick-borne parasitic disease caused by protozoa of the *Theileria* genus, leading to severe economic losses in livestock production. *Theileria annulata,* which is transmitted by ticks of the *Hyalomma* species, is the main causative agent of theileriosis in India. Following transmission, the parasite invades host immune cells, including B-lymphocytes, monocytes/macrophages, and dendritic cells, and manipulates host signaling pathways such as NF-κB and AP-1. This results in uncontrolled proliferation, inhibition of apoptosis, and a cancer-like transformation of infected cells. *Theileria*-induced host cell transformation is completely reversible upon buparvaquone (a known drug) treatment. It is hypothesized that *T. annulata* secretes effector proteins that mediate host cell transformation through specific host–parasite protein interactions. In this study, *in silico* analysis identified several secretory parasite proteins, three of which were cloned, expressed, and purified. Their subcellular localization was determined, and one of the parasite proteins was found to interact with a host protein through a pull-down assay. Further, this interaction was confirmed using yeast two-hybrid analysis. The identification of these novel parasite–host protein interactions provide insight into the molecular mechanisms underlying host cell transformation. It may reveal potential therapeutic targets for the control of tropical theileriosis.

α-Synuclein Overexpression Influences *Toxoplasma gondii* Infection and its Mediated Neurodegeneration

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Neurodegeneration is a pathological condition that includes loss of structure and function of neuronal cells resulting in functional defects in central nervous system. Among various factors in neurodegenerative instigations, pathogenic infections and misfolded protein aggregations are major risk factors for causing neurodegenerative disorders including Alzheimer's and Parkinson's disease. α-synuclein is a pre-synaptic neuronal protein involved in many functions majorly synaptic plasticity, neurotransmitter release, transcriptional regulation and immunological responses. α-synuclein can change its conformation under different types of stress and dysregulation of α-synuclein leads to neurotoxicity due to loss of function, and causes widespread changes in gene expression which transforms into development of multiple neurological disorders including Alzheimer's, Parkinson's, and Huntington's disease etc. *Toxoplasma gondii* is a neurotrophic parasite which causes toxoplasmosis with serious neuropsychiatric symptoms and altered neurotransmission in infected hosts. In infectious diseases, it is assumed that α-synuclein plays an important role in pathogenesis of infection-mediated neurodegeneration. However, the role of host cell α-synuclein in altering the expression of various genes involved inc ell survival and or neurodegenerative pathogenesis during *T. gondii* infection has not been elucidated. This study majorly focused on the expression and over expression analysis of α-synuclein during *T. gondii* infection. We have observed altered expression in the genes related to α-synuclein and over expression of α-synuclein influences the *T. gondii* infection in neuronal cells. The mechanism, and expression/aggregation patterns of α-synuclein during *T. gondii* infection is under investigation. This study may unravel cues to understand the effect of *T. gondii* infection in altering various cellular mechanisms leading to neurodegeneration.

Expression and Purification of Recombinant *Babesia gibsoni* Secretory Antigen 3 (rBgSA3) for Diagnosis of Canine Babesiosis

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Canine babesiosis is caused by species of tick transmitted intra-erythrocytic parasitic protozoa from the genus *Babesia*. *B. gibsoni* and *B. vogeli* are the two important pathogenic species of dogs in India and South Asia. *B. gibsoni* is more common and pathogenic than *B. vogeli*. The disease caused by *B. gibsoni* is also referred to as small piroplasmosis. *B. gibsoni* often results in chronic and subclinical infections that are difficult to detect via traditional blood smear microscopy. Early and accurate differential diagnosis is important since treatment and prognosis of both infections are different. Hence, molecular and serology based assays are often used for being more specific and sensitive than traditional microscopic methods. This study was conducted to express, purify and characterize the secretory antigen 3 of *B. gibsoni* for further application in serodiagnosis. The dog blood samples received from veterinarians for diagnosis were confirmed for *B. gibsoni* infection by PCR and microscopic examination. The *B. gibsoni* positive blood samples were used for extracting DNA. A 1771bp fragment of the intronless *bgsa3* gene was amplified by PCR. Following a 906 bp fragment of intronless *bgsa3* gene was amplified by nested PCR. This fragment was sequenced, analyzed and further cloned and ligated in to pET32a expression vector. The cloned recombinant plasmid was further sequenced for confirmation. Optimum recombinat BgSA3 (rBGSA3) expression was achieved in *Esherichia coli* BL-21 cells, 4 hours after induction with 06µM IPTG as a soluble protein. The His tagged rBgSA3, was purified by cobalt affinity chromatography. Purity and molecular weight were confirmed by SDS-PAGE (less than 63 kDa protein marker); its functional activity was confirmed by immunoblot. The specificity of the rBgSA3 was confirmed by dot blot assay indicating its potential as a diagnostic antigen.

Exploring the Therapeutic Potential of Dipeptidylcarboxypeptidase Inhibitor and Amphotericin B against Experimental Visceral Leishmaniasis

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Visceral leishmaniasis (VL) is the most severe form of disease, difficult to treat clinically with limited drug options having serious drawbacks (1). In recent trend, combination therapy has gained a lot of attention than existing monotherapy regimens with the goal of identifying effective, safer and shorter treatments (2). In present study, we explored the therapeutic potential of the compound 98/288, a *Ld*DCP inhibitor (3) with amphotericin B, a polyene antifungal drug repurposed for VL with excellent cure rate (100%). The *in vitro* drug interaction study revealed that the combination exhibited synergistic effect on both the stages of the parasite. The combination also exhibited a significantly higher *in vivo* efficacy than the monotherapies in experimental golden hamster model. Interestingly, in addition to increased efficacy, the combination trial also showed potential immunostimulatory effect by enhancing host immunity. Thereby this study establishes that the combination of 98/288 and amphotericin B is much more effective than monotherapy due to its immunotherapeutic activity and an effective strategy for treating VL. As a result, this study can be an part of successful endeavor in achieving the goal of elimination of VL in India.

Wuchereria bancrofti Serpin Wb123 Orchestrates uPAR-Dependent Alternative Macrophage Activation to Facilitate Filarial Immune Evasion

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Following the 2025 Nobel Prize in physiology or medicine honouring groundbreaking advances in regulatory T cell (Treg) biology and immunology, the intricate mechanisms of immune evasion by filarial parasites have garnered renewed interest. These persistent filarial nematodes exploit alternatively activated macrophages (AAMs) and Treg responses to undermine host immune defences. However, the molecular determinants driving filaria-induced AAM activation remain poorly defined. Through in-silico analysis, we identified fifteen putative filarial serine protease inhibitors (serpins), among which *Wuchereria bancrofti* serpin Wb123 emerged as pivotal immunomodulator. Recombinant Wb123 (rWb123) potently induced AAM activation, marked by elevated expression of CD163, arginase-1, IL-6 and pSTAT3, while profoundly suppressing lipopolysaccharide (LPS) and interferon-gamma (IFN-γ) driven classical macrophage activation. Strikingly, neutralization of rWb123 with the monoclonal antibody MAbG8 or targeted blockade of the urokinase plasminogen activator receptor (uPAR) abolished rWb123-mediated AAM induction, restoring proinflammatory responses to LPS-IFN-γ. These findings unveil Wb123 as a master regulator of uPAR-dependent AAM activation, illuminating a novel immune evasion mechanism that parallels Treg-mediated suppression highlighted in recent Nobel-recognized discovery. By pinpointing filarial serpins and uPAR as pivotal therapeutic targets, this study paves the way for innovative immunotherapies aimed at strengthening host immunity and eradicating filarial infections, aligning with the cutting-edge frontiers of immunological research.

A Novel *Plasmodium* Protein, PEPT is Critical for Maintaining Sporozoite Shape and Infectivity

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Malaria remains a major public health problem, causing 241 million cases and 627,000 deaths in 2022. Resistance to front-line antimalarial is rising; hence, newer treatment strategies are urgently needed. To understand the function of a novel protein, a hypothetical gene whose transcripts were highly upregulated in sporozoites was selected. Endogenous tagging of the gene with 3XHA revealed expression in blood, ookinetes, sporozoites, and exo-erythrocytic forms (EEFs). PEPT was localized on the parasite's membrane. Targeted disruption of PEPT had no effect on asexual blood stage propagation and the formation of gametocytes. We found normal development of PEPT KO parasites in mosquitoes. PEPT KO sporozoites showed reduced infectivity in mice. Observing PEPT KO sporozoites on day 18-20 post-feeding, we observed a loss of regular shape and bulb-like structures. These sporozoites were found to be impaired in their capacity to glide and a reduced number of merosomes were observed. We quantified PVM rupture, which was found to be impaired in PEPT KO parasites. Collectively, these results suggest that PEPT contributes to sporozoite shape maintenance and liver-stage infection. Our results have important implications for vaccine development.

FT 14

Discovery of Antimalarial Compounds from *Aegle marmelos* and *Syzygium aromaticum* Through *in vitro* and *in silico* Approaches

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Aegle marmelos (Rutaceae) and *Syzygium aromaticum* (Myrtaceae) are traditionally used in Indian medicine for various ailments, but their antimalarial potential remains underexplored. This study assessed the antimalarial efficacy and cytotoxicity of fractions and purified compounds from *A. marmelos* leaves and *S. aromaticum* flower buds using *in vitro* and *in silico* approaches. Standard techniques evaluated methanolic extracts and fractions (hexane, chloroform, ethyl acetate, methanol) against *Plasmodium falciparum* 3D7 (chloroquine-sensitive). HeLa cell cytotoxicity was determined using MTT assay. Structural characterization employed chemical tests and NMR. Molecular docking (AutoDock Vina, Maestro v12.8) and pharmacokinetic/toxicity predictions (SwissADME, ProTox-II) were performed. Methanolic extracts of *A. marmelos* and *S. aromaticum* showed significant inhibitory effects with IC_{50} values of 7.00 and 6.25 µg/mL, respectively. Lupeol exhibited the strongest activity (94.0% inhibition, IC_{50} 5.2 µg/mL), while β-sitosterol, 1,3-Dimethylpyrimidine-2,4(1H,3H)-dionein, and eugenol showed moderate activity (IC_{50} 12.6, 18.0, and 10.6 µg/mL, respectively). Chloroquine achieved 96.0% inhibition at 5 µg/mL. Lupeol showed low cytotoxicity (TC_{50} >100 µg/mL), while other compounds exhibited mild toxicity. Molecular docking revealed strong binding affinities of lupeol (−9.5 kcal/mol) and β-sitosterol (−9.4 kcal/mol) with ribosomal targets, supported by favourable drug-likeness and safety profiles. These findings scientifically validate the traditional use of *A. marmelos* and *S. aromaticum* as promising antimalarial sources.

Heterologous Expression of *Plasmodium falciparum* Artemisinin-Resistance Determinant Reveals a Novel Function as Oxidative Stress Sensor Under Heme-Regulated Proteostasis

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Resistance to the frontline artemisinin drugs (ART-R) elicit complex homeostatic changes that govern parasite's tolerance for ART-induced oxidative-damage and PfKelch13 (K13) remain the only confirmed *P. falciparum* ART-R determinant. K13 shares closest homology to human KEAP1, a well-characterized-oxidative-sensor. While the molecular mechanisms underlying oxidative stress response via Nrf2/KEAP1 interactions are well characterized in mammalian systems, the homologous components are *hitherto* unknown in *P. falciparum*, which encounters disproportionate amounts ROS via the oxidation of iron ion of host hemoglobin. Therefore, heterologous expression of K13 in a model non-erythroid mammalian cell line (Du145) with reduced levels KEAP1, reveal K13 mimics KEAP1 under H_2O_2 treatment by suppressing Nrf2. In addition, in presence of heme K13 dislodges Nrf2 and facilitates nuclear translocation to induce pro-survival. Thus, after validating Nrf2 as a *bona fide* interacting partner of K13, we next compared PfKelch13 *vis-à-vis* the KEAP1 interactome by using proximity biotinylation (BioID) which revealed only minor overlap (7-12%). Relative differences between K13 and KEAP1 interactomes and the enrichment of vesicle localization/lysosomal transport pathways in GO analysis further encouraged us to inspect the degradation mechanism which reveal that affinity for heme is critical for K13 stability, prevents autophagic removal, increases oxidative stress and ART susceptibility. Together, our study defines heme as a novel K13 substrate for sensitivity to ART and the power of alternate cellular models in studying frontiers of pro-oxidant stress.

Transfusion-transmitted *Plasmodium* spp. Infections and Safety Challenges for Malaria in the Indian Subcontinent: A Systematic Review

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Blood transfusion is a globally life-saving intervention, but blood-borne pathogens can threaten its effectiveness. While blood bags are systematically screened for viral and bacterial pathogens, parasite infections are generally overlooked. In this review, we analysed the current literature on transfusion-transmitted malaria (TTM) in India over the past five decades. This analysis is based on 122 studies involving more than 6.5 million individuals. The prevalence of *Plasmodium* parasitaemia in donors ranged from ~0% to 0.87% by light microscopy and ~0% to 2.3% by rapid diagnostic tests. The proportion of post-transfusion malaria (PTM) cases ranged from 0.8% to 6.8% across the studies. The risk of PTM is both time- and diagnosis method-dependent and relatively high in some regions of India. The clinical impacts of PTM range from mild to severe and even fatal outcomes. It is also crucial to address TTM given the often-neglected *Plasmodium malariae*, in addition to the prevalence of *Plasmodium vivax* and *Plasmodium falciparum*. The spread of drug-resistant and/or *pfhrp2* gene-deleted *P. falciparum* parasites is another threat in PTM. Blood screening could be achieved through point-of-care nucleic acid amplification techniques to guarantee safer transfusion. If neglected, TTM can become an obstacle to malaria elimination in the coming years.

Mitochondrial-Cytosolic [Fe-S] Cluster Trafficking and Protein Interactions in *Plasmodium falciparum*

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[Fe-S] clusters, which have critical roles in electron transfer reactions and catalytic properties of many important enzymes, are assembled by biogenesis through the ISC pathway in mitochondria. The cytosolic CIA pathway is dependent on mitochondrial ISC. Our laboratory previously identified the *Pf*ATM1 homolog localized to the mitochondrial membrane[1]. To probe transport of [Fe-S] from the mitochondria to the cytosol, we probed the interaction of *Pf*ATM1 with the putative CIA constituent *Pf*Nbp35. Immunofluorescence assay using antibodies generated against *Pf*Nbp35 showed its localization at the mitochondrial surface. *In vitro* interaction and pull-down experiments showed that the N-terminal extension of *Pf*ATM1 interacted with *Pf*NBP35. Both *Pf*Nbp35 and *Pf*Cfd1 (HCF101) bound [4Fe-4S] and were functional ATPases, an activity required for [Fe-S] transfer to carrier protein(s) of the CIA pathway. *Pf*Nbp35 could pull down *Pf*Cfd1/HCF101 from parasite lysate indicating their interaction. Since ATM1 knockdown in *Toxoplasma gondii* upregulates the NEET protein CDGSH1 while *Tg*ATM1 upregulation decreases CDGSH1 and CDGSH2 levels[1], we probed localization of the NEET proteins in *Plasmodium*. *Pf*CDGSH1 localized in proximity to mitochondria suggesting the possibility of NEET-*Pf*ATM1 crosstalk. Our results indicate that *Pf*Nbp35 and *Pf*Cfd1 are components of the *P. falciparum* CIA pathway, bind [4Fe-4S] with *Pf*Nbp35 directly interacting with *Pf*ATM1.

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Computational Dissection of the *Brugia malayi* Transcriptome Reveals Stage-and Sex-Specific Expression Patterns

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Brugia malayi, a causative agent of lymphatic filariasis, represents a major neglected tropical disease affecting millions of individuals worldwide. The ~90 Mb genome of *B. malayi*, encoding approximately 11,500 protein-coding genes, has provided valuable insights into its evolutionary relationship with *Caenorhabditis elegans* and has revealed conserved operonic organisation and adaptations for parasitism and symbiosis with the Wolbachia endosymbiont. However, the stage-specific and sex-related regulation of gene expression throughout its life cycle remains poorly characterised. In this study, RNA-sequencing data from eight developmental stages, early embryo, immature and mature microfilariae, infective third-stage larva (L3), fourth-stage larva (L4), adult male, adult female, and newborn microfilaria, were analysed using RPKM-normalised expression values. Distinct transcriptional profiles were observed across developmental transitions, with enrichment of genes involved in moulting, cuticle synthesis, and metabolism during larval stages, and reproductive and immune-related genes in adults. Comparative analysis revealed pronounced sex-biased expression associated with spermatogenesis, oogenesis, and hormonal signalling pathways. Functional enrichment further indicated dynamic regulation of metabolic and stress-response pathways. These findings provide a comprehensive overview of the transcriptional landscape of *B. malayi*, advancing our understanding of its developmental biology and identifying potential molecular targets for anti-filarial drug discovery.

Gender Biased Regulatory Role of m^{6}A RNA Modification in Human Parasite *Schistosoma japonicum*

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N6-Methyladenosine (m^{6}A), one of the most abundant post-transcriptional modifications in eukaryotic mRNAs, plays a pivotal role in regulating RNA metabolism. However, its functions in *Schistosoma japonicum,* a parasitic flatworm with a complex lifecycle involving dynamic transcriptional reprogramming, remain entirely unexplored. Although DNA methylation has been extensively studied in helminth development, the epitranscriptomic landscape is poorly understood. Here, we report the first evidence of m^{6}A methylation in *S. japonicum* and demonstrate its critical role in parasite biology. We showed that the m^{6}A writer complex METTL3-14 controls female reproductive development, including ovary morphogenesis and egg production, and orchestrates metabolic reprogramming essential for survival in male worms. Given the central role of eggs in host pathology and transmission, our findings uncover a key regulatory layer in schistosome biology and pinpoint METTL3-14-directed m^{6}A deposition as a potential target for anti-schistosomiasis interventions.

Unveiling Haplotype Distribution of Bovine *Cryptosporidium* Isolates In North India Inferred by 18S rRNA sequences

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Cryptosporidium is an enteric apicomplexan parasite of a variety of mammals including cattle and humans. A study was conducted to detect and identify the *Cryptosporidium* species in bovines of north India, to conduct risk factor analysis and to evaluate the haplotype distribution and genetic diversity among the detected species. A total of 1417 bovine faecal samples were randomly collected from 21 districts of three north Indian states: Himachal Pradesh, Uttarakhand and Uttar Pradesh. Faecal microscopy detected 11.8% infection rate where animal age and diarrhoea were identified as potential risk factors for disease occurrence. *Cryptosporidium andersoni, C. bovis, C. parvum* and C. *ryanae* were the detected species. The phylogenetic analysis revealed that all the analysed bovine *Cryptosporidium* isolates formed a monophyletic clade. Broadly, *C. parvum, C. bovis* and *C. ryanae* isolates were clustered together while *C. andersoni* isolates have formed a sister clade to these three species. A haplotype network indicated 34 haplotypes globally within four *Cryptosporidium* species from Asian, African, American, European and Australian isolates of bovine host. These haplotypes were classified into four distinct species-specific haplogroups. The overall bovine *Cryptosporidium* population dataset has depicted high genetic diversity ($\pi = 0.03625 \pm 0.00233$, $Hd = 0.88674 \pm 0.01517$).

Proteomic Characterization of Exosomes Derived from the Neglected Zoonotic Trematode *Artyfechinostomum malayanum*

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Artyfechinostomum malayanum is an emerging zoonotic trematode of public health concern, yet little is known about its biology and host interaction mechanisms. Extracellular vesicles (EVs), particularly exosomes or small extracellular vesicles, have recently been recognized as critical mediators in parasite-host communication, carrying a wide array of bioactive molecules including proteins, lipids, and nucleic acids. This study focuses on the proteomic profiling of exosomes derived from *A. malayanum*, aiming to elucidate their composition and identify potential biomarkers for identification and diagnosis. EVs were isolated from *in vitro* parasite culture media and subjected to further sub-fractionation, followed by LC-MS/MS analysis. Identified proteins revealed a diverse repertoire, including enzymes, transporters, immune-modulatory proteins, and exosomal markers. Gene Ontology analysis showed significant representation in cellular component categories like cytoplasm, membrane, and extracellular regions; biological processes such as metabolic activity and host modulation; and molecular functions including ATP binding and metal ion binding. Notably, a subset of proteins uniquely enriched in *A. sufrartyfex* exosomes and others conserved across trematode species are identified, suggesting their potential as candidate biomarkers for infection. This comprehensive proteomic characterization offers novel insights into the biology of *A. malayanum* and lays the foundation for future diagnostic tool development.

β-Catenin Modulates Macrophage Alterations to Regulate their Immunogenic Response

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Toxoplasma gondii exploits a host-intrinsic β-catenin pathway in macrophages to bypass absent TLR11/12 sensing in humans. We show that by activating PI3K-AKT-ß-catenin, the parasite drives ROS signaling and PINK1/PARKIN-mediated mitophagy, fostering replication. ß-catenin also stabilizes HIF-1a inducing pro-inflammatory M1 polarization. In contrast, genetic or pharmacologic ß-catenin ablation shifts macrophages toward an M2 phenotype, limiting parasite burden and highlighting ß-catenin as a key therapeutic target.

Taxonomic Evaluation of Digenetic Trematodes Infecting Small Ruminants

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Amphistome parasites are a major problem in small ruminants, especially in areas where environmental contamination is high. These are members of the Paramphistomatidae family, live in the reticulum and rumen and can cause serious gastrointestinal disorders, decreased productivity, and nutritional stress. It is difficult to identify amphistomes from systematic point of view because they are morphologically alike. In natural infections, these parasites often exhibit overlapping morphological characteristics, and the occurrence of mixed infections further complicates accurate identification. Closely related species share similar external morphology, leading to misidentification and underestimation of true diversity. Such limitations highlight the inadequacy of morphology alone for precise taxonomic resolution. To overcome these challenges, molecular characterization has become an essential complement to morphological observations. Recent advances in DNA technology now permit the rapid and reliable characterization of the genome thus providing a new tool for the identification of species and sub-species. Gene sequencing and PCR-based identification have made it possible to precisely characterise species and do phylogenetic analysis at the molecular level like 18s/28s/ITS2 and COI that have been helpful in identifying species. The present study emphasizes the necessity of this morpho–molecular approach for accurate identification of amphistome species in small ruminants, facilitating better understanding of their diversity, infection dynamics, and control measures.

Strengthening Vaccine-Induced Immunity: NOD2 Agonist Enhances Recombinant 78 kDa Antigen Response Against Visceral Leishmaniasis

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NOD-like receptors (NLRs) are critical cytoplasmic sensors essential for detecting and controlling intracellular pathogens such as *Leishmania* spp. Among them, NOD2 is well-characterized for mediating parasite recognition, intracellular killing, and coordinating both innate and adaptive immune responses. Despite progress, the understanding of NLR-mediated sensing of *Leishmania* remains incomplete. In this study, we investigated a second-generation vaccine formulation comprising the recombinant 78 kDa *Leishmania donovani* antigen (r78), administered alone or in combination with the NOD2 agonists desmuramylpeptide (DMP) or muramyldipeptide (MDP). BALB/c mice were immunized subcutaneously and challenged intravenously with 1×10^7 promastigotes. Immune responses and splenic parasite burdens were subsequently evaluated. The r78+DMP formulation elicited pronounced antigen-specific T-cell proliferation and a Th1-biased immune response, characterized by elevated reactive oxygen species, increased nitrite levels, and up-regulation of Th1-associated cytokines. This response coincided with reduced Th2 cytokines and a substantial decline in splenic parasite load, achieving up to 95% reduction at 8 weeks post-challenge. In contrast, r78+MDP conferred moderate protection, while r78 alone yielded minimal reduction comparable to un-vaccinated controls. Together, the results show that r78 + DMP elicits strong, coordinated immune protection, highlighting NOD2-based adjuvant integration as a promising strategy for next-generation visceral leishmaniasis vaccines.

Poster Presentations

P 001

A Novel Multitargeting Approach to Develop Resistance-Immune Antimalarials

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The persistent emergence of resistance to frontline antimalarials poses a significant challenge in malaria-endemic regions, primarily driven by mutations in drug targets and transporters. In light of this, developing multitargeting drugs has emerged as a promising solution. Since the likelihood of simultaneous mutations across multiple targets is extremely low due to fitness costs, multitargeting drugs could serve as "magic bullets," offering reduced dosages, enhanced efficacy, fewer drug-drug interactions, and improved safety. Among potential targets, kinases are particularly attractive as they regulate various stages of the *Plasmodium falciparum* life cycle. In our study,~2,000 kinase inhibitors were screened by high-throughput virtual screening (HTVS) against six validated *Pf* kinases (*Pf*PKG, *Pf*MAP2, *Pf*CDPK4, *Pf*TMK, *Pf*PK5, *Pf*PI4K). The MMGBSA and ADME analyses identified 21 promising multitargeting candidates, and hierarchical clustering revealed their structural divergence from existing antimalarials. The top six complexes' molecular dynamics simulations (MDS) confirmed stable interactions. The parasite growth inhibition assays (GIA) showed quercetin (IC_{50} 1.84µM) and myricetin (IC_{50} 3.93µM) as potent hits. Expanding the search, HTVS of ~150,000 kinase inhibitor compounds identified 125 hits, of which the top eight were selected for GIA (ongoing). The MDS confirmed stable interactions of these compounds with their targets. Two compounds have demonstrated potent activity (IC_{50} ~700nM and 4 µM) and were selected for further *in vitro* and *in vivo* toxicity evaluation. Five kinases have been expressed and purified, and their binding to related compounds was validated by biolayer interferometry. Compounds successful in GIA are being tested in kinase inhibition assays.

P 002

Unveiling The Role of Dithiol Glutaredoxin Protein in Drug Resistance, Parasite Survival, and Oxidative Stress Tolerance in *L. donovani*

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Leishmania donovani, the causative agent of visceral leishmaniasis, deals with several treatment challenges due to limited drug options, asymptomatic infections, and drug resistance. Managing redox metabolism is one of the major promising approaches for drug design. Glutaredoxins (Grxs) are low-molecular-weight oxidoreductases that play a significant role in cellular redox homeostasis. In the present study, we characterized the Grx1 protein of *L. donovani* (LdGrx1) and examined its localization, biochemical properties, as well as its role in drug resistance, parasite survival, and oxidative stress tolerance. The LdGrx1 protein was cloned, expressed, characterized, and its expression was evaluated in *L. donovani* Amp-B-sensitive Ag83 promastigotes. Further, the Grx1-overexpressor (Grx1-OE) parasite was generated, which showed increased tolerance to the Amp-B drug, upregulated antioxidant enzymes, decreased intracellular ROS, and had a corresponding increase in thiol levels. The upregulation of thiol pathway proteins in the Grx1-OE parasites indicates a key interaction between Grx1 and thiol pathway proteins in combating oxidative stress and maintaining redox homeostasis. The *in-vitro* assessment of THP-1 macrophage infectivity demonstrated that the LdGrx1 protein enhances parasite infectivity. Therefore, our findings provide the first insight into the role of the LdGrx1 protein in drug resistance, redox homeostasis, and parasite survival in *L. donovani*.

P 003

Taenia solium Cyst Derived Extracellular Vesicles Regulate IP6K1 Expression and Modulate Neutrophil Activity

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The Extracellular vesicles (EVs) present in secretome defines the immunological cascade and escape strategies in cestode parasites like *Taenia solium*. Neurocysticercosis (NCC) is caused by *T. solium* cysticerci. EVs secreted from the *T.solium* attenuate AKT/mTOR signalling pathway in macrophages, thereby modulating host immune responses. Similarly, neutrophils being the first-line defenders of the innate immune system utilize reactive oxygen species in regulating cellular signalling cascades and contributes to NCC immunopathogenesis. However, the role of EVs and neutrophils during NCC remains poorly understood. *T. solium* cyst derived EVs was used to treat differentiated HL-60 cells as well as primary human neutrophils. Gas Chromatography identified several metabolites present in EV associated with AKT/PI3K pathway. We found significant increase in the expression of inositol hexakisphosphate kinase 1 (InsP6K1) gene and less AKT activity, reduced ROS and defective bacterial killing. Our findings suggested that the *T. solium* cyst derived EVs modulate neutrophil function by suppressing the ROS production, upregulating IP6K1 and downregulating AKT-pathway in neutrophils, which may lead to the inhibition of the innate immune cells function and potentially contributing to immune evasion during NCC. Further investigations are required to elucidate the effect of reduced AKT activity on other cellular activity of neutrophils and its role in secondary infections during the NCC.

P 004

Ethnomedicinal Insights into *Piper chaba* as a Suitable Dewormer

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Helminth infections by nematode *Ascaridia galli* and cestode *Raillietina* sp. severely impair fowl health leads to poor growth and economic loss. Increasing resistance against known synthetic drugs highlights the need for plant based alternatives. Our study examined the anthelmintic potential of *Piper chaba* stem-bark methanolic extract in comparison to albendazole. The phytochemical rich extract showed strong efficacy: paralysis and mortality occurred at approximately 5.42 h and 7.66 h in *A. galli* and 3.54 h and 6.42 h in *Raillietina* sp. respectively, closely matching albendazole at same dose. Our extract showed dewormer activity, reducing survival in nematode (65.7%) and cestode (68.9%), closely matching efficacy of albendazole 78.2% and 72.3% respectively relative to DMSO control. Acetylcholinesterase (AChE) activity in excretory-secretory product of nematode markedly increased (5.96 vs. 3.64 µmol/min/mg in control) and showed slight rise in cestode (0.858 vs. 0.748 µmol/min/mg in control) indicating altered neural regulation and stress. Tissue level damage was confirmed by histological studies: cuticle, muscles and uterus were destroyed in nematodes, while cestodes showed scars, proglottid disintegration and villous damage in host tissue. Together, these findings highlight *Piper chaba* as a potent, eco-friendly and resistance-limiting alternative to synthetic dewormers, offering a sustainable solution for fowl parasite management.

P 005

Identification of New Protein Coding Potential in *Leishmania braziliensis* using a Proteogenomic Approach

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Leishmania braziliensis is a kinetoplastid protozoan parasite responsible for American tegumentary leishmaniasis, exhibiting complex genomic architecture and stage-specific adaptations that underlie its pathogenicity. Despite the availability of its reference genome, limitations in gene annotation persist due to the presence of hypothetical proteins, pseudogenes, and unrecognized coding regions. In this study, we employed a proteogenomic approach integrating publicly available high-resolution mass spectrometry data with a custom six-frame translated genome database to refine the genome annotation of *L. braziliensis* strain MHOM/BR/75/M2904. Utilizing stringent database-dependent searches with a 1% false discovery rate, we identified many unique peptides, of which 962 were genome search-specific peptides (GSSPs) mapping exclusively to unannotated genomic regions. These GSSPs facilitated the discovery of 49 novel protein-coding genes and correction of 207 existing gene models, including N- and C-terminal extensions. Our findings demonstrate the power of proteogenomics to uncover cryptic protein-coding regions and improve genome annotations beyond conventional predictions. The study underscores the importance of integrating proteomic evidence with genomic data to capture the full coding potential of kinetoplastid parasites, paving the way for improved diagnostics and interventions against leishmaniasis.

P 006

An Insight into the Taxonomy and Diversity of Protozoan Parasites Infecting Earthworms of Bankura and Purulia Districts of West Bengal

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Gregarines are a group of apicomplexan endoparasites that infect invertebrates, especially arthropods and annelids. Among earthworm populations, acephaline gregarines—those lacking a septum— are especially prevalent. A comprehensive survey spanning January 2024 to August 2025 was undertaken in different habitats from the Bankura and Purulia districts of West Bengal. The present study assesses both the diversity and geographical distribution of gregarines parasitising earthworms along with their seasonal variations, and host specificity. A total of ten distinct aseptate gregarine species belonging to different genera were recorded, out of which three were from *Monocystis* Stein, 1848, two from *Nematocystis* Hesse, 1909, four from *Stomatophora* Drzhevetskii, 1907, and one *Apolocystis* Cognetti de Martiis, 1923. Parasite identification and prevalence were determined through morphometric analysis. It was observed that gregarine infections were most prevalent during the monsoon season (>90%), followed by the post- monsoon period (>80%). Among the identified taxa, *Monocystis* Stein, 1848 showed a markedly higher prevalence compared to other species. These parasites primarily infect the seminal vesicles of their host earthworms. While most gregarine species showed high host specificity, some individual earthworms harboured multiple genera of acephaline gregarines simultaneously.

P 007

Nuclear Function of Phosphoglycerate Mutase 1 Enzyme and Its Possible Autoregulation Mechanism in *Plasmodium falciparum*

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Phosphoglycerate mutase (PGM) enzyme is involved in multiple metabolic pathways including glycolysis and pentose phosphate pathway. *Plasmodium falciparum* PGM1 (PfPGM1) is a tetrameric enzyme expressed in the different parasitic life stages, and its oligomerization is important for its activity both *in vitro* and *in vivo*. Moreover, a fraction of PfPGM1 is found in the nucleus. The presence of a glycolytic enzyme in the nucleus is intriguing and therefore, analysing its role in nucleus is necessary and this laid the foundation of our study. Our sub-cellular fractionation studies showed that PfPGM1 is mostly localized to the cytoplasm but small fraction also goes to the nucleus, in each stage in the asexual cycle of *P. falciparum*. This is also corroborated by our indirect immunofluorescence assays and live cell imaging. Surprisingly, over-expression of wild type PfPGM1 but not an oligomerization mutant form of PfPGM1 as GFP fusion protein, showed down regulation of the endogenous PfPGM1 protein. Further, the transcripts of endogenous PfPGM1 were also downregulated when PfPGM1 was overexpressed as GFP fusion protein as above. ChIP-qPCR analysis showed that affinity of PfPGM1 with its own promoter is significantly higher in wild type overexpression line compare to its mutants. In conclusion, we may suggest the possibility of autoregulation mechanism for PfPGM1 expression in *P. falciparum*.

P 008

The Drug-Resistant *Leishmania donovani* Inhibits Host's Ubiquitin-Mediated Cell-Autonomous Innate Immune Response by Increasing the Expression of *Leishmania* Deubiquitinating Enzymes (*Ld*-DUBs)

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Ubiquitination and deubiquitination are essential processes for host and microbial cell biology and physiology. Defects in these processes can cause various dysfunctions in living organisms. Deubiquitinating enzymes (DUBs) are responsible for removing ubiquitin from target proteins, editing ubiquitin chains, and processing ubiquitin precursors. Interferon-induced ubiquitin immune recognition of intracellular pathogens within pathogen-containing vacuoles (PVs) is a key immune defense against these PV-resident pathogens[1,2]. For example, ubiquitin decoration of PVs containing the protozoan parasite *Toxoplasma* promotes PV destruction and parasite killing[1]. While ubiquitin-mediated host defense against *Toxoplasma* has been extensively studied, the role of ubiquitin-mediated host defense against other protozoan pathogens (e.g., *Leishmania* spp.) remains unclear. We hypothesized that *Leishmania*, as a vacuolar pathogen, can be targeted by the host's ubiquitin system and subsequently delivered to the autolysosomal compartment, thereby limiting parasite growth. Additionally, *Leishmania* may evolve strategies to evade ubiquitin-mediated immune recognition, thereby increasing its virulence. In this study, we discovered that *Leishmania*-containing vacuoles (LCVs) are decorated with ubiquitin and are transported to autolysosomal compartments. Notably, we observed that drug-resistant strains, such as Antimony-resistant *Leishmania donovani* (SbR-*Ld*) strains interfere more with the ubiquitin-mediated cell-autonomous immune response and are more infective than Antimony-sensitive (SbS-*Ld*) parasites. Furthermore, we identified differential gene expression of specific *Ld*-DUBs in SbR-*Ld* versus SbS-*Ld* strains, which are likely important for amastigote proliferation within infected host cells[3]. CRISPR-Cas9-mediated knockout of a couple of these *Ld*-DUBs in SbR-*Ld* led to increased ubiquitin decoration of LCVs, fusion with lysosomal compartments, and restricted parasite growth compared to wild-type SbR-*Ld*. Overall, our findings suggest that *Ld*-DUBs play a key role in inhibiting the host's ubiquitin-mediated immune recognition of LCVs, thus supporting parasite survival and growth. These *Ld*-DUBs could serve as promising targets for future drug development.

P 009

Systematic Review and Meta-Analysis on the Prevalence of *Cryptosporidium* Infection among Children in India in 1985-2024

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Diarrhoea is the second leading cause of death in children under 5-years in India. Overall prevalence of diarrhoea in India stands at 7.3% though some districts recording as high as 20%. *Cryptosporidium* is the third major contributor to the diarrhoea-related morbidity, mortality, growth stunting, malnutrition and cognitive dysfunction in children. Nevertheless, the contribution of *Cryptosporidium* for the increased diarrhoeal burden in India is unclear, underscoring the need for a systematic review to address the existing knowledge gap. Studies reporting the prevalence in Indian children were systematically searched from inception through 2024 following PRISMA guidelines. Out of the 642 articles screened, only 42 met the eligibility criteria and were included in the systematic review. The data extraction process utilized critical data such as prevalence rates, age, sex, sample size, and clinical symptoms. Meta-analysis indicated that 11.29% children are infected with *Cryptosporidium* parasite. A significant heterogeneity among studies is observed with birth cohort studies reporting highest prevalence rate (35.65%). Subgroup analysis based on the age, indicated that the children below and above five years are similarly infected, highlight the need for more studies to determine the accurate burden of cryptosporidiosis, which appears to be a neglected disease in India.

P 010

Rutin, a Flavonoid Glycoside Dampens Filarial-Antigen Induced Pro-Inflammatory Consequences: Immunological and Structural Studies

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Lymphatic filariasis (LF) stands out to be the most incapacitating parasitic ailment resulting from pathogenic filarial nemotodes viz., *Wuchereria bancrofti* in humans, thus eliciting the activation of tumor necrosis factor receptor-1 (TNFR1) and toll-like receptor 4 (TLR4) which activate nuclear factor-κB (NF-κB) signalling. Initial interaction of filarial parasitic antigens is characterized by proinflammatory profile. Mukherjee et.al., have established the recognition and interaction of antigenic surface proteins in the initial phase of host-parasite interaction, perpetuating a cycle of persistent inflammation. In the present study, we employed an integrated approach comprising computational screening and experimental evaluation to identify phytocompounds capable of modulating these immune responses. Among the tested candidates, Rutin, a flavonoid glycoside, demonstrated potent anti-inflammatory and immunomodulatory activity by selectively attenuating TNFR1 and TLR4-mediated signalling. Structural docking analyses further supported the binding affinity of Rutin with these receptors, highlighting its potential mechanism of action. It has been found to enhance drug solubility and anti-inflammatory efficacy which can be formulated to develop nano-formulations, as an alternative approach towards ameliorating chronic inflammatory conditions associated with LF which will bring a social impact by supporting "Swasth Bharat" and contributing towards the Global Programme to Eliminate Lymphatic Filariasis (GPELF), a WHO initiative.

P 011

Characterisation of a Symbiotic Leech, *Batracobdelloides* sp. (*Annelida: Clitellata: Rhynchobdellida*) in a Freshwater Bivalve Mussel *Lamellidens marginalis* (Mollusca: Bivalvia: Unionida) from South 24 Parganas, West Bengal

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A symbiotic leech under the genus, *Batracobdelloides* was isolated and studied from the common freshwater bivalve mussel, *Lamillidens marginalis,* for the period of March 2024 to March 2025. Characterisation was carried out based on their morphology, ultrastructure and gene sequencing studies. PCR amplification was performed from the mtCOI gene using LCO and HCO forward and reverse primers. The leech has rice shaped body, two pair of eyes located on somites III and IV and presence of seven pairs of crop caeca. Scanning Electron Microscopy was performed which revealed the ultrastructure. Evolutionary history was inferred by using Maximum Likelihood method and Tamura-Nei model. Evolutionary analysis and construction of phylogenetic tree was carried out in MEGA 11 software. . Consensus sequence was submitted to the NCBI database and an accession number was provided justifying its uniqueness. This is the first report of the leech from selected study sites in South 24 Parganas, West Bengal.

P 012

Deciphering the Minimal Functional Domain of *Pf*RALP1, an Indispensable Malaria Parasite Protein

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The successful invasion of human red blood cells by *Plasmodium falciparum* merozoites depends on crucial protein-protein interactions (PPIs). Among these, the *Plasmodium falciparum* rhoptry-associated leucine zipper-like protein 1 (*Pf*RALP1) has emerged as a novel protein playing an essential role in the invasion process. However, there are still limited comprehensive studies on *Pf*RALP1. My research aims to provide a deeper understanding of *Pf*RALP1 and its functional significance during merozoite invasion. In this effort, I have identified the minimal functional domain of *Pf*RALP1 that replicates its role in the invasion process. The findings from my research will lay the groundwork for developing drugs or inhibitors that target *Pf*RALP1, thereby effectively preventing merozoite invasion.

P 013

LdZIP3 regulates Autophagy-to-Apoptosis Switch Under Zinc Stress in *Leishmania donovani*

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Visceral Leishmaniasis remains a significant global health crisis as no vaccine is available. Increasing drug resistance presses the need to identify novel molecular targets. *Leishmania* survival critically depends on zinc and its homeostasis. This study investigates the role of zinc importer, *LdZIP3*, in zinc homeostasis and parasite survival. Chelation of intracellular zinc using TPEN significantly reduced parasite viability by 72 hours, though partial viability was retained at 48 hours, suggesting a biphasic Zn stress response. Fluorescence imaging with FluoZin-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* upto 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.

P 014

Cyclins and CDKs of *Theileria annulata*: The Hidden Regulators of Parasite Biology

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Theileria, an apicomplexan parasite that infects bovine cells and causes Tropical Theileriosis in cattle, leads to huge economic losses of approximately US$800 million in India. *T. annulata-infected* cells exhibit several cancer-like characteristics. Targeting protein kinases in a cancerous cell is one of the most common methods in cancer therapy. Since *Theileria*-infected cells exhibit cancer-like phenotypes, it is hypothesized that targeting specific protein kinases could selectively eliminate *T. annulata*-infected cells. A previous study from our lab confirms the presence of 54 protein kinases in the *T. annulata* proteome. In this study, we selected the CMGC (CDKs) family protein kinases. CDKs exert their functions through interactions with partner cyclins, which play crucial roles in regulating cell cycle progression, division, and transcription, as extensively studied in mammals and other apicomplexan parasites. The phylogenetic studies show the presence of 8 TaCDKs and 4 TaCyclins in *T. annulata,* which were further validated by qRT-PCR. The yeast two-hybrid data confirm that one cyclin can interact with multiple CDKs. In-house antibodies were generated against all TaCDKs and TaCyclins, and their specificity was confirmed by western blot using lysates from infected and control cells. IFA revealed distinct localization patterns of TaCDKs and TaCyclins in schizont and merozoite stages. Colocalization studies further validated their interactions in schizonts and during synchronized S-phase and G2/M phases, indicating that a single cyclin can associate with multiple CDKs.

P 015

Molecular Insights into Ribosomal Protein Functions in *Plasmodium falciparum* Organellar Translation

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Malaria is a vector-borne parasitic disease caused by protozoan parasites of the genus *Plasmodium*. According to the World Malaria Report 2024, an estimated 269 million cases were reported globally in 2023, with approximately 593,000 deaths. The pursuit of new therapeutic targets is crucial in the fight against malaria, particularly as drug resistance continues to compromise the efficacy of existing treatments. *Plasmodium* harbours two indispensable endosymbiotic organelles: the mitochondrion and the apicoplast, both of which possess distinct prokaryotic-like translation machineries that have been successfully exploited for the development of antimalarials targeting organellar protein synthesis. Ribosomal protein L10 and L12 interact with each other and form the stalk of 50S ribosomal subunit, which allows the binding of initiation and elongation factors during protein translation. The expression levels of ribosomal protein L10 and L12 were found to be linked with CAP sensitivity. Most recently, a report demonstrated that CAP inhibits protein translocation step and interfere in between ribosomal RPL10 and RPL12 protein. Here, we identified one homolog of *Pf*RPL10 and two homologs of *Pf*RPL12 ($PfRPL12_{Apico}$ and $PfRPL12_{Mito}$). We successfully expressed these parasitic protein recombinantly to investigate their interaction. Additionally, we generated transgenic parasite lines overexpressing these proteins with the C-terminal eGFP tag through transient transfection and determined their subcellular localization.

P 016

Characterization and Evaluation of the Phytochemical Profiles and Biological Potential of *Adhatoda vasica* and its Green Synthesized Nanoparticles

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A. vasica, commonly referred to as Vasa or Vasaka, is a significant therapeutic herb in traditional ayurvedic practice. This study examines the therapeutic potential of leaves from *A. vasica* in treating Mosquito- borne diseases like Japanese Encephalitis (JE). The aqueous leaf extracts of *A. vasica* were employed as a reducing and capping agent for the nanoparticle synthesis. The synthesized nanoparticles were confirmed by using various characterization techniques such as Scanning Electron Microscopy (SEM), Dynamic Light Scattering (DLS), UV-Visible Absorbance Spectroscopy, Fourier Transform Infrared Spectroscopy (FT-IR). The analysis of plant- derived compounds in the leaf extracts using methanol, ethanol, chloroform and water showed the existence of alkaloids, terpenoids, phenols, tannins and glycosides. The leaf extracts of *A. vasica* and their nanoparticles demonstrated significant antibacterial, antioxidant and larvicidal efficacy. They also demonstrated efficient antibacterial efficacy against *S. epidermidis*, a bacterium known to cause secondary infections, in the case of Lymphatic Filariasis. The leaf extract and its AgN's act as a good radical Scavenging Agent. The synthesized silver nanoparticles exhibited a better larvicidal effect (LC_{50}=34.38ppm) when compared to the leaf extracts. Subsequent studies will be carried out to analyze the efficacy of *A. vasica* and its nanomaterial through evaluating the macrofilaricidal activity, particularly against Japanese Encephalitis (JE) and conducting *in vitro* analysis.

P 017

SPV-22 Protein of *Leishmania donovani* Identified as a Potential Biomarker for the Detection of VL, PKDL, and VL-HIV Co-Infection Patients

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Visceral Leishmaniasis (VL) is a life threatening protozoan disease caused by parasite *Leishmania donovani*. VL-coinfection has been identified as one of the emerging challenge for VL control. Diagnosis test applied to identify VL usually are not potential to detect VL-coinfected cases, since lower sensitivity and specificity is observed. Diagnosis of VL using rk39 strip test available but in case of VL-coinfection show decreased sensitivity and specificity, limits their use. Further, several modern tools and techniques are available for the diagnosis of PKDL. The sensitivity of gold standard parasitological test for PKDL in case of macular lesion also decreased. To overcome this diagnosis related serious issues associated with PKDL and VL co-infection, we are working to develop the novel biomarker with significant specificity and sensitivity for the detection PKDL and VL co-infected patients. In this study, we have identified SPV-22 protein from *Leishmania donovani* and further expressed and purified by affinity chromatography. We performed Immunoblotting and ELISA for detection of sensitivity and specificity using the sera samples disease groups (VL, PKDL, VL-HIV, VL-HIV-TB and VL-TB) and control (HIV, TB, Dengue & healthy control). We found antibodies titre in disease group was significantly higher as compared to control group. This result suggested that this protein could be a better target with diagnostic potential for the detection of VL, PKDL and VL Co-infected patients using sera samples.

P 018

Targeted Disruption of the *Plasmodium berghei* IMP4 Gene Reveals its Critical Role in Parasite Transmission in the Mosquito

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Ookinete to oocyst transition in mosquito midgut is one of the important bottlenecks in Plasmodium's life cycle. Several Plasmodium proteins like P25/P28, CTRP, LIMP, and pore forming PPLPs are reported to play an important role in ookinete motility or midgut invasion. In the course of gliding and midgut invasion, numerous host midgut genes actively promote or inhibit parasite development in mosquitoes. Genes such as LANB2 and CP contribute to parasite growth in the mosquito midgut, whereas LRIM1, Rel2, and WASP act antagonistically, inhibiting parasite development. The comprehensive understanding of ookinete traversal across the epithelial layer of the mosquito midgut and its interaction with mosquito midgut proteins remains elusive. Here, we have identified a parasite protein IMP4 (IMP1-like Protein) which is important for oocyst formation in the mosquito midgut. We disrupted the IMP4 gene by double cross-over homologous recombination and analyzed the mutant phenotype throughout the Plasmodium life cycle stages. IMP4 KO parasites showed normal asexual blood stage propagation in mice, suggesting its non-essential role during this stage. Further, analysis of parasite stages in mosquito revealed that mutant parasites form gametes, zygote, and ookinetes normally. However, ookinetes failed to form oocysts in the mosquito midgut, significantly reducing the oocyst numbers. The expression of several mosquito genes is altered during ookinete invasion. Furthermore, transcript analysis of mosquito midgut genes showed a marked downregulation of *AsCLP1*, *AsMyo1*, and *AsLANB2* in mosquitoes infected with *IMP4* KO parasites compared to wild-type infections. These findings highlight a critical role of IMP4 in facilitating oocyst formation and underscore its potential importance in malaria transmission biology.

P 019

Prevalence of Ectoparasite Infestation in Tangkhul Hui (Haofa), Ukhrul District, Manipur

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Abstract: The present study aimed to conduct a preliminary survey for the identification of ectoparasites, focusing on the prevalence of natural infestations in owned local indigenous hound dogs called Tangkhul Hui(Haofa), comprising 70 dogs, during April-September 2025 in different villages of Ukhrul District, Manipur, India. During this investigation, different species of ectoparasites, ticks, lice, and fleas were found, namely, *Rhipicephalus sanguineus*, *Rhipicephalus microplus*, *Haemaphysalis sp*, *Trichodectes canis*, *Ctenocephalides canis*, *Ctenocephalides felis* and *Xenopsylla sp*. Among the collected ectoparasites, the maximum number of *Ctenocephalus canis*(65%) and *Ctenocephalus felis*(86%) was observed, followed by *Xenopsylla sp* (46%) and *Trichodactus canis*(48%). Among the ticks, the most abundant one is *Rhipicephalus sanguinus*, having 38%, followed by *Haemaphysalis sp* (27%). *The Rhipicephalus microplus*, which is one of the least collected during this report, i.e. only 15%. Their attachment to the dog may affect the animal's health in several ways. They extract blood and reduce the comfort of their host. Out of these 70, 54 dogs were found infected with ectoparasites and were identified using standard taxonomic keys. The present study highlights the importance of taking care of ectoparasitic infestation to minimise the health-related conditions of dogs.

P 020

First Report on Anticoccidial Drug Resistance in *Eimeria* species of Broilers from Kashmir, a North-Western Himalayan Region

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This study presents the first report of anticoccidial drug resistance in *Eimeria* species infecting broiler chickens in Kashmir, North India, a region with a temperate climate favorable for oocyst survival. Field isolates of *Eimeria* spp. from poultry farms in the Kashmir Valley were evaluated following WAAVP guidelines to assess current anticoccidial resistance status. The oocyst inoculum comprised seven morphologically identified species, of which five (*E. acervulina, E. maxima, E. mitis, E. praecox,* and *E. tenella*) were confirmed molecularly using the ITS-1 rDNA marker. Forty-one-day-old Cobb broilers were randomly divided into four groups (n=10): Group I (infected + amprolium), Group II (infected + sulphaquinoxaline), Group III (infected untreated), and Group IV (uninfected control). Drug efficacy was determined by the percentage global index (%GI) based on weight gain, feed conversion ratio, lesion score, oocyst index, and mortality. The %GI values were 58.15 for sulphaquinoxaline and 56.86 for amprolium, indicating partial resistance, a moderate loss of efficacy without complete treatment failure. Morpho-molecular analysis identified five *Eimeria* species pre-treatment and detected four species post-treatment, suggesting emerging resistance. Phylogenetic analysis of ITS-1 sequences showed close genetic relatedness of local isolates with reference *Eimeria* strains in GenBank. Notably, this is the first report of ITS-1 sequences for *E. maxima* and *E. praecox* from Kashmir. In conclusion, the study provides evidence of anticoccidial resistance in broilers from Kashmir and highlights the need for regular monitoring, rotational drug use, and integrated control strategies.

P 021

The Role of *Leishmania donovani* Homoserine Kinase (LdHSK) in Managing Oxidative Stress and Enhancing Parasite Survival within the Host

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Visceral leishmaniasis (VL), a life-threatening disease of 71 countries, is caused by *Leishmania donovani*. Emergence of drug resistance highlights the urgent need for new therapeutic targets. Amastigotes survive within macrophage phagolysosomes by neutralizing harmful reactive oxygen and nitrogen species (ROS/RNS) using the thiol-based antioxidant system. Homoserine kinase (HSK), a key enzyme in this pathway, can be crucial for protecting the parasite from oxidative stress. Gene expression analysis showed that under oxidative stress (induced by H_2O_2 and menadione), expression of HSK and thiol metabolic pathway enzymes [Trypanothione synthetase (TryS), Trypanothione reductase (TR), cytosolic tryparedoxin (cTXN), and cytosolic tryparedoxin peroxidase (CTP)] were increased compared to the control. This suggests HSK plays a role in detoxifying ROS and helping the parasite withstand oxidative damage. To explore the role of HSK in oxidative stress and parasite survival, HSK was cloned, expressed, and purified using affinity chromatography. Polyclonal antibodies raised in rabbits were validated via immunoblotting against parasite lysate, and immunolocalization confirmed HSK's presence in the cytosol. Interestingly, Western blot analysis also indicated that HSK is secreted, pointing to its involvement in host-parasite interactions. Ongoing gene knockout and overexpression studies aim to further clarify HSK's function in thiol metabolism, stress response, and parasite survival within the host.

P 022

LetM1-Like Transporter in Mitochondria of the Malaria Parasite has Metal Ion Specificities Distinct from its Homologs

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Metal ion levels are tightly regulated in the malaria parasite by metal transporters which play an essential role in metal homeostasis. The P. falciparum genome encodes a single LetM1 protein annotated as a homolog of the yeast mitochondrial membrane protein Mdm38 (K^+/H^+ antiporter) and human LetM1 (Ca^{2+}/H^+ antiporter) with 26% and 22% sequence identity, respectively. However, PfLetM1 lacks conservation in the Ca^{2+} binding domain and has a long N-terminal extension, LETM-like domain and a transmembrane domain with three proline residues. We expressed and purified recombinant PfLetM1. BN-PAGE analysis of recombinant PfLetM1 detected multimers; higher order assembly of >250kDa, disrupted by β-ME treatment, was seen in parasite lysate. The selectivity of PfLetM1 for metal ions was tested by intrinsic spectrofluorimetry[1]. There was change in intrinsic PfLetM1 fluorescence with increasing concentrations of Fe^{2+} and Zn^{2+}, but not Ca^{2+}, Na^+, Mn^{2+}, Mg^{2+} and K^+; interaction was independent of H^+ (pH). PfLetM1 expression in E.coli provided greater tolerance to iron indicating its role in iron efflux. Immunofluorescence microscopy using antisera localized the protein to the parasite mitochondrion in blood stages. Our results suggest a role for PfLetM1 in Fe^{2+}/Zn^{2+} homeostasis in the P.falciparum mitochondrion, a role not previously identified for a LetM1 homolog.

P 023

Molecular Detection of *Wolbachia* and Its Vertical Transmission in *Phlebotomus argentipes* (Sandfly) in Bihar, India

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Wolbachia, an intracellular bacterium, belonging to α-proteobacteria (Rickettsia), have maternal inheritance and are commonly found in ovaries, intestines, salivary glands and thoraces of insects. Recently, certain *Wolbachia* phenotypes have been used to control a number of vector-borne diseases caused by *Plasmodium* and viruses, including Zika, dengue, and chikungunya. In *Phlebotomus argentipes*, of Bihar there is no any available reports on *Wolbachia* distribution. In order to understand the function of *Wolbachia* in sandfly populations, this knowledge gap must be addressed. Our findings showed the presence of *Wolbachia* in sandflies collected from two VL non- endemic districts, Patna and Nawada, which have a low endemicity for Visceral Leishmaniasis. Sandflies collected in Muzaffarpur and Vaishali (high prevalence of visceral Leishmaniasis) where negative for *Wolbachia* distribution. The consistent presence of approximately 611 base pair (bp) PCR fragments specific to the *wsp* gene in samples from both the F1 generation of sandflies reared in a laboratory and the natural population of sandflies collected from Patna and Nawada suggests the presence of *Wolbachia* and stable vertical transmission of *Wolbachia* in *Phlebotomus argentipes*. The finding of *Wolbachia* in *Phlebotomus argentipes* F1 generation indicates that *Wolbachia* may survive and be sustainably maintained in wild sand fly populations over a number of generations. For future prospective, *Wolbachia*-based vector control techniques, this persistence is essential**.** Nevertheless, this work offers important new information on the distribution and effects of *Wolbachia* in Bihar's sand fly populations. It creates new study opportunities and suggests possible vector management methods to lessen the incidence of Visceral Leishmaniasis.

P 024

Eosinophils and Macrophages Drive the Early Host Response Following Infection with the Third-Stage Larvae of *Brugia malayi* (Bm-L3)

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Lymphatic filariasis (LF), caused by filarial nematodes such as *Brugia malayi* and *Wuchereria bancrofti,* is characterized by lymphatic dysfunction, inflammation, and chronic immunopathology. Although eosinophils and macrophages are central to the innate immune response against helminths, their temporal coordination during early infection remains unclear. In this study, *B. malayi* third-stage larvae (L3) were administered intraperitoneally into BALB/c mice, and peritoneal exudate cells were analyzed at days 0, 3, and 21 post-infection using flow cytometry. Results showed that inflammatory eosinophils (iEos) expanded markedly from day 3 to day 21, while resident eosinophils (rEos) remained stable, suggesting a parasite-driven Th2-skewed environment favouring eosinophil effector activity. In contrast, macrophages exhibited a biphasic pattern, with an early increase at day 3 followed by a significant decline by day 21, indicating transient activation and possible immune suppression or exhaustion mediated by the parasite larvae. Taken together, our findings reveal a coordinated, yet contrasting behaviour of eosinophils and macrophages during early filarial infection, and emphasise the importance of time-dependent immune cell modulation in shaping the host–parasite interaction and provide a foundation for understanding how *B. malayi* manipulates host innate immunity to establish chronic infection.

P 025

Structural-Functional Characterization of Dynamins (*Pf* Dyn 1-3) in Human Malaria Parasite

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Plasmodium falciparum is an intracellular parasite that completes its multistage asexual life cycle (rings, trophozoites, and schizonts) in the human host and sexual stage in the female mosquito. The transition from uninuclear to multinuclear stage (rings to schizonts), requires faithful division of its organelles (mitochondrion, nucleus, ER, apicoplast, peroxisomes, and golgi) and proper partitioning of these functional organelles to each daughter cell. To ensure this successive proliferation, the parasite has evolved an efficient fission-fusion machinery which not only helps in organellar division but also may participates in the membrane-remodelling. *Plasmodium* encodes diverged Dynamin family, wherein, *Pf* Dyn1-2 are eukaryotic DRPs, and Dyn3 is bacterial dynamin-like protein. TEM study shows multimeric structure ring and filaments and limited proteolysis study shows differences in their dynamics. We have functionally characterized *Pf* Dynamins lipid binding and fusion properties. Cellular fractionation reveals differential partitioning of *Pf* Dyn1-3 into organellar fractions. Our ongoing work is focused on the expression and localization of parasite dynamins and identification of their adaptor proteins which play role in fission-fusion processes.

P 026

In-silico Modelling of the *Cryptosporidium* Proteasome to Map Binding Sites for Selective Inhibitors

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The proteasome, a multi-catalytic protein complex vital for cellular homeostasis and survival, is a validated drug target. Disrupting its function is often lethal to parasitic organisms, making it a promising therapeutic avenue. The well-studied *Plasmodium* proteasome is a validated drug target because its β5 subunit structurally distinct from humans, making it highly vulnerable to selective inhibitors. This strategy holds significant promise for *Cryptosporidium*, an obligate intracellular parasite causing severe diarrheal disease with no effective therapeutic options. The lack of structural information for the *Cryptosporidium* proteasome is a critical impediment to drug development. The study aims to overcome this by determining its structure, comparing its proteolytic sites to *Plasmodium* and human proteasomes, and mapping crucial binding sites for novel inhibitors using a comparative *in-silico* modelling approach. *Cryptosporidium* and *Plasmodium* proteasome sequences were obtained from CryptoDB, PlasmoDB, and UniProt, followed by sequence alignment with ClustalW. *Cryptosporidium* proteasome subunit structures were modelled using SWISS-MODEL, the *Plasmodium* proteasome structure from PUBsum. This study successfully predicted the 3D structure of the *Cryptosporidium* 20s proteasome core particle. Comparative analysis revealed several *Cryptosporidium*-specific inter-domain interactions relative to the *Plasmodium* proteasome, providing a crucial blueprint for developing *Cryptosporidium* selective drug candidates.

P 027

Mechanism of Sphingolipid Biogenesis in *Toxoplasma gondii*

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Toxoplasma gondii, an obligate intracellular parasite, depends on lipids for membrane synthesis and organelle biogenesis. Lipidomic analysis of acutely infectious tachyzoites revealed ethanolamine phosphoryl ceramide (EPC) and sphingomyelin (SM) as the key sphingolipids present during the lytic cycle. Tachyzoites can produce sphingolipids *de novo*, however the underlying enzymes and their physiological relevance are unknown. We identified three sphingolipid synthases (SLS1-3) encoded by the parasite genome. Conditional depletion of SLS1 compromises tachyzoite replication and thereby disrupts the lytic cycle. Proteomic and lipidomic analyses of the SLS1-depleted strain indicated significant decline in abundance of several proteins and dysregulation of major lipids. We are now analyzing membrane biogenesis and metabolic adaptation in the mutant.

P 028

Evaluating the Therapeutic Potential of Solamargine against Visceral Leishmaniasis in a Murine Model

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Visceral leishmaniasis (VL), caused by *Leishmania donovani*, remains a fatal neglected tropical disease with limited treatment options and rising drug resistance. In this study, solamargine, a natural steroidal glycoalkaloid identified through an *in silico* drug screening, was evaluated for its *in vivo* antileishmanial potential in BALB/c mice infected with *L. donovani*. Three doses of solamargine were administered via oral route, with the 80 µg/mL highest dose producing the most significant reduction in parasite burden as determined by qPCR analysis. Immunological studies revealed that solamargine treatment enhanced macrophage-mediated defense mechanisms, marked by increased production of nitric oxide and reactive oxygen species relative to the infected controls, contributing to effective parasite clearance. Immunophenotyping demonstrated a modulation of T-cell populations including elevated $CD4^+$ helper and $CD8^+$ cytotoxic T-cell populations, indicating a shift towards protective Th1-type immune response. Gene expression analysis further revealed upregulation of inducible nitric oxide synthase (iNOS) and nuclear factor kappa B (NF-κB), supporting the activation of pro-inflammatory pathways essential for parasite control. Collectively, these findings highlight Solamargine as a potent antileishmanial and immunomodulatory agent, acting via direct parasite suppression, enhanced oxidative responses and iNOS/NF-κB activation. Solamargine, therefore represents a promising candidate for safe and effective therapeutic against visceral leishmaniasis.

P 029

Functional Expansion and Diversification of HSP40s in Human Malaria Parasite

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The human malaria parasite has evolved an efficient protein folding machinery to keep its metastable and aggregation prone proteome in a functional state. The HSP40 family shows specific expansion across cellular compartments (cytosol, nucleus, apicoplast, mitochondria, and exported RBCs) with significant evolutionary divergence from human orthologs. Interestingly, plasmodial HSP40s show interspecies sequence variation, and their differential localization hint towards their HSP70-independent roles. We have explored the conformational and functional diversity of cytosolic, organellar and exported HSP40s. Biochemical studies suggested that cytosolic and apicoplast localized HSP40s suppress aggregation more efficiently than nuclear and exported HSP40s, while exported HSP40 effectively binds and protects unfolded substrates. Peptide-spot array showed that apicoplast-resident HSP40 had higher binding to hydrophobic and neutral peptides, whereas cytosolic HSP40 binds to hydrophilic peptides. BLI experiments gave additional information about differences in their binding kinetics (k_{on} and k_{off} rates). Stress experiments showed that these proteins vary in their expression and immunofluorescence signal under proteotoxic stress conditions. These findings indicate that the compartment-specific HSP40s exhibit distinct variation in their chaperoning activity. Understanding the functional diversity of *P. falciparum* HSP40s may give insights into their canonical and non-canonical roles in parasite survival.

P 030

A Study on Gastrointestinal Parasitism of Common Myna in Newtown, West Bengal, India

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The Common Myna (*Acridotherestristis*), a widely distributed urban-adapted passerine, is an omnivorous bird closely associated with anthropogenic habitats. To determine the prevalence and diversity of gastrointestinal parasites in this species, coprological examinations were conducted from March 2024 to March 2025 in New Town, West Bengal. A total of 303 fecal samples were collected from free-ranging mynas and preserved in 90% alcohol and 2.5% potassium dichromate solution. Samples were processed using centrifugal sedimentation and flotation techniques following Soulsby (1982) and examined microscopically (10× and 40×) for the detection and identification of parasitic forms. Out of 303 samples examined, 219 (72.3%) were found to be positive. Protozoa exhibited the highest relative abundance (55.3%), followed by nematodes (23.7%) and trematodes (21.0%). Seasonal analysis showed infection peaks highest during the monsoon (58.9%), followed by summer (27.0%) and winter (14.2%). Among the 219 infected Common Mynas, 16.5% of birds were co-infected with two or more species of GI parasites. The predominance of protozoan infections and their strong seasonal trend indicate a humidity-driven transmission pattern. The study concludes that monitoring the Common Myna can serve conservation purposes in urban environments by revealing parasite patterns that reflect the health of the ecosystem.

P 031

Functional Characterization of Exo1 in DNA Double Strand Break Repair and Mismatch Repair in *Plasmodium falciparum*

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The malaria-causing *Plasmodium* parasite relies on robust DNA repair mechanisms to counteract host-induced genotoxic stress and replication errors. Homologous Recombination (HR) facilitates double-strand break repair, while Mismatch Repair (MMR) corrects replication errors to maintain genome fidelity. Defects in MMR have been associated with elevated mutation rates and the emergence of drug-resistant strains. Despite its biological significance, the MMR pathway in *Plasmodium* remains poorly characterized. Nucleases play a critical role in MMR by excising mismatched DNA segments. In this study, we explored the functional relevance of the exonuclease EXO1 by generating a knockout line in *Plasmodium falciparum*. The EXO1-deficient parasites are defective in DNA-double strand break repair and displayed mild tolerance to the alkylating agent N-methyl-N′-nitro-N-nitrosoguanidine (MNNG), suggesting a partial impairment in MMR-associated DNA damage processing. Additionally, we investigated the interaction of EXO1 and FEN1 nucleases with known MMR proteins PfMSH2-1 and PfMSH2-2. These preliminary findings support the hypothesis that DNA repair nucleases are shared components of the HR and MMR pathways, potentially mediating functional crosstalk between them.

P 032

Deciphering the Role of Srs2 Helicase in *Plasmodium* DNA Repair and Genome Stability

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Plasmodium parasites frequently face double-strand DNA breaks (DSBs), which are predominantly repaired through homologous recombination (HR). Although HR maintains genomic stability, it can also drive genome rearrangements that diversify virulence genes, highlighting the need for strict regulation. In our search for factors that may restrain HR, we identified a helicase with partial sequence similarity to the yeast anti-recombinase ScSrs2, suggesting a potential role in controlling illegitimate recombination and preserving genomic integrity. PfSrs2 associates with critical DNA repair proteins in *Plasmodium falciparum*, and its expression is upregulated upon DNA damage, implicating it in double-strand break repair. Remarkably, the Δ*Pbsrs2* loss-of-function mutant exhibits reduced sensitivity to DNA-damaging agents, suggesting that PbSrs2 may act as an anti-recombinase. This is further supported by the observation that Δ*Pbsrs2* parasites grow faster than wild-type, likely reflecting more efficient repair of endogenous DSBs. In contrast to that, we discovered a defect of the Δ*Pbsrs2* mutant, displaying a complete abrogation of sporogonic development, resulting from the total absence of oocyst formation.

P 033

Proteomic Insights into Macrophage Modulation by *Taenia solium*: Linking Proteasome Dynamics to Immune Evasion Mechanisms

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Taenia solium is a parasitic cestode, and its larval form is known to cause neurocysticercosis (NCC) and associated epilepsy worldwide. The cysticercal larva can establish long-term infections in host tissues, often by modulating immune responses to promote its survival. We differentiated the human THP-1 cell line into macrophages and treated them with *T. solium* cyst antigen for 24 hours. Furthermore, global proteomics was done using nano-LC-MS/MS. Differentially expressed proteins (DEPs) were identified with statistical thresholds (adjusted p-value < 0.05, Log fold change ≥ 1.5). Pathway enrichment and protein–protein interaction analyses were performed using GO, KEGG, and STRING databases. Proteomic profiling revealed extensive remodeling of macrophage protein networks upon antigen exposure. Key changes included the upregulation of proteasome and immunoproteasome subunits (PSMA1, PSMA4, PSMB9, PSMD3, PSMD14), and cell cycle checkpoints (G2/ M checkpoints), along with the suppression of selected pro-inflammatory mediators (PRKAR1A, LYN). Enrichment analysis highlighted pathways in proteostasis, complement regulation, and metabolic adaptation. In conclusion, *T. solium* cyst antigens induce a coordinated shift in macrophage proteome towards an immunoregulatory phenotype, potentially contributing to parasite persistence and immune evasion. This is the first proteome-wide characterization of macrophage responses to *T. solium* cyst antigens, offering new insights into host–parasite interplay.

P 034

Understanding the Nuclear Function of Heat Shock Protein 90 and its Co-Chaperones in *Plasmodium* DNA Repair

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Heat shock protein 90 (Hsp90) is a highly conserved molecular chaperone essential for protein folding. Its non-canonical nuclear functions include DNA repair, and transcriptional regulation. Co-chaperones such as Aha1 and FKBP35 are essential for modulating the nuclear functions of Hsp90. In *Plasmodium falciparum*, PfHsp90 has been found to regulate the transcription of PfSir2 under heat-stress. In this study, we investigated the nuclear dynamics of PfHsp90 and its co-chaperones following genotoxic stress. Upon DNA damaging condition the expression of PfHsp90 is not upregulated, however two of its co-chaperones PfAha1 and PfFKBP35 are induced. We observed that PfHsp90 translocate to the nucleus upon MMS-induced DNA damage in a PfAha1 dependent way. Furthermore, deletion of the charged linker (ΔCL) region of PfHsp90 disrupts its interaction with PfAha1, highlighting the importance of this region for nuclear localization. These findings reveal a critical role for PfHsp90 in parasite survival under genotoxic stress.

P 035

Mechanistic and Metabolomic Insights into Apicoplast-Targeting Drugs and Apicoplast Metabolism in *Toxoplasma gondii*

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The apicoplast of *Toxoplasma gondii* and other Apicomplexan parasites harbors essential metabolic pathways and housekeeping functions of prokaryotic origin, making it a parasite-specific drug target. Drugs that inhibit apicoplast housekeeping functions typically cause the well-known phenomenon of delayed death, characterized by loss of the apicoplast in dividing parasites, underscoring the essentiality of its metabolic pathways. In this context, we screened the MMV Pathogen Box library against *T. gondii* to identify inhibitors that induce delayed death. Among the shortlisted compounds, we investigated MMV688345 and validated its activity against the DHFR domain of TgDHFR-TS. Canonically, antifolates targeting DHFR-TS cause immediate parasite growth arrest; however, intriguingly, MMV688345, a pyrimidine-derived antifolate, exhibits delayed death in *T. gondii*. We hypothesize that MMV688345 causes partial nucleotide depletion, with preferential allocation to the nucleus and mitochondrion, leaving the apicoplast nucleotide pool vulnerable and resulting in delayed inhibition. The mechanism underlying delayed death, the associated apicoplast segregation defect, and the essentiality of the organelle remain incompletely understood. Together, our studies integrate drug mechanism and metabolomic analyses to reveal apicoplast-linked vulnerabilities, offering new insights into parasite biology and potential avenues for rational antiparasitic drug development.

P 036

Myeloid-derived Suppressor Cells - A New Target for Modulating Host Immune Responses during Experimental Cerebral Malaria

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A dreadful manifestation of *Plasmodium berghei* ANKA infection leads to experimental cerebral malaria which is marked by vigorous immune activation and neuroinflammatory damages leading to fatal outcomes. Although a myriad of drugs have been used for protection against infection but resistance has been detected for nearly all of them and therefore it's time that we invest in alternative strategies. Effective host immune responses rely on a balanced cytokine and immune modulators responses and any disruption may lead to severe disease pathogenesis thus marking the importance of immune modulators as new promising therapeutic interventions. The immune system comprises of a dynamic system of cellular interactions and signalling cascades and within this sytem the Myeloid-derived suppressor cells (MDSCs) have gained promising attention due to their ability to modulate adadptive immune responses and resistance to immunotherapeutic strategies especially in cancer. Although studied in cancers, role of MDSCs in malarial pathophysiology remains elusive. Classically, MDSCs are known to exert anti-inflammatory and immuno-suppresive roles in pathological conditons but in contrary our current research deciphers how MDSCs expansion promotes exaggerated inflammation and their role modulating T-cell responses thus making them a prominent target for new therapeutic approaches to subvert disease severity.

P 037

Biochemical and Biophysical Characterization of *Leishmania donovani* Threonine Synthase

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Leishmania donovani, the protozoan parasite responsible for visceral leishmaniasis, causes severe systemic infection which may be fatal if left untreated. Threonine Synthase (TS) is a crucial enzyme that catalyzes the conversion of O-phospho-L-homoserine to threonine, an essential amino acid required for protein synthesis and as a metabolic precursor for lipid biosynthesis, both essential for parasite growth and survival. In this study, the TS gene from *L. donovani* was cloned into pET30a (+) expression vector and heterologously expressed in *Escherichia coli* BL21 (DE3). Protein expression was induced with 0.3 mM IPTG at 20 °C and recombinant TS containing an N-terminal His-tag was purified under native conditions using Ni–NTA affinity chromatography. SDS–PAGE analysis demonstrated a single band of approximately 73.5 kDa, confirming purity and expected molecular weight. Enzymatic activity was measured by quantifying inorganic phosphate released from O-phospho-L-homoserine using a colorimetric assay, and preliminary kinetic parameters were determined under optimized conditions. Circular dichroism spectroscopy at room temperature indicated that TS has a secondary structure composition of 35% α-helix, 26% β-sheet, 7% turn, and 32% random coil. Further kinetics and biophysical characterization of recombinant enzyme are underway. Parasite TS is absent in humans and other mammals and an attractive target for selective drug development.

P 038

Plasmodium DNA Ligase I is Essential for DNA Replication and Liver Stage Development

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DNA ligases are ubiquitous enzymes that catalyze the formation of phosphodiester bonds in double-stranded DNA, which is an essential step in DNA replication and repair. Malaria parasites' nuclear genome encodes a sole DNA ligase likely to function in nuclear and organelle DNA replication/repair. DNA ligase I (DL1) is a primary ligase that joins Okazaki fragments during DNA replication and nick seals during long-patch base excision repair (BER). In this study, we found that Plasmodium DNA ligase I expressed and localized to the nucleus and is essential for DNA replication. Several attempts to disrupt the DNA ligase I gene have been unsuccessful, indicating that it is essential for parasite viability. Next, we used the Flp/FRT-based conditional mutagenesis system to conditionally silence the gene function in P. berghei sporozoites for functional liver-stage studies. DL1-cKO sporozoites did not transition to blood-stage infection and appeared to be completely attenuated in the liver. We found that DL1-cKO sporozoites invade hepatocytes normally but do not significantly replicate 36 hpi. DL1-cKO EEFs undergo limited nuclear replication and do not increase in 36 hpi. This work provides critical evidence from rodent malaria studies supporting the importance of DL1 for DNA replication.

P 039

In-vitro Phytotherapeutic Modulation of Acetylcholinesterase Secretion in *Ascaridia galli*, an Inverse Host-Parasite Relationship

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Esterase enzyme Acetylcholinesterase (AChE), is a modulatory substance which terminates synaptic transmission by hydrolysing the neurotransmitter acetylcholine into acetate and choline. *Ascaridia galli* releases AChE for establishing the infection and its level indicates the level of stress. *Ascaridia galli* is the most notorious gastrointestinal nematode parasite of poultry fowls *Gallus* spp. This study evaluated the anthelmintic activity of *Azadirachta indica* (leaves), *Cucurbita moschata* (seeds), *Punica grantum* (peels) on *Ascaridia galli* to find a plausible scientific rationale behind their use and their effect on helminth. *Ascaridia galli* releases excretory/secretory substances containing AChE under stress because of the different phytochemicals or secondary metabolites present: alkaloids, flavonoids, terpenoids, tannins etc. Alkaloids and terpenoids occur in abundance in all studied plants. *Azadirachta indica* leaf extract showed highest acetylcholinesterase inhibitory activity compared to others. In in-vitro anthelmintic assays, the pumpkin seeds showed comparatively better effects against *A. galli*. The paralysis and mortality time of test parasites were recorded for each experiment. Histological sections of treated worm showed degradation of compact structure of *Ascaridia galli*. Body wall shrinkage and separated cuticle from epidermal layer was observed. Overall result shows that traditional plant extracts are more effective than the synthetic drug in the present in-vitro assessment.

P 040

Repurposing of Propafenone, an FDA Approved Anti-Arrhythmic Drug for Antileishmanial Therapy

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Leishmaniasis being the disease of old and new worlds, still continues to be categorized as a Neglected Tropical diseases. With the advent of numerous drug discoveries and a surplus of active pharmaceutical ingredient molecules available at our disposal, pursuing the discovery of new molecules is tedious and requires significant resources. To overcome these challenges, drug repurposing can be an effective approach, repurposing molecules initially designed for one condition to treat other diseases. In this study, we evaluated the antileishmanial activity of propafenone hydrochloride which is generally used as an anti-arrhythmic drug. *Leishmania donovani*, the causative agent of visceral leishmaniasis was used as the test organism. Propafenone treatment was found to reduce the viability of both promastigote and amastigote forms of *Leishmania donovani*. It has been shown to affect the morphology and found to be detrimental to the body and flagellum of the parasite. Propafenone causes depolarization of the mitochondrial membrane thus affecting its functioning and ATP generation as evident by low ATP levels compared to the control. It also induced cell cycle arrest at the G_2/M phase and oxidative stress in the parasites, highlighting its potential as an antileishmanial agent.

P 041

Comparative Studies on Host Blood Metabolites Changes in Severe and Non-Severe Malaria Using Rodent Model

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Malaria continues to be a significant global health burden, with approximately 241 million cases and an estimated 630,000 deaths reported annually. The disease exhibits a broad spectrum of clinical outcomes, from asymptomatic infections to severe and potentially fatal consequences. In vitro and ex vivo metabolomic approaches are informative but limited in their ability to reflect the full complexity of host metabolic responses shaped by genetic, immunological, physiological, and environmental variation. In this study, we employ mass spectrometry-based temporal profiling of serum metabolites in mice infected with Plasmodium berghei (ANKA)-lethal and Plasmodium yoelii (17XNL)-non lethal to investigate the differential metabolic responses elicited by these parasites within the host. Our previous studies identified significant reductions in glycolytic intermediates, purine nucleotide precursors, tryptophan, and its bioactive derivatives during late-stage malaria. Additionally, several metabolites exhibiting greater fold-changes remain uncharacterized. By utilizing an advanced metabolomics kit-based protocol, we aim to expand and refine our metabolite profiling, enabling the quantification of a broader range of metabolites with improved accuracy. With the successful completion of this project we hope to identify the metabolites which shows increasing or decreasing trends in correlation to disease progression, these metabolites can be considered as biomarkers for onset of severe malaria. This proof of concept will also lay the foundation for future studies on blood metabolite changes in human malaria patients.

P 042

Integrative Taxonomic Description of a Novel Septate Gregarine, *Stylocephalus epistupaformis* N. sp., Parasitising the Darkling Beetle *Gonocephalum depressum* Fabricius, 1801, in West Bengal, India

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The genus *Stylocephalus* Ellis, 1912 (Apicomplexa: Eugregarinorida) exhibits wide distribution and taxonomic diversity, yet only ~0.31% of beetle hosts have been studied, with most species defined by light microscopy alone. In the present study from West Bengal, India, 32% of the investigated *Gonocephalum depressum* were found infected with septate gregarine. Trophozoites (446.98–596.15 µm) possess an epimerite complex (59.89–109.73 µm) comprising a stupa-shaped epimerite proper (4.82–9.72 µm) seated on an elevated base and a cylindrical diamerite with basal tumidus. Epicytic folds (3–4/µm) extend along the body except the epimerite. Gamonts (562.50–856.21 µm) show cratered protomerites and narrowly obdeltoid deutomerites with elliptoid nuclei containing numerous karyosomes; fold density is 3–4/µm in the protomerite and 4–4.5/µm in the deutomerite. Associations are frontal. Gametocysts (178.51–219.73 µm) are papillated, rupturing to release obovoid oocysts (9.78–12.61 × 8.10–10.40 µm) extruded in coiled chains, each with two orbicular micropyles. Phylogenetic analysis of SSU rDNA places the species within Stylocephalidae, forming a strongly supported clade (100%) with a branch length of 0.041 substitutions per site. Morphological and molecular evidence supports recognition of *Stylocephalus epistupaformis* n. sp., a novel and taxonomically well-defined member of the genus *Stylocephalus*.

P 043

Genomic Surveillance Unveils Regional Drug Resistance Dynamics in Indian *Plasmodium falciparum* Populations

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India has achieved a remarkable reduction in *Plasmodium falciparum* malaria cases, yet the rapid spread of antimalarial drug resistance threatens the 2030 elimination goal. To address this challenge, we applied PfMDR15, a newly developed multiplexed genomic sequencing panel targeting 15 drug-resistance genes, to 238 clinical isolates collected across six Indian states. The panel, designed for field use and compatible with low-parasitaemia dried blood spots, enabled high-resolution genotyping and revealed striking regional diversity in resistance patterns. In the Northeast (Tripura and Assam), chloroquine resistance remained entrenched, dominated by Pfcrt K76T and the CVIET haplotype, together with Pfaat1 S258L, and extensive Pfdhfr–Pfdhps quintuple and sextuple haplotypes indicating complete sulfadoxine–pyrimethamine resistance. Central India displayed variable chloroquine resistance and early signatures of lumefantrine tolerance (Pfmdr1 Y184F, Pfaat1 S258L). Parasites from Delhi exhibited close genomic similarity to those from the Northeast, reflecting regional connectivity with Southeast Asia. In Chhattisgarh and Telangana, Pfcrt wild-type alleles predominated, though Pfmdr1 Y184F was frequent. Crucially, no WHO-validated Pfk13 mutations associated with artemisinin resistance were detected, supporting sustained efficacy of ACTs in India. Notably, this study reports the first detection of Pfaat1 S258L in India—previously known only from African isolates—suggesting convergent evolution of drug resistance. These findings underscore the urgent need for integrated genomic surveillance to complement therapeutic efficacy studies. PfMDR15 offers a rapid, scalable platform for real-time monitoring of resistance trends, guiding drug policy and strengthening India's capacity to safeguard ACT efficacy and achieve malaria elimination by 2030.

P 044

Identification of Novel Inhibitors Targeting *Leishmania donovani* Peroxins

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Visceral leishmaniasis (VL) is caused chiefly by *Leishmania donovani,* a kinetoplastid that relies on glycosomes, peroxisome−like organelles to compartmentalise key metabolic enzymes[1]. Glycosomal biogenesis are regulated by peroxins (PEX) which are functionally conserved, but their sequence being poorly conserved across lineages make them attractive target for therapeutic approaches[2]. In our study, we present a characterisation of specific *L. donovani* peroxins that are involved in cargo protein translocation. To explore the functional interactions, protein-protein docking simulations were performed leading to the identification of potential binding pockets in the protein-protein interface followed by target based high throughput screening of PEX inhibitors. Candidates were identified by *in-silico* screening of compounds using inhouse repository. Genes encoding the target peroxins were cloned, expressed and purified. These purified proteins were assayed by Surface Plasmon Resonance for quantification of its binding affinities and kinetics. Biochemical assay for cytotoxicity and cell viability tests were performed for the compounds. Finally, the compounds identified were subjected to *in-vivo* assay to access its efficacy in disrupting the protein-protein interactions offering a potential novel therapeutics against the parasite.

P 045

Investigation of the Comorbidities Associated With Bancroftian Filariasis in and Around Asansol, West Bengal, India

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Bancroftian filariasis, vector-borne parasitic disease caused by the nematode *Wuchereria bancrofti* remains a major public healthcare challenge in the endemic regions of West Bengal, India. Nationwide Mass Drug Administration (MDA) has shown limited success due to insufficient coverage, poor compliance, and low awareness, contributing to continued parasite transmission. Furthermore, various comorbidities associated with Bancroftian filariasis exacerbate disease severity and hinder therapeutic outcomes. The recurrent incidence of secondary bacterial and fungal co-infections poses significant clinical challenges, particularly in immunocompromised hosts. Here the opportunistic fungi *Pichia guilliermondii* must be held accountable. In addition to the classical clinical manifestations of lymphatic filariasis (LF), such as lymphedema, lymphangitis, hydrocele, and elephantiasis, affected individuals have also been reported to exhibit a spectrum of metabolic and systemic comorbidities, including diabetes mellitus, hypertension, obesity, and cardiovascular disorders. Asansol, a coal-mining region in West Bengal identified as an LF-endemic zone, has not undergone systematic evaluation of comorbidities related to primary disease manifestations. This study aims to address this gap by conducting a comprehensive assessment of comorbid conditions associated with Bancroftian filariasis in and around Asansol. The findings are expected to provide critical insights for developing more effective disease management and control strategies.

P 046

UHPLC-QTOF-MS/MS Phytochemical Screening, Antimalarial and Anti-Inflammatory Potential of Medicinal Plant *Erycibe paniculata* (Roxb.) via Regulation of Nitric Oxide Synthase and NF-κB Signalling Pathway: a Network Pharmacology and *in-vitro* Analysis

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Erycibe paniculata (Roxb.) has been used as ethnomedicine for treating fever and inflammatory diseases[1]. However, its antimalarial properties remain scientifically underexplored. This study aimed to identify its bioactive Phytoconstituents and evaluate anti-malarial, anti-inflammatory and cyto-protective effects. Phytochemical profiling was performed using UHPLC-QTOF-MS/MS analysis[2] and analysed through Network pharmacology. Different extract of *E. paniculata* was screened for anti-malarial activity using the SYBR Green I-based fluorescence assay[3] and Pf-LDH assay[4] against *Plasmodium falciparum 3D7*. Cyto-protective effect[5] was examined in HEK-293 and RAW 264.7 cells. The anti-inflammatory potential was evaluated by assessing nitric oxide (NO) inhibition, iNOS expression, and effect on pro and anti-inflammatory cytokines involves in NF-κB signalling pathway in RAW 264.7 macrophage cells by ELISA and qRT-PCR analysis. The UHPLC-QTOF-MS/MS analysis revealed that *E. paniculata* extract dominated by phytochemicals like coumarins, flavonoids, alkaloids and Quinic acid derivatives. *E. paniculata* exhibited potent anti-malarial activity with an IC_{50} value of 26.29±1.42 in SYBR Green I and 15.48±1.19 µg/ml in Pf-LDH assay. The *E. paniculata* extract significantly inhibited NO production, iNOS expression and modulate the inflammatory cytokines i.e. TNF-α, IL-6, IL-10 in RAW 264.7 cells without showing cytotoxicity in HEK-293 and RAW 264.7 cells (CC_{50} > 300 µg/ml) with better selectivity index indicating its safety effects.

P 047

Traditional Medicinal Plants as Anthelmintics: Documenting Local Knowledge in Sikkim Himalaya

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Anthelmintic drug resistance is rising which is a challenge to the ongoing preventive methods Smith & (Zarlenga, 2006; Nirala et al., 2019). Plants have a history of use in treating parasitic infections. The present study documents the traditional knowledge of anthelmintic medicinal plants used by local and indigenous communities of Sikkim Himalaya. Ethnobotanical surveys were conducted across different locations of Sikkim, Kalimpong and Darjeeling Himalaya, employing semi-structured interviews with traditional healers, farmers and village elders following snowball sampling method. A total of 300 respondents were interviewed and 93 plant species belonging to 36 families were recorded to possess anthelmintic properties. Relative Frequency of Citation (RFC) was applied to assess the cultural importance and reliability of reported species. Commonly cited taxa included members of the families Poaceae, Rutaceae and Asteraceae. Several species, *notably Imperata cylindrica, Cannabis sativa, Thysanolaena latifolia, Coix lacryma-jobi* demonstrated high RFC values, indicating local consensus regarding their efficacy against parasitic infections. The study highlights the significance of indigenous knowledge in identifying bioresource potential for novel anthelmintic agents emphasizing the need for further validation and documentation of medicinal flora in the Himalayan region.

P 048

Cysteine Protease Falcipain 3 is a Potential Enzyme for Proteolytic Processing of Histone Acetyltransferase PfGCN5 in *Plasmodium falciparum*

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Despite more than 150 years of research, malaria continues to be a major global cause of illness and death. The *Plasmodium falciparum* PfGCN5 histone acetyltransferase (HAT) plays a crucial role in regulating gene expression via epigenetic mechanisms. It is an essential enzyme that catalyzes the acetylation of histone proteins. PfGCN5 is a chromatin-remodeling enzyme of approximately 170 kDa, containing conserved bromodomain and acetyltransferase domains located at its C-terminal region. PfGCN5 undergoes proteolytic processing, however, the specific protease responsible for this process remains unidentified. Immunoprecipitation followed by LC-MS/MS analysis to identify PfGCN5 interacting proteins uncovered the presence of food vacuole proteins, such as the cysteine protease Falcipain 3 (FP3), in addition to the canonical members of the PfGCN5 complex. Subsequently, cysteine protease inhibitor E64d resulted in the inhibition of PfGCN5 processing along with concomitant enrichment and colocalization of PfGCN5 and FP3 around the food vacuole as shown by confocal microscopy and Electron Microscopy. The nuclear protein PfGCN5 undergoing proteolytic cleavage by the food vacuolar protease FP3 is an unusual and atypical phenomenon in eukaryotic organisms. Targeting FP3, involved in Proteolyic processing of PfGCN5 may offer a novel strategy for drug development.

P 049

Diversity of Plant-Parasitic Nematodes Associated with Banana Crops in West Bengal, and First Record of *Sclerolabia camerunensia* (*Dorylaimida: Thornenematidae*) from India

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Banana (*Musa paradisiaca* L.) is a nutritionally important and economically valuable crop, widely cultivated in tropical and subtropical regions. India is the world's largest banana producer, contributing around 20% of global output, with West Bengal as one of the key producing states. However, banana production is significantly affected by plant-parasitic nematodes (PPNs), which cause notable yield losses. Despite their impact, updated and region-specific data on PPN diversity in West Bengal remain limited. This study reports the first record of the nematode species *Sclerolabia camerunensia* from India, isolated from the rhizospheric soil of banana during a faunistic survey in West Bengal. Previously known only from Cameroon (Carbonell & Coomans, 1985), the Indian population was morphologically characterized and critically compared with the type material to confirm its identity. In addition to this novel record, the study documented a diverse assemblage of 89 plant-parasitic nematode species, belonging to 48 genera, associated with banana cultivation in the region. This comprehensive data enhances current knowledge of nematode biodiversity in banana agroecosystems and underscores the need for continued surveillance and the implementation of location-specific nematode management strategies to sustain banana productivity in West Bengal.

P 050

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

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Lymphatic filariasis (LF), a debilitating vector-borne disease-causing chronic disability, lacks adulticidal drugs or vaccines, highlighting the need for novel intervensions[1]. As early infection is asymptomatic and chronic stages are incurable, transmission-blocking vaccines are ideal. VSPs modulate host-immunity and recently emerged as vaccine candidates and biomarkers. Therefore, we evaluated vaccine potential of VSPs of *Aedes aegypti* in LF. VSPs were isolated and their diversity and structural integrity were confirmed by proteomic and molecular analyses. Co-infection with L3 larvae and VSPs, mimicking natural transmission, did not confer protection. However, VSP pre-immunization in Balb/c mice reduced L3 burden by ~69%, whereas in *Mastomys* ~76% and ~57% protection was observed against microfilariae and adult worms, respectively. Encouraged by these results, we investigated VSP-mediated host immunomodulation and underlying mechanisms. Poor cross-reactivity of infected animal sera with VSPs and minimal ADCC suggested a limited role for humoral immunity. *Ex-vivo* analyses of splenocytes and peritoneal macrophages revealed distinct immunological signatures: VSPs promoted cellular proliferation, macrophage-specific phagocytosis, and enhanced ROS/RNS production. A mixed Th1/Th2 inflammatory cytokine response was observed. Hence, cellular immunity likely underlies the observed protection. Collectively, these findings suggest that VSP-mediated cellular immunity underlies protection, highlighting their strong potential as novel vaccine candidates against LF.

P 051

A *Plasmodium* Lipase is Critical for the Disruption of the Parasitophorous Vacuole Membrane and Egress of Hepatic Merozoites

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Malaria parasites develop within a specialized vacuole delimited by the parasitophorous vacuolar membrane (PVM). The exit of *Plasmodium* from host cells is a well-regulated process involving several molecules that disrupt the PVM to continue the life cycle. While several protein classes play a role in *Plasmodium* PVM rupture and merozoite egress, the first class of enzymes one would consider to act on membranes is lipases. However, the role of a lipase in PVM disruption remains unknown. Here, we characterized the role of UIS28 (upregulated in infective sporozoite gene 28) in *Plasmodium berghei.* Bioinformatic studies indicated that UIS28 is a class 3 lipase containing a fungal lipase-like domain. We found that UIS28 localizes to the PVM in infected hepatocytes. To understand the role of UIS28 in PVM disruption, we validated its lipase activity and found that it breaks down lipids into glycerol. Parasites lacking UIS28 develop normally in the blood and mosquito stages and mature fully into hepatic merozoites but show a defect in egress from host hepatocytes. We quantified the PVM rupture and reported that mutant parasites exhibit delayed egress due to impaired PVM disruption. Together, our results demonstrate that UIS28 is a lipase involved in the disruption of the PVM and the successful egress of hepatic merozoites.

P 052

Cytogenetic Approach of *Ascaridia galli* from Manipur, India

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Ascaridia galli is a species of helminths, known for its low chromosomal count, suitable for studying the meiotic as well as mitotic chromosomes. Despites of its advantages on meiotic chromosomes study, exploration of this species from Manipur is scanty. For this work the male adult parasites from chicken were collected from three different districts of Manipur, between January 2023 -September, 2025. Whole length of the testes was dissected out and washed with hypotonic solution for one hour. The cytogenetic approach was done with the help of Porter and Martin (1977) with slight modifications. The meiotic chromosomes of the *Ascaridia galli* consisted of five or four bivalents in most of the cells. The whole meiotic stages starting from Leptotene to Telophase II were observed from single slide. The study highlights on cytogenetics of this species. In future, comparative studies from different localities covering large study areas will give well and formidable aspects of this species.

P 053

Divergent Mechanisms of Cell Division and Morphogenesis in *Plasmodium*

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The malaria parasite *Plasmodium* exhibits a complex life cycle characterized by rapid proliferation, extensive morphological transformations, and adaptation to diverse host environments. During the blood stage, the parasite undergoes asexual replication within red blood cells, producing merozoites whose cyclic release and reinvasion cause disease symptoms. A subset of parasites differentiates into sexual forms, the gametocytes, which are essential for transmission to mosquitoes. To navigate these developmental transitions, Plasmodium relies on dynamic microtubule (MT) networks that govern cell division, polarity, shape and motility. However, the molecular mechanisms regulating MT organization remain poorly understood. Motor proteins such as kinesins and dynein are key modulators of MT function. Notably, Plasmodium spp. possesses a streamlined kinesin repertoire of only nine members and limited sets of dynein, reflecting evolutionary adaptation. The parasite's divergent MT-based processes and unique regulatory proteins present promising avenues for therapeutic intervention.

P 054

Understanding the Role of Lipid Metabolism in Mediating Artemisinin Tolerance in *Plasmodium falciparum*

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Plasmodium falciparum is the deadliest malaria species that infects humans. It has developed advanced ways to escape both antimalarial treatments and the immune defenses of its host. Even with the widespread use of antimalarial drugs, the rise and global spread of resistant strains present major challenges to malaria eradication. Artemisinin is the last line of defense, and the development of resistance to this drug is a significant concern. Kelch13 mutations, including C580Y, R539T, I543T, and Y493H, are often linked to artemisinin resistance, but they do not work alone. Recent studies show that broader metabolic changes, particularly in lipid metabolism, help parasites survive the stress caused by drugs through dormancy and metabolic adjustments. In this study, we used Kelch13 mutant and revertant lines to define a "resistant lipidome" using transcriptomic and LC/MS-based lipidomic analyses. Our findings show clear changes in lipid class distribution and flow in resistant parasites. Additionally, population genomics analysis of about 2,500 field isolates from Africa and Southeast Asia pointed to a possible α/β hydrolase with multiple mutations alongside Kelch13 changes. Biochemical tests suggest that it acts as a serine hydrolase with possible lipase activity, linking it to lipid remodeling processes that are key to resistance traits. Overall, this research demonstrates that lipidomic changes are a defining feature of artemisinin-resistant *P. falciparum*. This is driven by both Kelch13-related and genome-wide mutations. It highlights the value of metabolic profiling for monitoring resistance and opens new paths for targeting metabolic enzymes as future antimalarial strategies.

P 055

Role of Trypanothione Synthetase Protein in Amphotericin B and Miltefosine Resistance against *Leishmania donovani*

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Leishmania donovani is a protozoan parasite responsible for Visceral Leishmaniasis, a severe and often fatal disease flipping between human host and sand fly vector. Each year, around 20 - 30 thousand new cases are reported across approximately 78 endemic countries. Current treatments, such as Amphotericin B (AmpB) and Miltefosine (Mil), are increasingly compromised due to rising drug resistance, along with challenges including high cost, teratogenicity, and complex administration route. This alarming situation calls for the urgent need for new therapeutic targets that can bypass resistance mechanisms and streamline drug development. *Leishmania donovani* Trypanothione synthetase (LdTryS), a key enzyme in the thiol metabolic pathway of trypanosomatids, plays a central role in maintaining the parasite's redox balance and has been identified as a validated drug target through several prior studies. This study investigates the involvement of LdTryS in drug resistance by analyzing its expression in drug-sensitive and -resistant *L. donovani* promastigotes strains including; wild-type (LdWT), AmpB-resistant clinical isolate (LdAmpBRCI), AmpB-resistant lab-generated (LdAmpBRLG) and Mil-resistant lab-generated (LdMilRLG). Additionally, LdTryS expression was evaluated under oxidative (hydrogen peroxide, menadione) and heavy metal (arsenic, cadmium) stress conditions. The results demonstrated significant up-regulation of LdTryS protein expression as well as enzymatic activity in resistant strains under induced stress conditions, suggesting its active role in stress response and redox regulation. These findings underscore the contribution of LdTryS to thiol-based drug resistance and stress tolerance in *L. donovani*, and thus its potential as a promising therapeutic target for the drug development against Visceral Leishmaniasis can be explored in future.

P 056

Prevalence and Identification of Gastrointestinal Helminths in Domestic Ducks of Imphal East and West District, Manipur

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Helminthic infections are associated with poor absorption of nutrients, retarded growth, weight loss, and reduced egg and meat production leading to great economic loss. This study was carried out to determine the prevalence of Gastrointestinal helminths in two group of domestic Fowls in Imphal East District, Manipur. A total of 153 Gastrointestinal tracts of domestic ducks, 70 from Imphal West and 83 from Imphal East district were collected randomly from voluntary local meat centers and examination for the presence of Helminths is done in Parasitology Laboratory, D.M. College of Science, Dhanamanjuri University. The study shows overall prevalence of 37.90% with similar rate in both the districts. 3 types of helminths ,1 cestode and 2 nematodes were recovered and identified using identification key by Soulsby which includes *Raillietina spp.* (39.28), *Ascaridia galli* (57.14%) and *Heterakis gallinarum* (3.57%). Mixed infection with more than 1 type were found to be 44.82%. The study revealed that there is prevalence of GI helminths in Domestic ducks and may further help in establishing the preventive measures, control and management of poultry whenever there is outbreak of infection, poor growth or production.

P 057

Plasmodium Sporozoite Invasion Protein (SIP) Plays a Critical Role during Sporozoite Infection of the Mammalian Liver

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The *Plasmodium* sporozoites invade hepatocytes and develop into exo-erythrocytic forms (EEFs) by forming a parasitophorous vacuolar membrane. Parasite divides its nucleus and forms thousands of hepatic merozoites, which are released as merosomes and infect erythrocytes. Several proteins are implicated during this process; however, the role of several hypothetical proteins remains unknown. Here, we disrupted the sporozoite invasion protein (SIP) gene in *Plasmodium berghei*. We found that disruption of SIP did not affect parasite development in asexual blood stages. Next, we checked the transmission in mosquitoes and assessed ookinetes, oocysts, and sporozoite formation and found these stages were comparable to wild-type parasites. Inoculation of salivary gland sporozoites in mice delayed the pre-patent period compared to WT parasites. Invasion assay indicated that SIP is critical for sporozoite invasion of hepatocytes. These findings indicate that SIP is crucial for sporozoite invasion of hepatocytes.

P 058

ILC2–Eosinophil Crosstalk Shapes the Pathogenesis of Tropical Pulmonary Eosinophilia

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Tropical Pulmonary Eosinophilia (TPE) is a hyperresponsive immune syndrome in response to trapped microfilariae within the lungs of some patients infected with *Wuchereria bancrofti* and *Brugia malayi*. The cross-talk between eosinophils and Innate Lymphoid Cells Type 2 (ILC2) that shapes the immunopathology of TPE has not been thoroughly explored. To investigate this, we developed a mouse model of TPE wherein BALB/c mice were first sensitized with frozen *B. malayi* Mf, followed by intravenous injection of live Mf. ILC2 recruitment was monitored in lung tissue and other anatomical compartments at Days 4, 6, 8, and 10 post-challenge by multicolour flow cytometry. Functional assays, including adoptive transfer of ILC2, Cell migration assays, and air pouch experiments, were performed to assess their role in eosinophil recruitment. Our data demonstrated a rapid and significant expansion of ILC2 in the lungs and other anatomical compartments following the administration of live B. malayi Mf, peaking on Day 6 and declining thereafter. Findings from the murine air pouch assay and cell migration experiments confirmed that ILC2 promoted the recruitment of inflammatory (iEos) and resident eosinophils (resEos), underscoring their potent eosinophil-chemotactic capacity. In conclusion, the present study highlights the hitherto unexplored role of ILC2 cells in facilitating eosinophil recruitment during TPE thus opening up avenues to target the ILC2-eosinophil axis for better management of TPE.

P 059

3-O-p-Coumaroylquinic Acid Exhibits Potent Antimalarial Activity against *Plasmodium falciparum* Through Mitochondrial Disruption and Food Vacuole Dysfunction

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Plasmodium falciparum remains the most virulent species responsible for human malaria, posing a persistent global health challenge. While several natural compounds have been identified with antimalarial potential, phenolic compounds represent a relatively underexplored but promising class of bioactives. In this study, we investigated the antimalarial efficacy and cellular mechanisms of 3-O-p-coumaroylquinic acid (opCQA), a phenolic compound possessing two bioactive functional sites. Our *in vitro* assays demonstrated that opCQA effectively inhibited *P. falciparum* growth during the erythrocytic stages, indicating strong intrinsic antimalarial activity. Notably, synergistic evaluation with artemisinin revealed a combination index (CI) of 0.3636 at a 1:1 ratio, suggesting a highly synergistic interaction. Fluorescence-based assays indicated that opCQA caused a marked disruption of the mitochondrial membrane potential (ΔΨM), analogous to the effect of artemisinin, alongside alterations in the parasite's food vacuole morphology resembling chloroquine-induced vacuolar swelling. These findings imply dual targeting of parasite mitochondria and food vacuole functions. Further, opCQA-treated parasites exhibited nuclear condensation, DNA fragmentation, and apoptotic-like features, consistent with programmed cell death. Gene expression analyses revealed downregulation of food vacuole–associated genes (*PfFP2, PfPM2, PfPM4, Pffvrt1*) similar to chloroquine exposure and suppression of mitochondrial cytochrome gene *Pfcytc1,* as observed with artemisinin. Interestingly, *PfnPrx,* an oxidative stress response gene, was upregulated, suggesting compensatory redox mechanisms. In cytotoxicity assays, opCQA exhibited moderate antiproliferative effects against A549 and U87 cancer cell lines (CC_{50} = 153.5 ± 1.3 µM and 212.87 ± 0.8 µM, respectively), while maintaining low toxicity toward Vero cells (CC_{50} = 693.4 ± 0.3 µM). Collectively, our findings identify 3-O-p-coumaroylquinic acid as a promising dual-target antimalarial candidate capable of disrupting mitochondrial and food vacuole functions in *P. falciparum.* The compound's synergistic potential with artemisinin underscores its translational value for next-generation combination therapies against drug-resistant malaria.

P 060

To explore the Diagnostic Potential of SPV55 as Diagnostic Biomarker for the Detection of VL, PKDL and VL Co-Infection Patients from Sera Samples

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Human visceral leishmaniasis (VL) is a life-threatening protozoan disease caused by parasite belonging to *Leishmania donovani*. HIV-VL has been identified as one of the emerging challenges for visceral leishmaniasis control as current diagnosis test not much specific and sensitive for VL-HIV co-infection. The current biomarker rK39 used to detect VL and their co-infection have false positive result in malaria, TB and pregnant cases and also show false negative in case of VL co-infection due to poor immunity that is the major disadvantage of rK39 test. Both HIV and VL are endemic in Bihar, India. Bihar is the most endemic state for VL-HIV in India, an estimated 2-7% of VL cases are co-infected with HIV. Parasite identified by skin slit smear of PKDL Patients is considered, as gold standard but has very low sensitivity of only 58%. To overcome this diagnosis related issues associated with PKDL and VL co-infection we are working to develop the novel biomarker with significant specificity and sensitivity for the detection PKDL and VL co-infected patients. In this present study a SPV55 protein was expressed and purified. The purified protein was used for Immunoblotting and ELISA was done of this protein using VL, PKDL, VL-HIV, VL-HIV-TB &VL-TB patient's serum with HIV, TB, Dengue as positive control showed strong binding or high antibodies titer with patients' sera samples. This result suggested that this protein could be a better target with diagnostic potential to for the detection of VL, PKDL and VL Co-infected patients.

P 061

Understanding the Role of Host-Parasite Interaction in Malaria Pathogenesis

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Plasmodium falciparum, the malaria parasite, has developed a variety of strategies to survive and proliferate inside the host, leading to numerous complications. During the intraerythrocytic stage, *P. falciparum* expresses variant surface antigens (VSA) at the infected erythrocyte (IE) membrane to interact with the endothelial cell receptors. This phenomenon is known as cytoadherence. One of these VSAs, PfEMP1, expresses a single protein on the membrane but possesses a repertoire of around 60 clonally variant *var* genes. However, more than one *var* transcripts are found to be expressed in the population. In this study, we aim to elucidate the mechanism of selective translation from a pool of co-expressed *var* transcripts and the tissue-specific interactome in *P. falciparum* infection. To quantify the expression of PfEMP1 in a single cell, oligonucleotide tagged antibodies will be raised against unique antigenic peptide sequences of PfEMP1s. Since the antibodies carry unique DNA barcodes, they can be sequenced alongside the transcriptome of a single IE. Also, to identify the tissue-specific interactions, we have generated different endothelial receptor specific *Pf*3D7 lines. RNA sequencing of these panned lines shows that multiple *var* genes were enriched in the population. To mimic the physiological condition, we have used the microfluidics system and visualized this interaction between IEs and the host cells real-time under controlled flow of RBCs. Altogether, this study will help to understand the tissue specific sequestration pattern of the malaria parasite *P. falciparum*.

P 062

An Account of Ectoparasites Infesting *Felis catus* and *Canis lupus familiaris in* Two Districts of Imphal, Manipur, India

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Ectoparasite infestation in domestic cats (*Felis catus*) and dogs (*Canis lupus familiaris)* pose a significant health concern, affecting both animal welfare and public health. This study examines the occurrence and species distribution of ectoparasites infesting cats and dogs in two districts of Manipur. A total of 105 cats and 395 dogs from Imphal East and West Districts of Manipur were randomly selected and examined for a period of one year and six months (November 2023-April 2025). Among all examined cats,30 cats in Imphal West and 32 cats from Imphal East were infested. Three flea species including *Ctenocephalides felis (39.68%) Ctenocephalides canis* (30.15%), *Xenopsylla nubica* (15.87%) and two louse species i.e *Heterodoxus spiniger (*7.93%) and *Heterodoxus longitarsus (*6.45%)were found. Out of the total dogs inspected,123 dogs from Imphal West (66.49%) and 145 dogs from Imphal East (69.05%) were infested by one or more ectoparasites. The most prevalent ectoparasite was *Ctenocephalides canis (59.28%)*, followed by *Ctenocephalides felis (19.54%), Rhipicephalus sanguineus (12.05%), Heterodoxus spiniger (6.84%) and Otodectes cynotis canis (2.28%)*. The high prevalence of ectoparasites indicated that cats and dogs were more susceptible to ectoparasite infestation. Extensive public education about pet related zoonosis is needed to create awareness in public and to reduce the risk of zoonotic diseases of public health importance.

P 063

Genome-Wide Analysis Provides First Evidence of Distinct and Heterogeneous *Plasmodium vivax* Populations across India

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India is a highest contributor country in global *Plasmodium vivax (Pv)* cases and harbors half of the global *Pv* burden. Despite being major cause of health burden, insights into population genomic structure and diversity of *Pv* parasites from India remain scarce. Whole genome sequencing of five *Pv* field isolates from previously unexplored, *Pv* endemic regions of India was performed using Illumina platform. Comparative population genomic analyses were conducted by integrating these Indian isolates with global *Pv* population datasets to assess population structure and diversity. In addition, the markers associated with antimalarial drug resistance and vaccine candidate genes were also analyzed. Principal Component Analysis revealed a distinct genetic profile of studied samples. Notably, while distinct, the genetic makeup of our samples bore closer resemblance to other samples from India and Western Asia region. Admixture analysis showed shared ancestry between West Asian and Indian isolates but no ancestry with Southeast Asian (SEA) and Maritime SEA *Pv* population. We also observed various widely reported SNPs across putative antimalarial drug resistance and vaccine candidate genes. Our findings present the first comprehensive evidence for the existence of genetically heterogeneous *Pv* populations across diverse geographical locales within India at the whole-genome level.

P 064

GC–MS Guided Phytochemical Characterization and Mechanistic Evaluation of *Holarrhena pubescens* (Buch. Ham.) Wall. Roots as a Dual Antimalarial and Anti-Inflammatory Agent

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Malaria remains a major global health concern, necessitates the discovery of new therapeutic interventions. *Holarrhena pubescens* is a traditional medicinal plant renowned for its therapeutic potential against infectious and inflammatory diseases[1].This study aimed to elucidate the bioactive chemical constituents of *H. pubescens* root extract using Gas Chromatography–Mass Spectrometry (GC–MS) and to evaluate its antimalarial and anti-inflammatory efficacy. Phytochemical profiling of most prominent extract of *H. pubescens* roots was conducted using GC-MS analysis[2], followed by network pharmacology to predict potential molecular targets and biological pathways. sequential solvent extracts of *H. pubescens* root were evaluated for their antimalarial efficacy against *Plasmodium falciparum* 3D7 using the SYBR Green I fluorescence and Pf-LDH assays[3]. The cytotoxicity properties of the extracts were analysed in HEK-293 and RAW 264.7 cell lines[2]. Anti-inflammatory activity was further investigated in LPS-stimulated RAW 264.7 macrophages by examining pro-inflammatory cytokine gene expression involved in biological pathway of malaria, through ELISA and qRT-PCR techniques. From the sequential solvent extracts the methanolic root extract exhibited the strongest activity with an IC_{50} value of 27.12 ± 2.19 µg/mL and no cytotoxicity to the normal HEK 293 cells (CC_{50}:492.21 ± 3.42 µg/mL) and macrophage RAW 264.7 cells (CC_{50}:536.21 ± 3.19 µg/mL). The extract significantly inhibited the proinflammatory cytokines i.e. TNF-α, IL-6, IL-1β, and regulate the anti-inflammatory cytokine IL-10. Phytochemical analysis of methanolic extract revealed the presence of alkaloids, flavonoids, terpenoids, and phenolics classes of compounds known for antimalarial properties.

P 065

RNA Polymerase II Mediated Regulation of Antigenic Variant Genes in *Plasmodium falciparum*

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The deadliest malaria parasite, *Plasmodium falciparum*, possesses a predominantly euchromatic genome and lacks the canonical topologically associating domains (TADs), lamin-associated domains (LADs), or CTCF-associated loops found in higher eukaryotes. Despite this comparatively "loose" chromatin organization, tight regulation of clonally variant gene families, such as *var* genes, is crucial for pathogenesis. "Just-in-time" expression of only one or a few of these genes enables immune evasion, a key survival strategy. Heterochromatin Protein 1 (PfHP1) plays a critical role in maintaining transcriptional silencing at these loci. In our study, we report that PfHP1 forms condensates and mutations affecting DNA binding and oligomerization disrupt this repression by altering its capacity to form heterochromatic condensates. We further explore the role of RNA Polymerase II in the maintenance of heterochromatin and genome organization in malaria parasites through changes in epigenetic modifications and ncRNAs using high throughput sequencing techniques.

P 066

Cracking the Cuticle: Targeting Calumenin to Combat Filarial Parasites

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Lymphatic filariasis (LF), a debilitating neglected tropical disease affecting 51 million people worldwide, imposes substantial socioeconomic burden. Current mass drug administration (MDA) programs effectively reduce microfilaremia but lack macrofilaricidal activity, and no vaccine is available. Therefore, identifying new therapeutic targets is essential. The nematode cuticle, a complex extracellular matrix, serves as a major barrier to drug penetration and immune clearance; thus, targeting molecules involved in cuticle biosynthesis may enhance parasite susceptibility. Calumenin, a calcium-binding EF-hand protein localized in the endoplasmic reticulum, plays a crucial role in cuticle formation, collagen processing, and ER homeostasis in nematodes. Although well-studied in *Caenorhabditis elegans*, its role in filarial parasites remains unexplored. *Brugia malayi* calumenin (BmCALU1) exhibits low sequence identity (~46.13%) and marked structural divergence (RMSD ~20 Å) from its human homolog, indicating selective druggability. We demonstrate ubiquitous expression of BmCALU1 across all developmental stages and its presence in excretory-secretory products, suggesting a role in host–parasite interactions. Recombinant Bm-CALU1 was cloned, expressed, and structurally characterized by CD spectroscopy. Biophysical assays and gene silencing are going on to uncover its role in cuticle formation and parasite viability. *In silico* screening for potential inhibitors may establish BmCALU1 as a novel molecular target for macrofilaricidal drug discovery.

P 067

Genetic Variations in TLR2 and TLR4 Affect Susceptibility to VL and PKDL in Endemic Areas of Bihar

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Leishmaniasis, a tropical vector born disease, caused by protozoan parasite *Leishmania donovani* and transmitted by *Phlebotomous argentipus.* In India, Leishmaniasis is mainly found in two forms visceral leishmaniasis (VL) and its dermal sequel Post kala azar dermal Leishmaniasis (PKDL). The disease progression from asymptomatic to VL and PKDL depend on various factors including host genetic factors, immune responses, nutritional status and parasitic factors. Toll like receptors (TLRs) recognizes the leishmania parasite and regulate innate immune response involves in phagocytosis, maturation, microbicidal activity of phagosomes by induction of iNOS expression and production of pro-inflammatory and anti-inflammatory cytokines. Genetic variations in TLR2, TLR4 and TLR9 alter susceptibility to infectious and inflammatory diseases. The genetic variation in TLR2 and TLR4 might influence the susceptibility or resistance to VL and PKDL cases. Targeted gene sequencing using Illumina Nextseq 2000 platform for TLR 2 and TLR4 gene was performed in 72 VL cases, 50 PKDL cases 12 Asymptomatic VL cases and 58 endemic healthy controls (192 cases). Genetic variations single nucleotide variants (SNV), Insertion/deletion found both in TLR 2 and TLR 4 gene. Theses genetic variations affect the outcome of *Leishmania donovani* infection in endemic districts of Bihar.

P 068

Novel PA1-Like Protein Homologue from *Taenia solium* Drives Cellular Rewiring of Glucose Metabolism and Immune Responses Underlying Anti-Inflammatory Effects

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Taenia solium, the pork tapeworm, is the causative agent of Neurocysticercosis (NCC), responsible for over 70% of acquired epilepsy cases in endemic regions. Its excretory-secretory proteome (ESP) interacts directly with the host immune cells, yet a vast plethora of molecules still remain uncharacterised. To delve into the molecular complexity of ESPs, we performed proteomic analysis of ESPs, identifying 247 proteins with both pro- and anti-inflammatory potential, ranging from 4–88 kDa. Among these, we focused on a novel PA1 like protein homologue. We investigated the role of PA1 in macrophage metabolism and immune modulation. The quantification of mRNA transcripts and corresponding protein levels was determined using qRT-PCR and immunoblot analysis. The internalization assay revealed efficient uptake and uniform distribution of PA1 in differentiated THP-1 macrophages. Notably, PA1 decreased glucose uptake by reducing GLUT1 expression, disrupting cellular glucose homeostasis followed by the induction of M2 phenotype in macrophages and impairment of autophagic flux. Furthermore, thermal stability of the protein was assessed through MD simulations and temperature sensitivity assays. Collectively, these findings identify PA1 as a stable ESP effector capable of rewiring macrophage metabolism and modulating immune responses, providing novel insights into host–parasite interactions during *T. solium* infection.

P 069

Investigating Persistence to Artemisinin in Human Malaria Parasites

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Artemisinin (ART) is a crucial frontline drug used in the treatment of the human malarial parasite, *Plasmodium falciparum*. It leads to rapid parasite clearance, yet paradoxically, ART monotherapy is associated with frequent recrudescence of infections. Moreover, in a clonal parasite population, only a fraction of the cells survives, suggesting persistence to ART and involvement of stress responses and transcriptional heterogeneity. Here, we aim to characterize the persister population in wild-type and PfKelch13-mutation backgrounds and identify the cellular factors driving ART-induced dormancy and recovery of *Plasmodium falciparum* parasites. Preliminary findings reveal that the persister cells have active mitochondria and are transcriptionally active, which can serve as a biomarker to distinguish persister cells from dead or pyknotic forms in the population. The study findings will lead to a better understanding of the mechanisms at play, including the role of persister cells and potential tolerance strategies used by the parasites.

P 070

Taenia solium Antigens Attenuate *Helicobacter pylori*-Driven Tumorigenic Signalling in Gastric Epithelium

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Taenia solium and *Helicobacter pylori* are most prevalent pathogens in many developing countries, often coexisting within the same environment and population. Chronic helminthic infections like *T. solium* typically induces a Th2-skewed immune response that suppresses inflammation, whereas *H. pylori* triggers chronic gastric inflammation (mostly Th1) leading to atrophic gastritis, intestinal metaplasia, and potentially gastric carcinoma. Despite their overlapping distribution, the immunological interplay between these two infections remains poorly understood. In this study, we investigated the modulatory effects of *T. solium* crude lysate (CL) on *H. pylori*-infected human gastric adenocarcinoma (AGS) cells. AGS cells were pre-treated with *T. solium* CL prior to *H. pylori* infection. Intracellular reactive oxygen species (ROS) level was quantified to assess oxidative stress, while cell viability, proliferation, and apoptosis were evaluated using MTT and complementary assays. Gene expression analysis of mTOR, MAPK, PDK1, and HIF-1α was performed by RT-PCR, and the activation of PI3K, AKT, mTOR, HIF-1α, and PTEN signaling pathways was assessed by Immune-blotting. Pre-treatment with *T. solium* CL significantly reduced *H. pylori*-induced ROS production, suppressed cell proliferation, and enhanced apoptosis in AGS cells. Furthermore, it downregulated tumour- and hypoxia-associated genes while activating the tumor suppressor PTEN. These findings suggest that helminth-derived molecules may attenuate *H. pylori*-driven oxidative stress and oncogenic signaling, potentially offering protective effects against inflammation-mediated gastric carcinogenesis.

P 071

SPV16 Protein Identified as a Novel Biomarker for the Detection of PKDL and VL Co-Infected Cases in Bihar

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Visceral Leishmaniasis (VL) is a life-threatening protozoan disease caused by *Leishmania donovani*. Bihar is the most endemic state for VL in India; an estimated 2-7% of VL cases are co-infected with HIV. HIV has been identified as one of the emerging challenges for VL control. For Diagnosis of leishmaniasis the rk39 strip test frequently used but in case of VL-HIV the sensitivity of rK39 decreased. In some other disease like TB and also in pregnant women it shows false positive result. Although, VL is usually associated with higher titer of antibodies against leishmanial antigens facilitating the serological diagnosis, the repression of the immune system by HIV virus reduces the titer significantly in co-infected patients. In case of PKDL, mostly in macular lesion the sensitivity of gold standard test is decreased, which is also reported in Bihar. To overcome this diagnosis related issues associated with PKDL and VL co-infection, we are working on to develop the novel biomarker with significant sensitivity and specificity for the detection of PKDL and VL co-infected patients. In this study, we have identified SPV16 protein and analyzed its potential as biomarker by Immunoblot and ELISA for the diagnosis of VL, PKDL, VL-HIV, VL-HIV-TB and VL-TB patient's serum with HIV and TB as control. Our results showed that the SPV16 protein identified antibodies in patient's sera samples. IgG- Isotyping was also done to know the type of immune response of antigen before and after treatment, thus our SPV16 antigen is recognized of VL, PKDL and VL-HIV patients significantly instead of control, such as HIV and TB, suggested that our antigen have higher potential or even better than rK39 test to detect PKDL and VL co-infected patients in Bihar.

P 072

De novo Reconstruction and Characterization of var Gene Repertoires from Clinical Isolates of *P. falciparum*

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Plasmodium falciparum, the causative agent of malaria in humans, exhibits antigenic variation to evade immune response from the human host. It mediates pathogenicity by undergoing cytoadherence within blood microvasculature leading to severe complications. Cytoadhesion is mediated through various Variant Surface Antigen (VSAs), which are presented at the surface of the infected erythrocytes. The major VSA protein PfEMP1, encoded by a 60-member hypervariable *var* gene family undergoes Mutually Exclusive Expression (MEE) pattern of expression, to safeguard its antigenic repertoire and exhibit antigenic variation. There is a considerable diversity in terms of sequence and structure within the *var* gene family across different isolates found at multiple geographical regions. We have attempted to create an optimized workflow for *de novo* reconstruction and characterization of *var* gene repertoires from clinical isolates of *P. falciparum*. Our analysis successfully recovered partial to near-complete *var* gene sequences from multiple clinical isolates, revealing extensive inter-isolate diversity in domain composition and sequence mosaicism. This approach enables comprehensive profiling of *var* repertoires from field samples, overcoming limitations of reference-based mapping. *Var* repertoires from clinical isolates would behave like "antigenic fingerprints", revealing how parasites diversify or switch expression to escape host immunity across geographical locations. Finally, in future, mapping *var* gene diversity may reveal conserved epitopes or domains that could be exploited for vaccine designing.

P 073

Aminoacyl-tRNA Synthetase: An Essential Target for Drug Discovery

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Aminoacyl-tRNA synthetases (aaRSs) are essential enzymes responsible for feeding charged tRNAs into the protein synthesis machinery. Beyond this traditional function, these enzymes also play roles in several metabolic and signalling pathways important for cell viability. Some aaRSs have additional pockets in addition to the three substrate binding sites. Inhibition of these enzymes stops protein translation. In recent years, aaRSs have been recognized as targets for drug discovery for the infectious and human diseases. Over the past ten years, we have determined co-crystal structures with inhibitor molecules for four aaRS enzymes of Plasmodium and Toxoplasma parasites[1-5]. Among the 20 aaRSs, phenylalanine-tRNA synthetase (PheRS) has unique features as it consists of two subunits and forms an $(\alpha\beta)_2$ heterotetramer. The N-terminal domain of the α-subunit in apicomplexans and human PheRS contains three DNA binding domains (DBDs), whereas the bacterial enzymes have a coiled-coil structure. The α-subunit contains all three conserved characteristic motifs of class II aaRS, and it recognizes substrates and performs catalytic functions, while the β-subunit recognizes $tRNA^{Phe}$ and performs editing function. Eukaryotic enzymes have an auxiliary pocket and three non-conserved residues between apicomplexans and human enzymes that determine the specificity of the inhibitor[4,5]. Detailed structural comparison and structure-guided inhibitor design will be discussed in detail.

P 074

Understanding the Role of RBR-E3 Ligase in Regulation of Cellular Homeostasis in Human Malaria Parasite

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Human malaria parasite fine-tunes its cellular processes through multiple post-translational modifications of which ubiquitination is the most abundant. Among the E3 ligases, *Plasmodium falciparum* has an evolutionary diverged RBR-E3 ligase which has both RING and HECT-like features with zinc-coordinating domains. This enzyme is expressed throughout the erythrocytic stage of parasite life cycle. *Pf*RBR-E3 ligase catalyzes K6, K11, K48 and K63 mediated polyubiquitination hinting towards its probable biological roles in DNA repair, proteasomal degradation, mitochondrial quality control. Small molecule-mediated perturbation using genotoxic (MMS) and proteotoxic (MG132, FCCP and artemisinin derivative) revealed differences in the immunofluorescence profile of *Pf*RBR-E3. We observed that this ligase interacts with UBCH5 and UBC13 family of E2-conjugating enzymes. Through mutational analysis in *Pf*RBR-E3 ligase, we identified residues in RING1 and RING2 domains critical for ubiquitination activity and protein stability. Pull down assay revealed the interaction of RBR-E3 ligase with folding machinery components suggesting the close coordination between the two machineries to influence the fate of the substrates. Ongoing experiments include identification of client substrates and regulators of RBR-E3 ligase. The findings will help in design of PROTACS mediated degradation of RBR-E3 ligase substrates in human malaria parasite.

P 075

Structural and Functional Insights into *Anopheles* Chemosensory Protein-Insecticide Interactions

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Malaria, a vector-borne disease caused by Plasmodium parasites and transmitted by Anopheles mosquitoes, remains a significant global health challenge, underscoring the importance of vector control as a key strategy for disease prevention. However, the efficacy of this strategy is increasingly undermined by the widespread emergence of insecticide resistance, particularly against pyrethroids. While genetic and metabolic mechanisms of resistance are well characterized, behavioral and sequestration-based mechanisms involving chemosensory proteins (CSPs), are less understood. CSPs play a vital role in mosquito olfactory system, mediating host-seeking and other behavioral processes by transporting odorants molecules to olfactory receptors. Recent evidences suggest that CSPs may also bind insecticides like pyrethroids, contributing to resistance through sequestration. To understand the structural features of Anopheles CSPs and their ligand binding mechanisms, we performed a comprehensive structural, biophysical and computational characterization, evaluating their binding affinities toward various insecticides and identifying key residues potentially involved in pyrethroid interactions through site-directed mutagenesis. High-resolution crystal structures of Anopheles CSPs were solved in apo forms-representing the first such structures reported from Anopheles species. Their strong binding affinities with pyrethroids support the hypothesis of CSP-mediated sequestration. These findings provide new insights into the role of CSPs in insecticide resistance and establish a foundation for developing novel vector control strategies that target CSP–insecticide interactions to combat pyrethroid resistance in malaria vectors.

P 076

Integrative AI and Structure-Based Virtual Screening Approach to Identify Selective Inhibitors of *Plasmodium falciparum* HSP90β

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As increasing evidence suggests that *Plasmodium* parasites are developing resistance to potent antimalarial drugs, there is an urgent need to identify novel therapeutic candidates. To develop a cost-effective strategy for inhibitor discovery, we have established a yeast-based assay system capable of evaluating potential inhibitors against PfHSP90β. *Plasmodium falciparum* HSP90β is a constitutively expressed and previously uncharacterized protein, recently identified by our lab to possess distinct chaperone activity. Given its essentiality in *Plasmodium* and its unique divergence from human orthologs, PfHSP90β represents a promising drug target. Initiating the search for inhibitors, a large ligand dataset was filtered from multiple databases based on availability and pharmacological properties and analysed using a malaria inhibitor prediction AI/ML model, which generated predictive scores and shortlisted top compounds. These were subjected to *in silico* docking using Auto Dock Vina and Smina against PfHSP90β (PDB ID: 3IED), PfHSP90α (3K60), and human HSP90α (6GPW) and HSP90β (6N8Y). Comparative binding affinity analysis revealed several ligands with strong and selective affinity toward PfHSP90β, interestingly these showed minimal interaction with PfHSP90α and human counterparts. The identified lead compounds are being validated in our yeast-based assay system to confirm selectivity, providing a foundation for developing targeted antimalarial therapeutics against the parasite's chaperone machinery.

P 077

Characterization of a Novel Apicomplexan Specific Chimeric Protein

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Toxoplasma gondii is an intracellular pathogen with an exceptional ability to infect a wide range of host cells. The parasite infection is regulated by signaling cascade proteins which drive its lytic cycle in host cells. We report a novel chimeric protein which, evolutionarily, is conserved across Apicomplexans but absent elsewhere in the tree of life. This protein consists of an N-terminal KDAC domain and an IPK domain at the C-terminus. The protein localizes as puncta in the parasite cytosol and its conditional knockdown severely impairs the lytic cycle due to disruption of cell division. Proteomics of conditional mutant revealed dysregulation of multiple pathways affected by the protein depletion. Overexpression, on the other hand, also impacted multiple pathways. We are currently analyzing proteomics data and investigating the biochemical function of KDAC-IPK in *T. gondii*.

P 078

Understanding the Role of Key Protein in Maintenance of Cellular Homeostasis and Developmental Progression in *Plasmodium falciparum*

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Plasmodium falciparum, is a unique and dynamic unicellular organism which possesses organelles like mitochondria, endoplasmic reticulum (ER), apicoplast, golgi apparatus, etc. which form an intricate network of communication amongst themselves. Disturbance in organelle homeostasis by dysregulation of Ca^{2+} signalling, drug stress, inhibition of the proteasome machinery etc. leads to apoptosis like cell death in the parasite. Here, we have explored the putative role of an ER membrane spanning protein, Transmembrane coiled-coil domain containing protein in *Plasmodium falciparum*. In humans, this protein is known to actively prevents Ca^{2+} stores from overfilling, acting as a "Ca^{2+} load-activated Ca^{2+} channel". Moreover, in humans, it is known to associate with the Sec translocon and ribosomes across the ER membrane. The expression and localisation of this protein was analysed throughout the asexual blood stages of the parasite. Inducible knockdown showed a disruption of parasite development from trophozoite to schizont stage, inducing a delayed and impaired developmental cycle progression. Further, we show the association of this protein in organelle Ca^{2+} homeostasis. These results establish the essential role of this protein in cell survival, asexual stage development and cellular homeostasis.

P 079

Field-Based Assessment of Lymphatic Filariasis Transmission Dynamics and Intervention Outcomes in Varanasi District

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Lymphatic filariasis is a neglected tropical disease that imposes a significant burden on the mental, physical, and socioeconomic health of millions of people worldwide. India bears a considerable amount of this burden, with LF endemic in 21 states and union territories. The government of India implemented nationwide Mass Drug Administration and Triple Drug Therapy in different districts, including Varanasi, under Global Programme to Eliminate Lymphatic Filariasis (GPELF). The study is sought to assess IDA coverage in Varanasi from 2019 to 2021 and evaluate its effect on LF transmission through night blood surveys and Transmission Assessment Surveys(TAS) conducted in 2022.The coverage of IDA significantly improved from 74.79% in 2019 to over 80% in 2020–2021. MF rates declined when checked in sentinel sites from 0.94% to 0.15% and similarly in spot check sites it went from 0.46% to 0.10% from duration of 2019 to 2021.TAS 2022 which involved 19,852 eligible participants showed an overall MF rate of 0.1%, excluding areas such as Cholapur with a reported mf rate of 1.34%. This study highlights that the IDA is effective in lowering the transmission of LF in Varanasi, while ongoing surveillance and targeted interventions are still required to achieve the elimination goal by 2027.

P 080

Genes2Me Malaria-Q: A Portable 90-Minute RT-PCR Solution for Comprehensive Malaria Detection from Dried Blood Spots Using Rapi-Q and OnePCR Platforms

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Malaria continues to be a major global health burden, necessitating rapid, accurate, and field-deployable diagnostic tools. We developed a cold-chain free single-tube lyophilized RT-PCR assay for the detection of all five human malaria parasite types (*Plasmodium falciparum, P. vivax, P. malariae, P. ovale, P. knowlesi*) by targeting a conserved genomic region with two portable genes2me POCT platforms:

1. OnePCR System – an integrated single-sample solution that performs automated nucleic acid extraction and RT-PCR amplification in a single cartridge, delivering sample-in to result-out within 90 minutes.
2. Rapi-Q Platform – a higher-throughput solution combining Rapi-X16 for simultaneous extraction of 16 samples with Rapi-Q, capable of analyzing 8 samples per run. Both platforms support whole blood and dried blood spot (DBS) samples, enabling versatile field applications.

The study was performed at National Institute of Biologicals, Noida, UP, India, and results were compared against microscopy and Altona RealStar Malaria PCR kit. The assay

demonstrated excellent diagnostic accuracy, with a sensitivity of 100-96.7% and specificity of 100% across sample types and parasite species. These findings establish Genes2Me Malaria-Q as a robust, portable, and reliable diagnostic solution for malaria detection at the point of care.

P 081

STR Polymorphisms as a Putative Regulator of Gene Expression in *Plasmodium falciparum*

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Although strongly associated with PfK13 mutations, it is becoming clear that artemisinin resistance of *Plasmodium falciparum* is a complex genetic trait underlying a multifaceted molecular mechanism. Transcription-phenotype association studies (TPAS) carried out by our group identified a broad spectrum of genes that can be linked with artemisinin resistance by their differential transcriptional levels. Here, we investigated the polymorphisms of short tandem repeat (STRs) regions of the *P. falciparum* genome with the goal of identifying sequence polymorphism of these putative DNA regulatory elements that could potentially drive transcriptional variations. We identified a broad spectrum of multiallelic sequence genotypes in whole genome sequences of *P. falciparum* field isolates collected during the large-scale epidemiological survey Triple Artemisinin Combination Therapy - Cambodia and Vietnam (TACT-CV) between 2018 and 2020. Using expression quantitative trait loci, we identified a subset of polymorphic STRs (~300) located in the proximity/within genes that associate with transcriptional levels of their target genes (a.k.a. cis-eSTR). Enrichment analysis on these genes with altered expression from the pathways linked with parasite development. Further, the overlap between cis-eSTRs and cis-eQTLs indicated that at least 31 genomic regions, each representing a complex genetic haplotype. We also found 20 genes whose expression was associated with multi-allelic STRs that overlap with TPAS analysis. Overall, these results suggest that the highly dynamic, multiallelic STR polymorphisms located in the proximity of *P. falciparum* genes contribute to the currently ongoing evolution of artemisinin resistance by modulating transcriptional activities. It is feasible to speculate that these eSTRs function in the context of PfK13-SNP (as "a genetic background") but can also function independently, giving rise to alternative artemisinin resistance phenotypes. Our results strongly suggest these polymorphic STRs are probably the missing heritability that links gene-expression regulation among the field isolates and further drives parasite evolution.

P 082

Differential Expression of Metallopeptidase M32 Protein Contributes to Miltefosine Drug Resistance in *Leishmania donovani*

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Leishmaniasis is a spectrum of diseases, from mild cutaneous to life-threatening visceral disease. Miltefosine (MIL) is the only oral therapy for leishmaniasis, yet resistance is rising. Our comparative proteomics study of clinical *Leishmania donovani* isolates revealed a 2 to 3-fold increase in the M32 family metallopeptidase in MIL-resistant versus sensitive parasites, with increased enzyme activity in both promastigotes and intracellular amastigotes. Biophysical analyses show *Ld*MP is predominantly α-helical, thermostable (T_m 63 ˚C), and chemically denaturable (50% unfolding at 3.59 M urea or 0.31 M guanidine HCl). Functional validation demonstrated that *Ld*MP overexpression increases MIL resistance, growth rate, ATP levels, and tolerance to MIL-induced reactive oxygen species, whereas knockdown diminishes resistance, implicating *Ld*MP causally in resistance. As M32 metallopeptidase are absent from most eukaryotes except trypanosomatids, metallopeptidase offers a selective therapeutic target. These findings nominate metallopeptidase as both a drug target and a resistance biomarker. The development or screening of inhibitors designed to target the metal-binding site and exploit the structural stability of the parasite enzyme presents a viable strategy to counter resistance and regain effectiveness against visceral leishmaniasis, whether administered alone or with miltefosine.

P 083

Plasmodium falciparum Plasma Membrane-Localized and Metal Ion-Selective ZIP Family Transporter, *Pf*ZIPCO

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Membrane transporters are crucial for cellular growth and replication as they mediate the uptake of essential solutes. Slc transporters function in the efflux of metal ions from the cytosol to the extracellular milieu (ZnTs) or their influx into the cytosol from outside the cell and from intracellular vesicles/organelles (ZIPs). *P. falciparum* encodes two ZIPs, ZIP1 and ZIPCO, the latter conserved only in some apicomplexan parasites. Our lab has shown that *Pf*ZIP1 has stage-dependent localization to the apicoplast and plasma membranes with preferential binding to Zn^{2+} compared to Fe^{2+} (1). We expressed *Pf*ZIPCO in *E. coli* and checked the metal binding affinity of the purified protein with different metal ions by spectrofluorimetry. In contrast to *Pf*ZIP1, *Pf*ZIPCO exhibited preferential binding to Fe^{2+} compared to Zn^{2+}. *Pf*ZIPCO formed dimers *in vitro* and antibodies generated against *Pf*ZIPCO recognized bands of expected monomer and dimer sizes in the parasite lysate. Subcellular localization through IFA showed that *Pf*ZIPCO localizes primarily to the plasma membrane across asexual and sexual blood stages. In *E. coli,* membrane-localized rec*Pf*ZIPCO increased iron toxicity, indicating its role in Fe^{2+} influx across the bacterial membrane. *Pf*ZIPCO level increased upon intracellular iron chelation, indicating its role in parasite response to metal ion stress.

P 084

Biochemical Profiling and Untargeted Metabolomics in Bancroftian Microfilaremic Cases

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Lymphatic Filariasis, a mosquito-borne parasitic infection, is caused by three closely related filarial worms: *Wuchereria bancrofti*, *Brugia malay*i, and *Brugia timori*. Its spread has been reported in specific regions of the Caribbean, South America, the Middle East, Africa, Southeast Asia, and the Pacific. LF manifests with diverse clinical signs. These symptoms encompass observable conditions such as lymphedema, hydrocele, and elephantiasis, as well as asymptomatic or subclinical infections marked by circulating microfilariae that facilitate disease progression. Diagnosing LF infections is challenging since standard approaches such as microscopy, serological tests, and antigen detection typically exhibit insufficient sensitivity during the asymptomatic stages of infection. In this study, High-Resolution Accurate Mass Spectrometry (HRAMS)-based metabolomics was employed to thoroughly examine host metabolic responses in 84 patients with microfilaremic states before IDA administration, contrasting their metabolic profiles with those of 48 amicrofilaremic controls. Metabolites were annotated with the Compound Discoverer 3.3.3.20 Platform and analysed with MetaboAnalyst 6.0. Multivariate and univariate statistical analyses revealed obvious metabolic distinctions separating the microfilaremic individuals from the normal controls. These results highlight a change in the host serum metabolome of the microfilaremic stage of LF, which may serve as potential biomarkers for disease monitoring and therapeutic targeting.

P 085

Differential Gene Expression Profile across *Leishmania* Parasite and its Implication for Disease Pathogenesis

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Leishmaniasis, which is brought on by a variety of *Leishmania* species, is still a serious global health issue. In order to find comparable gene expression patterns, this work examines the transcriptome responses of human skin tissues infected with *Leishmania tropica* and *Leishmania braziliensis*. For *L. tropica* and *L. braziliensis*, high-throughput RNA sequencing yielded 21.21 million and 85.03 million raw reads, respectively. Both 18.92 million and 80.72 million readings were kept after quality cutting. 2,734 genes were shared by the 3,604 DEGs found in *L. tropic* infections and the 6,121 DEGs found in *L. braziliensis* infections, according to differential gene expression analysis. Twelve of them had divergent patterns, showing species-specific modulation, while 2,722 displayed consistent expression trends, indicating conserved host responses. 1,351 genes were kept for in-depth examination after further refining. Toll-like receptors and Th1 cytokines are upregulated, suggesting an early immune activation meant to curb parasite growth. On the other hand, increased expression of T cell fatigue markers such TIM-3, PTGER2, and LAG3 indicates immunological failure, which promotes parasite persistence. Th1-mediated macrophage activation is suppressed by a strong Th2 response, especially increased IL-4, which increases parasite survival. Furthermore, the immunological balance is further altered by the over expression of the anti-inflammatory cytokine IL-10, which may facilitate immune evasion and accelerate the course of disease.

P 086

Integrating β-Sitosterol and Ultra-Diluted Malarial Antigen for Enhanced Antimalarial Efficacy

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Malaria control is challenged by insecticide resistance, absence of a fully effective vaccine and the emergence of multidrug-resistant *Plasmodium* strains which highlights the urgent need for novel therapies. Plant derived compounds continue to play a pivotal role in malaria treatment and represent promising leads for the development of new antimalarial drugs. The present study investigates the therapeutic potential of β-sitosterol in combination with an ultra-diluted malarial antigen (UdMAg) in BALB/c mice. The anti-plasmodial activity was evaluated using the modified Knight and Peter's suppressive test by monitoring parasitaemia and survival analysis through Kaplan-Meier survival curves. The combination treatment significantly reduced the parasitaemia and enhanced survival compared to the monotherapy groups. Biochemical and immunological assessments including analyses of immune cell profiles, nitric oxide production, reactive oxygen species generation and cytokine levels revealed balanced immune modulation with no signs of toxicity. The study demonstrated a safe and cost-effective strategy for the treatment of malaria. The combination treatment which involves a ultradiluted malarial antigen and active metabolite promotes sustainable and accessible therapeutics for the control of malaria.

P 087

A Phylogeny-Based Approach to Find New Molecular Regulators of Stage Conversion in *T. gondii*

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Toxoplasma gondii, that causes toxoplasmosis, infects ~30% of the world population and poses risks to immunocompromised individuals. Its complex lifecycle includes sexual stages in felines and asexual stages that can infect all warm blooded animals. These asexual stages involves a fast replicating tachyzoite stage and a latent, cyst-forming bradyzoite stage. Under stress conditions, tachyzoites differentiates to bradyzoite-cysts, making them resistant to the host-immune responses and drugs. Hence, understanding this stage conversion is critical. Recent studies have identified key molecular regulators that impact the bradyzoite differentiation like BFD-1 and BFD-2 (Bradyzoite Formation Deficient -1 and 2), eIF1.2 (eukaryotic initiation factor 1.2) and many AP2 transcription factors. Due to the complex differentiation pathway, that responds to multiple stressors, we hypothesize that there are many unknown regulators to be discovered. For this, we employed a phylogeny-based approach, hypothesizing that regulators of stage conversion must be conserved among the bradyzoite-cyst forming members of the family *Sarcocystidae* (*Toxoplasma*, *Hammondia*, *Neospora* and *Besnoitia*) and not in others (*Sarcocystis*, *Cystoisospora*) which form non-bradyzoite cysts. As a non-cyst forming control *Eimeria* and *Cyclospora* were also used from the *Eimeriadae* family, closest to the *Sarcocystidae.* A standalone BLAST of the entire proteome of *T. gondii* against the proteomes of the organisms mentioned was performed, and conservation patterns were identified using K-means clustering in R. Clusters revealed genes that are conserved in *Sarcocystidae*, genes conserved in cyst-forming organisms, highly conserved genes and genes found only in *T. gondii*. In each cluster, a comparison of transcript abundance in bradyzoites compared to tachyzoites under various experimental condition was carried out using published datasets. These analyses revealed candidate pathways and genes including kinases, transcription factors, and RNA binding proteins which may play regulatory roles during stage differentiation.

P 088

Dihydroxy and Paraamine Imidazoquinoline Derivatives as TLR7/8 Adjuvants: Enhancing Recombinant 78 kDa Antigen-Mediated Immunity Against Visceral Leishmaniasis

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Visceral leishmaniasis continues to impose a substantial global health burden, as one of the most severe forms of leishmaniasis, underscoring the urgency of developing effective vaccine interventions. We evaluated the immunomodulatory efficacy of two Toll-like receptor (TLR) 7/8 agonists para-amine and dihydroxy imidazoquinoline derivatives as adjuvants with recombinant 78 kDa *Leishmania donovani* antigen (r78). BALB/c mice were immunized with varying doses of the adjuvanted formulations and evaluated at before-challenge and after-challenge (8 and 16 weeks). Both adjuvants induced a dose-dependent reduction in splenic parasite burden, accompanied by increased reactive oxygen species ROS and nitric oxide production, correlating with elevated *iNOS* and *NF-κB* expression. Immunophenotyping revealed robust $CD4^+$ T cell expansion and moderate but sustained $CD8^+$ T cell responses. Cytokine profiling indicated a Th1-biased immune response, with elevated IFN-γ and TNF-α and reduced IL-10, IL-13 and IL-27 levels. Notably, the immune response induced by the para-amine derivative declined by 16 weeks post-challenge, whereas the dihydroxy imidazoquinoline adjuvanted group maintained heightened cytokine and cellular responses. This is the first report demonstrating the use of para-amine and dihydroxy imidazoquinoline derivatives as TLR7/8-based adjuvants with r78 antigen, establishing dihydroxy imidazoquinoline as a superior and durable adjuvant candidate for vaccine development against VL.

P 089

Designed to Disarm: Structure Based Hits against *Plasmodium falciparum* Lysophospholipases

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Lipid synthesis, catabolism and trafficking are crucial for the malaria parasite's survival. *Plasmodium falciparum* possesses a large family of phospholipases, consisting of 27 putative proteins that have been charted out, of which a few have been studied in detail. Our current work is based on two essential lysophospholipases from *Plasmodium falciparum*, *Pf*LPL1 and *Pf*LPL3, previously characterized by our group. *Pf*LPL1 plays an essential role in lipid homeostasis linked with the hemozoin formation pathway. *Pf*LPL3 localises to the parasitophorous vacuole (PV) and in the tubulovesicular networks (TVN) extensions within host erythrocyte. With our structure-guided approach, we are designing new lead compounds targeting these enzymes that are absent from the host's protein repertoire. In our attempts to elucidate the three-dimensional structure of these proteins, we have cloned, overexpressed and purified both the enzymes and proceeded with their crystallization. Parallely, we have screened for chemical fragment library, that explores a greater chemical space, giving us leverage to design new compounds. Additionally, we have also obtained lead compounds by screening the “Malaria box” library. With our multipronged approach, we aim to identify new therapeutic chemical leads.

P 090

Iron Homeostasis as a Key Regulator of Survival, Redox Balance, and Infectivity in *Leishmania donovani*

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Leishmania donovani relies on iron and heme acquisition within mammalian macrophages to sustain metabolic and redox balance. However, the precise impact of iron availability on parasite survival and pathogenesis remains inadequately understood. In this study, we systematically evaluated the effects of iron supplementation and depletion on *Leishmania* growth, oxidative stress responses, and infectivity. Our results demonstrate that low iron concentrations (0-1000 µM) enhance parasite proliferation and maintain normal morphology, whereas higher concentrations (2000-4000 µM) are toxic, inducing cell death and DNA fragmentation. Both iron excess and its deficiency activated caspase-like pathways and DNA damage, implicating iron-mediated apoptosis mechanisms. Lower iron supplementation enhanced parasite resistance to oxidative stress by reducing reactive oxygen species (ROS) levels and maintaining intracellular thiol pools. At the molecular level, iron-rich conditions upregulated key Fe-S cluster proteins (Nbp35, IscU) and thiol-related enzymes (TryS, TryR, CS, SAT, cTXNPx), supporting metabolic and antioxidant functions. Conversely, iron chelation using deferoxamine (0–50 µM) impaired parasite growth and disrupted redox homeostasis, thereby exacerbating oxidative damage. Iron deficiency downregulated these proteins, except for frataxin protein, which was upregulated because it is an iron storage protein. Iron supplementation increased *L. donovani* infectivity while iron depletion significantly reduced parasite viability using THP-1 macrophages. Thus, our findings highlight the pivotal role of iron in regulating parasite virulence, redox homeostasis, and host-pathogen interactions.

P 091

Comprehensive Profiling of *Taenia solium* Neuropeptides Reveals Potent Anti-Inflammatory Roles and Therapeutic Potential in Neurocysticercosis

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Neurocysticercosis, caused by larvae of *Taenia solium*, is the leading helminthic infection of the central nervous system and major cause of acquired epilepsy globally. Viable cysts modulate or suppress host immunity, directly influencing symptom onset. Antigenic fractions from viable cysts, including proteins and neuropeptides, interact with and mimic host nervous and immune cells. While neuropeptides of other helminths are studied, *T. solium* neuropeptides and their biological effects remain largely unknown. Here, for the very first time, we employed an in-silico approach to identify and annotate precursor and mature neuropeptides of *T. solium*. Following bioinformatic characterization, neuropeptides from viable cyst were purified using acidified methanol extraction. Their immunomodulatory roles were then systematically assessed via functional assays on freshly isolated PBMCs. A total of 194 mature neuropeptides from 134 precursors were identified and classified through in-silico analysis, revealing key roles in extracellular matrix organization, receptor, and tyrosine kinase signaling. Tricine SDS-PAGE confirmed that peptides extracted from cyst fluid predominantly ranged from 1–6 kDa, aligning with known neuropeptide profiles. Functional assays- RT-qPCR and intracellular ROS analysis on PBMCs, demonstrated robust anti-inflammatory activity of these neuropeptides.

These neuropeptides, can therefore, act as promising novel targets for therapeutic strategies against neurocysticercosis and associated neuro-immunological disorders.

P 092

Zoonotic Invader Species From Freshwater Gangetic Ecosystem to Arabian Sea Coastal Ecosystem

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The transformation of metals and elements on body of nematodes in freshwater Gangetic ecosystem into a range of rare earth metals was the order of variation in degree of intense interactions of environmental parameters with metal and mineral constituents. The dynamics of infections of nematodes in response to the occurrence as well as fluctuations in specific rare earth metal content was the order of variation in degree of intense interactions of environmental parameters with metal and mineral constituents. The strong toxic effect of even lower concentrations of cadmium occurred on a majority of aquatic organisms that survived merrily on aquatic resources. Resultantly the suppression of calcium uptake giving way to hypocalcemia followed by embryonic deformities are effectuated in larvae as well. It was thus hypothesized on lines of earlier illustrations that the potential of parasitic nematodes would enable these organisms to accumulate trace metals, that are toxic in nature, to a much higher extent in roundworms than in the host fish.

P 093

Hsp90 Mediated Cellular Homeostasis of Ribonucleotide Reductase and Its Impact on *Plasmodium* Replication

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Plasmodium falciparum undergoes endo-reduplication during the schizont stage, in which it's nuclear, mitochondrial and apicoplast genomes are replicated multiple times without cytokinesis. *Plasmodium* adopts this mode of cell division three times during their sexual and asexual developmental cycle. Due to these unusual dynamics of DNA replication, replication proteins appear to be excellent drug targets. We are working on understanding the mechanism behind the intracellular stability of ribonucleotide reductase, which catalyses the reduction of ribonucleotides to deoxyribonucleotides. Previously we identified the specific cochaperone of PfHsp70-1 that is involved in the earlier folding intermediates of PfR2 (ribonucleotide-reductase-subunit-2)[1]. We find that PfR2 requires additional maturation by *Plasmodium* Hsp90 chaperone system. Our study establishes PfR2 as a direct client of Hsp90 and shows that under Hsp90 inhibitory condition, PfR2 undergoes proteasomal degradation which subsequently results in significant reduction in the dNTP pool of the parasite. We have identified one Hsp90 cochaperone that is essential for PfR2 stability. The targeted disruption of that cochaperone shows moderate destabilisation of PfR2 resulting in higher sensitivity of parasites towards hydroxyurea, a catalytic inhibitor of PfR2. We propose that targeting the axis between Hsp90 and that cochaperone can be employed as a strategy to target parasite DNA replication.

P 094

Optogenetic Illumination of cAMP Signaling in *Toxoplasma gondii*

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Toxoplasma gondii is a ubiquitous intracellular parasite capable of infecting a wide range of hosts. Its developmental progression and virulence are governed by cAMP, cGMP, calcium and lipid signaling, but the effector proteins of these pathways are primarily unknown. To decipher the players of cAMP signaling during the lytic cycle of *T. gondii*, we engineered parasites to express a photo-activated adenylate cyclase. Our optogenetic strain enabled rapid, spatiotemporal, reversible and inheritable light-dependent induction of parasite cAMP. Phosphoproteomic analysis identified >13000 phosphopeptides, of which 1,061 were modulated upon light-activation of cAMP-PKA. A meta-analysis of our data with the PKAr-AID[1] strain identified several shared proteins, including six candidates containing the canonical PKA consensus motif. We examined one such protein by conditional mAID-mediated degradation, demonstrating its critical role in parasite growth. Our subsequent work disclosed a protein complex involved in DNA replication. Proteomics of the mutant revealed a decline in DNA-binding proteins, including the complex subunits and AP2 factors. We are currently analyzing the effect of cAMP-PKA-dependent phosphorylation on the cell cycle by mutagenesis studies.

P 095

An Apicoplast-Resident Phospholipase is Essential for Lipid Homeostasis and Lytic Cycle of *Toxoplasma gondii*

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Asexual reproduction of the intracellular parasitic protist, *Toxoplasma gondii*, requires membrane synthesis, organelle biogenesis and lipid homeostasis. In order to decipher a role of phospholipase enzymes in these processes, we identified nine phospholipases, including three previously known proteins, present in the acutely-infectious tachyzoite stage. Genomic tagging revealed varying localization of novel phospholipases, of which *Tg*PL4 is expressed in the apicoplast, indicating its role in organellar lipid remodelling. Conditional depletion of TgPL4 by a tetracycline-regulatable system impaired the lytic cycle, with severe defect in cell division. Proteomic and lipidomic analyses of the PL4-depleted strain suggested a dysregulation of protein and lipid syntheses. We are now investigating the importance of PL4 in biogenesis of apicoplast and other organelles.

P 096

Role of Angiotensin Pathway in Cerebral Malaria

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Cerebral malaria (CM), a severe neurological manifestation associated with *Plasmodium falciparum* infection, often leads to coma and death, and survivors may suffer from long-term neurocognitive impairments. Despite the availability of potent anti-malarial drugs, none of them provide complete efficacy in preventing or treating CM. This underscores the urgent need for adjuvant therapeutic strategies that can mitigate malaria-associated neurological damage. In this study, we explored the protective potential of targeting a shared molecular pathway between hypertension and cerebral malaria. Recent evidence implicates the angiotensin II type 1 (AT1) receptor in both hypertension and malaria pathogenesis. AT1 activation contributes to blood–brain barrier (BBB) disruption during CM through upregulation of β-catenin. Therefore, we investigated the antihypertensive AT1 receptor blockers, irbesartan and losartan, for their ability to prevent CM in Plasmodium infected C57BL/6 mice. Treatment with these drugs resulted in reduced expression of β-catenin, TCF, LEF, ICAM-1, and VCAM-1, while restoring the levels of VE-cadherin and vinculin, key components essential for maintaining BBB integrity. Additionally, drug-treated mice exhibited decreased pro-inflammatory and elevated anti-inflammatory cytokine levels, suggesting attenuation of inflammatory responses. Notably, the mean survival time of treated mice increased significantly, even without concurrent anti-malarial therapy. When combined with the anti-malarial agent α/β-arteether, irbesartan or losartan achieved an 80% cure rate, compared to 60% with α/β-arteether alone. These findings highlight the potential of AT1 receptor blockers as effective adjuncts to standard anti-malarial therapy for preventing cerebral malaria associated neuropathology.

P 097

Environmental Interactions Triggering Dynamics of Anisakid Zoonoses in Sagar

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The subtle intermixing of species of tapeworms and roundworms has come to notice after emergence of Betwa-Ken Link canal in Sagar M.P. in 2025. The cestodes in freshwater fish, *Mastacembelus armatus* were *Polyonchobothrium armatii* (Malhotra, 1983) and *Gangesia sanehensis* (Malhotra, Capoor and Shinde, 1980) detected prior to emergence of Link canal. However, post-Link canal formation between Betwa-Ken, the fauna changed to the extent that the roundworms with strong anisakid features of buccal tooth atop cephalic complex, a prominent physalopterid cephalic collar along with typical anisakid ventricular complex comprising a bulbous ventriculus, intestinal ceca and ventricular appendix replaced roundworms with primitive characteristics in *M. armatus* in the fresh water body. This also contradicted the earlier hypothesis that members of Anisakidae water invariably inhabitants of marine habitat fishes because at the Rajghat reservoir of Sagar, Madhya Pradesh, the newly evolved roundworms were essentially inhabitants of freshwater eel in India. The intricacies of the effect of transformation of morphological characteristics of roundworms from marine ecosystem to Ken-Betwa Link Canal (*i.e*. freshwater) shall be discussed.

P 098

Geospatial Mapping and Influence of Seasonal Variability on Mosquito Vector Density in Chandigarh (North India)

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Mosquito-borne diseases remain a serious global public health concern including India, where rapid urbanization and variable weather create ideal conditions for vector abundance. In order to map and identify mosquito vector hotspots, the current research combines entomological monitoring with meteorological and geographical data from four ecologically divided habitats of Chandigarh. Adults were collected periodically (June to November) throughout a three-year period (2022–2024), followed by entomological identification and morphometric analysis. Using Geographic Information System (GIS) tools, vector hotspots were mapped and overlayed with meteorological factors including maximum and minimum temperature, humidity, and rainfall to assess spatial correlations. During present studies, hotspot analysis and spatial trends revealed significant presence of several mosquito vectors of malaria, dengue or chikungunya, Japanese encephalitis, and filariasis belonging to genus *Anopheles, Aedes,* and *Culex* in urban and rural survey areas, strongly associated with high rainfall and humidity thresholds during pre-monsoon to post-monsoon periods. The results highlight the importance of seasonally prevalent vector surveillance in efforts to control mosquito borne illnesses. By using an integrated strategy, disease outbreaks may be better predicted and vector control tactics can be planned in advance.

P 099

Functional Characterization of EhPIG-K, the Catalytic Subunit of the GPI Transamidase Complex Essential for GPI Anchor Biosynthesis in *Entamoeba histolytica*

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Glycosylphosphatidylinositol (GPI) anchors are essential for tethering surface proteins that mediate host–parasite interactions and contribute to the virulence of *Entamoeba histolytica*. The terminal step of GPI anchor attachment is catalyzed by the GPI transamidase (GPI-T) complex, with EhPIG-K functioning as its catalytic subunit. In this study, we investigated the subcellular localization and functional significance of EhPIG-K in the biosynthesis of GPI anchors. Immunofluorescence microscopy revealed that EhPIG-K localizes predominantly to a reticular network resembling the endoplasmic reticulum, extending beneath the plasma membrane and around the perinuclear region, sites characteristic of GPI anchor biosynthesis. Functional assays using a fluorescent peptide substrate confirmed its peptidase activity and defined optimal pH and divalent cation requirements. To assess its physiological significance, *E. histolytica* cell lines overexpressing (EhPIGK-S) and silenced (EhPIGK-AS) for EhPIG-K were generated under a tetracycline-inducible system. Silencing of EhPIG-K significantly reduced trophozoite proliferation, erythrophagocytosis, and viability as compared to overexpressing and wild-type cells. Moreover, surface expression of the Gal/GalNAc lectin, a key GPI-anchored adhesion molecule, was significantly reduced in EhPIG-K-silenced trophozoites. These findings identify EhPIG-K as a crucial catalytic component of the GPI-T complex, essential for GPI anchor biosynthesis, surface protein localization, and parasite virulence, highlighting it as a potential therapeutic target for amoebiasis.

P 100

Identification of Theranostic Biomarkers in VL-HIV Patients through Plasma Proteomic Profiling

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Visceral leishmaniasis (VL) and HIV co-infection present significant healthcare challenges in resource-limited settings, worsening immunosuppression and complicating diagnosis and management, leading to increased mortality rates. The current serological test, rK39, is less reliable for diagnosing VL-HIV co-infection due to a low humoral response. This study aimed to identify theranostic biomarkers through plasma proteomic profiling in patients co-infected with VL-HIV. A total of 20 baseline blood samples were analysed, including those from VL-HIV co-infected patients (VL-HIV), HIV-positive individuals (HIV), VL patients (VL), and healthy controls (HC), collected between February 2022 and December 2024 at ICMR-RMRIMS, Patna. Using mass spectrometry and bioinformatics, we identified distinct proteomic profiles in the following comparisons: VL-HIV vs. HI, 37 proteins were downregulated and 64 were upregulated; VL-HIV vs. HIV, 36 proteins were downregulated and 77 were upregulated; and VL-HIV vs. VL, 96 proteins were downregulated and 68 were upregulated. Moreover, protein-protein interaction analysis identified biologically relevant clusters in each comparison, with key proteins such as HLADRB1, C4B, and CHI3L1 showing significant interconnections. These findings underscore the need for more accurate diagnostic tools and point to potential biomarkers that could enhance the understanding and management of VL-HIV co-infection, highlighting an essential step toward improving patient outcomes in affected regions.

P 101

Preliminary Studies on Prevalence of Ectoparasitic Infestation in Horse, Manipuri Pony (*Equus ferus caballus)* from Manipur, India

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Ectoparasite infestations are a growing concern for horse owners and equine veterinarians alike. This infestation is a primary threat to cost-effective livestock production by damaging skin and transmitting multiple diseases among animals. The prevalence of ectoparasites infestation increases around the world and information on successful treatment and control of ectoparasites infestation in horses, especially in Manipur is very limited. In a preliminary studies on prevalence of ectoparasites in Manipuri Pony, a total of 48 pony(29 males and 19 females), were examined from different valley areas of Manipur.Out of 48 pony ,20 were found to be infested.In the present investigation,one species of ectoparasitic lice *Bovicola equi* and two genera of ticks *Rhipicephalus sp* and *Ixodes sp* were revealed in the infested pony .Among them,*Rhipicephalus sp* showed the highest occurrence having 85% followed by *Ixodes sp*(35%) and *Bovicola equi*(10%). This work is the new investigation report for such kind of studies. The investigation has started from May 2025 to September 2025 at different localities of different Districts of Manipur and the collected specimen was brought for identification in the Parasitology laboratory, Department of Zoology, Dhanamanjuri University, Imphal, Manipur, India.

P 102

A Human Skeletal Muscle Cell Model for Toxoplasma-Induced Subversion

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Toxoplasma gondii is an obligate intracellular parasite, which infects nucleated cells in a wide range of species. Skeletal muscles serve as a key niche for chronic development and tissue reservoir for parasite transmission, yet these host cells remain underutilized for parasite-host interaction studies. This work deployed immortalized human skeletal muscle cells (KD3) to discover physiological and infection-relevant myogenic factors. We present comprehensive multi-omics profiling of KD3 cells, including proteome and transcriptome, identifying myogenic biomarkers, regulatory networks and microRNAs that govern muscle differentiation. Comparative analysis with the conventional murine C2C12 model and ex-vivo mouse/human samples disclosed human-specific networks, highlighting clinically-relevant pathways for infection and pathophysiological diseases. Our extended work demonstrated parasite-triggered impairment of myogenesis in KD3 cells and identified many potential host-cell differentiation and infection determinants.

P 103

The Multifunctional Autophagy Pathway as a Potential Drug Target for Malaria

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Autophagy plays a crucial role in organelle turnover, metabolic reprogramming, and intracellular remodeling during the differentiation of Plasmodium parasites that transition between mosquito and mammalian hosts. The metamorphosis of sporozoites into EEFs is characterized by spectacular changes in parasite morphology and major interior reconstructions, accompanied by the loss of organelles that are unnecessary for the replication of the liver forms. Organelles required for stage-specific function are preserved, whereas the superfluous ones that have completed their roles are discarded. The role of autophagy in maintaining pathogenicity and cell homeostasis in parasitic protozoans is emerging. Some of the autophagy-related proteins' unique features suggest possible new targets for drug discovery. Previously, our knockout studies of autophagy-related E1-like enzyme Atg7 in rodent malaria parasite P. berghei had established its essentiality in blood and liver stage development whereas, here, we identified several compounds from the Maybridge library with a high docking score against PfAtg7. These compounds also inhibit apicoplast biogenesis and parasite development in both blood and liver. We failed to express the full-length protein to establish the target-specific activity of selected drugs. Furthermore, we codon-optimized the catalytic domain of the same and successfully achieved a bacteria-driven expression of it as a GST fusion protein. The purified protein was used to set up fluorimetry experiments. The identity of the catalytic domain was confirmed via ATP binding assay with appropriate GST control. Moreover, we also validated some selected compounds that killed the parasite in an in-vitro culture to be target-specific inhibitors of Atg7. Overall, this study further proves the indispensable role of Atg7 in Plasmodium blood and liver stages and, thus, highlights its potential to be a potent antimalarial drug target for killing the parasite.

P 104

Comparative Lipidomics Identifies Strain-specific Metabolic Signatures in *Toxoplasma gondii*

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Globally-prevalent and clinically-relevant intracellular protozoan parasite, *Toxoplasma*, comprises only a single extant species (*T. gondii*) but displays strain-specific diversity with differential virulence and host adaptation. While the strain-specific polymorphism at the protein level is understood, the lipid repertoire of common parasite strains are not known. We performed targeted and untargeted lipidomic analysis of GT1 (Type I) and ME49 (Type II) strains and identified >1200 metabolites, including phospholipids, sphingolipids and neutral lipids. Notably, 38 and 4 lipid species were found exclusively in GT1 and ME49 strains respectively, which can be exploited as diagnostic markers. Comparative lipid abundance analysis confirmed strain-specific profiles and pathway enrichment revealed pronounced differences in glycerophospholipid and sphingolipid metabolism. Our findings provide novel insight into the metabolic heterogeneity of *T. gondii* strains, potentially contributing to strain-specific pathogenic traits.

P 105

Optogenetic Dissection of cGMP signaling in *Toxoplasma gondii*

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Toxoplasma gondii, an obligate intracellular parasite of high clinical importance, relies on finely orchestrated signaling cascades to control its lytic cycle in mammalian hosts. The second messenger, cyclic guanosine monophosphate (cGMP), is a critical regulator of parasite motility, which in turn drives the process of invasion and egress (in/out of host cells). Dissecting its spatiotemporal dynamics and signaling mediators has been hindered by the limitations of genetic and pharmacogenetic methods. To overcome this, we deployed a novel optogenetic strategy to control parasite signaling precisely. We adopted a light-activated guanylate cyclase and generated multiple optogenetically-modified parasite strains to investigate the interconnected signaling pathways. Brief, targeted illumination of parasites facilitated rapid, temporally precise, and reversible modulation of intracellular cGMP. Photo-induction of the cGMP-PKG pathway activated the parasite locomotion-dependent invasion and egress. Next, we coexpressed the light-activated guanylate cyclase with a genetically encoded calcium biosensor in the parasite and demonstrated a direct control of cytosolic Ca2+ by cGMP. Subsequent phosphoproteomic analysis of our light-activated parasites revealed PKG-dependent phosphorylation of several proteins potentially regulating ion homeostasis and parasite motility. We are currently testing the roles of these novel cGMP-signaling mediators.

P 106

In vitro Monitoring of Malaria-Infected Mice Blood Using Photoacoustic Technique

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Pathogen prevalence among human is co-evolving with host physiology and successful diagnosis of pathogenic disease is extremely depends on fast track detection of pathogenic load. Malaria is such kind of disease caused by *Plasmodium sp.* in most tropical countries including India. Development of optics based non-invasive malaria diagnostic protocol may be beneficial compared to invasive biochemical tests to eliminate expertise. Swiss albino mouse was selected as a host and infected with *Plasmodium berghei*. Mice were sacrificed to collect blood as a sample and parasitemia % are counted by Giemsa staining while further verified by fluorescent staining. Photoacoustic (PA) and UV-Visible experiment were conducted within few hours after collection of blood. There were variable PA signal observed in different parasitemia counts (%) starting from 0% to 18%. PA signals measured at 660 nm and 980 nm wavelengths were selected for oxygen saturation (SO_2) calculation. SO_2% in uninfected blood sample was observed to be significantly higher in compared to infected blood samples. While hemoglobin concentrations were lower in infected samples compared to uninfected ones. However, UV-Vis experiment result did not display a correlation with infection level. This observation exhibits that a strong relationship between PA spectra (vis-à-vis derived parameters- SO_2%, hemoglobin concentration) and malaria infection level.

P 107

Molecular Characterization (sequencing) of Heat Shock Protein-70 gene of *Trypanosoma evansi* using PCR

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The present study was carried out to isolate the Heat Shock Protein 70 gene of *Trypanosoma evansi* using PCR. *Trypanosoma evansi* infected camel was confirmed by examination of Giemsa stained blood smear of camel blood. After confirming infection, the *T. evansi* collected from camel blood were propagated in Swiss albino mice and the blood of mice was collected from heart region after dissecting the mice which had massive infection. DEAE cellulose chromatography was done for purification of trypanosomes from blood of mice. DNA extraction was done from collected pellets of *Trypanosoma evansi* using the phenol-chloroform extraction followed by ethanol precipitation. The desired amplicons of HSP-70 genes were then amplified by PCR using gene specific primers. Amplified PCR products were analyzed on 1.2% agarose gel stained with ethidium bromide and identified on the basis of size of the HSP-70 genes. The amplicons of expected size were purified from the 1% low melting agarose gel employing illustra GFX PCR DNA and Gel Band Purification Kit. The DNA fragment of interest was then ligated to the pGEM- T Easy vector and ligated mixture was transformed into *Escherichia coli* JM109 strains. Screening of recombinants was done by Restriction Enzyme digestion of plasmid DNAs using EcoRI and found that the release of DNA fragments 1956 bp. After confirmation of clones of HSP-70 genes the plasmid DNAs was sequenced. The bioinformatic analysis of Heat Shock Protein-70 gene was done by using Bio Edit Sequence Alignment Editor, ExPASy: SIB Bioinformatics Resource Portal and NCBI open Reading Frame finder. Six Open Reading Frames were generated (3 sense and 3 Anti sense) for both Cysteine Protease and Heat Shock Protein-70 genes by ExPASy and NCBI ORF finder.

P 108

First Report of *Sarcocystis* spp. in a Cat in Rewa, Madhya Pradesh

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Sarcocystis is an obligate intracellular, cyst-forming isosporan coccidian parasite of carnivores, especially dogs and cats. A stray cat entered a house in the Veterinary College Campus, Rewa during late night or early morning hours in search of milk, curd, or similar food items. Incidents of empty bowls or utensils previously containing milk, along with random defecation on clothes emitting a characteristic pungent odour, were reported. The faecal matter appeared brownish, watery or runny in consistency, containing coarse fibrous material but no mucus. Faecal samples adhered to the clothes were collected and brought to the laboratory for examination. Microscopic examination revealed the presence of *Sarcocystis* spp. sporocysts, observed as single or multiple banana-shaped structures within each microscopic field, each containing four sporozoites. This appears to be the first report of *Sarcocystis* infection in a cat from the Rewa district. Furthermore, this tissue-dwelling parasite is a significant cause of sarcocystosis in growing ruminants and swine. This report may contribute valuable insights into the epidemiology, life cycle, and development of effective control strategies for *Sarcocystis* spp. infection in domestic animals.

P 109

From Code to Chip: Towards Development of a SARS-CoV-2 Biosensor and Mechanistic Insights from a COVID-19 Diagnostic Perspective

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Rapid and precise detection of infectious agents remains central to global health surveillance and epidemic control. Biosensors, miniaturized analytical devices integrating biological recognition with signal transduction, are advancing medical diagnostics due to their sensitivity, specificity and portability. This study outlines an early step towards developing a novel lectin-based lateral flow assay (LFA) biosensor for early and rapid SARS-CoV-2 detection through an integrative "code-to-chip" approach combining *in silico* molecular analysis and *in vitro* validation. Molecular docking followed by MD simulation identified multiple Cucurbitaceae seed lectins (CSLs) having strong predicted affinity and stable glycan-binding interactions with N-linked glycans of SARS-CoV-2 spike glycoprotein. *In silico* evaluation confirmed functional activity, non-toxicity and robust glycan recognition of SARS-CoV-2 spike glycoprotein by selected CSLs that are being prioritized for biosensor fabrication. Lectin selection and device performance would be optimized through combined computational and wet-lab results. These insights guide development of an LFA biosensor for early, rapid and label-free detection of SARS-CoV-2 in clinical and environmental samples, offering field-level applicability with minimal reagents, high sensitivity and instant visual readout. Importantly, proposed design framework and glycan-recognition principle are adaptable to diverse viral and parasitic pathogens, positioning lectin-based biosensors as promising next-generation tool for point-of-care diagnostics and epidemic preparedness.

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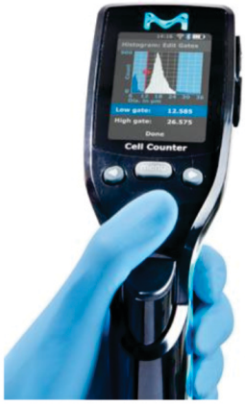

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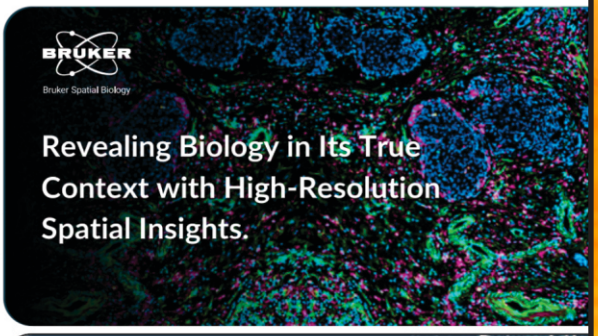

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