MIRA International Research Network

Franco-Indian workshop on infectious diseases & antimicrobial resistance

An international initiative to strengthen research and innovation capabilities aimed at discovering new therapeutic and preventive approaches to pathogenic bacteria and antibiotic resistance

May 26-28, 2025, Toulouse

Centre de biologie intégrative
Amphithéâtre Nicole Le Douarin

CEFIPRA

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Brochure produced by Aélia Farge

Summary

Franco-Indian Workshop on Infectious Diseases and Antimicrobial Resistance

An international collaborative initiative to strengthen research and innovation capabilities aimed at discovering new therapeutic and preventive approaches to pathogenic bacteria and antibiotic resistance.

Date: May 26-28, 2025

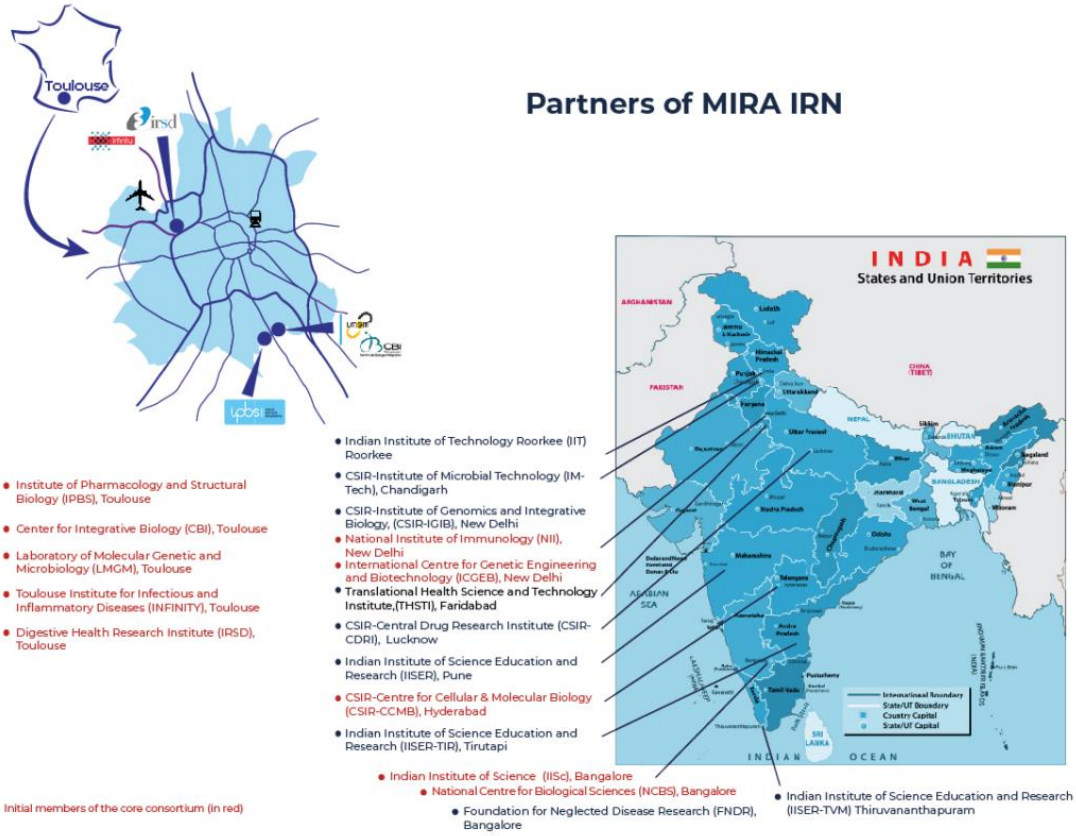
Venue: Centre for Integrative Biology, Amphitheatre Nicole Le Douarin and Reception Hall – 169 rue Marianne Grunberg-Manago, Université de Toulouse, 31062 Toulouse, France

Keywords: emerging or re-emerging infectious diseases, antimicrobial resistance and tolerance, surveillance/epidemiology, antimicrobial target, alternative therapies, host directed therapies, pathogenic bacteria, tuberculosis, ESKAPE, diagnostics, vaccines

Audience: academic and private scientists with an interest in the field of (re-) emerging infectious diseases and antibiotic resistance and wishing to expand their international cooperation; Master and PhD students

Recent decades have seen unprecedented changes in human-environment interactions and increasing disruption of ecosystems and climate, creating a particularly favourable context for the (re-)emergence of infectious diseases. In addition to this threat, the worldwide spread of (multi-)antibiotic resistant bacteria represents a major short-term threat to global health. It is therefore essential and urgent to strengthen our efforts to put in place all the necessary means for prevention, surveillance and response to emerging infectious diseases and antibiotic resistance.

By combining their strengths and complementary resources, 9 core consortium international partners (4 French and 5 Indian) have made up the MIRA* "International Research Network (IRN)" in 2023, with the support of the CNRS (2023-2027). In 2024, 9 additional Indian partners joined the network, further strengthening this international collaborative effort. The 18 members of the current MIRA network share the common objective of strengthening their research and innovation capacities, from the most fundamental research to applied research for the discovery of new therapeutic and preventive approaches against pathogenic bacteria, through an international collaborative approach. The partners are committed to developing and structuring various areas of cooperation and implementing strategies to sustain their joint efforts in support of global health.



Initiated by the MIRA consortium, this Franco-Indian workshop is jointly organised in Toulouse by the IPBS, the LMGM-CBI, Infinity and the IRSD, with the main financial support of the CNRS and the CEFIPRA, and additional support of the Region Occitanie, the French Embassy in India, the ANTABIO company, the federative research structure "Biology and Biotechnologies for Health" (SFR-B2S) and the University of Toulouse.

For 4 half-days, 45 scientists in the field - 22 from India and 23 from France, including 26 men and 19 women ; among them, 10 young researchers, 6 from France and 4 from India, including 6 women and 4 men - will have the opportunity to present their work and exchange ideas with a view to developing, structuring or strengthening collaborative research programmes. The workshop is in hybrid format to allow participants to all over the world to attend.

This scientific and information event is designed to encourage the research community to develop cooperation with India, and to present existing funding mechanisms. The participation is open free of charge to the entire community. Masters and PhD students and postdoctoral fellows are strongly encouraged to participate to introduce themselves to international cooperation from their first research experience and offer them opportunities in India or France for further studies or career development.

More information: https://www.ipbs.fr/mira2025/

MIRA = **Maladies Infectieuses émergentes et Résistance aux Antibiotiques** / Emerging Infectious Diseases and Antimicrobial Resistance

Scientific Committee

Olivier Neyrolles

CNRS Research Director and Director of the Institute of Pharmacology and Structural Biology (IPBS), Toulouse, France

French Coordinator of the IRN MIRA

Olivier Neyrolles, an agricultural engineer by training, earned his PhD in microbiology at the Pasteur Institute, Paris. After postdocs at Imperial College London and Saint-Louis Hospital, he joined Prof. Brigitte Gicquel's lab at the Pasteur Institute, specializing in mycobacterial biology. He became a CNRS Research Associate (2004) and later founded his lab at IPBS (2007). Now CNRS Research Director, he focuses on tuberculosis and respiratory pathogens. His work has earned major awards, including the CNRS Bronze and Silver Medals, the Sanofi-Institut Pasteur Award, and the Coup d'Élan Prize from the Bettencourt Schuller Foundation, and his lab is recognized for its innovation, global collaborations, and mentoring of young scientists.

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Amit Singh

Professor at Center for Infectious Disease Research
(CIDR), Indian Institute of Science (IISc), Bangalore, India

Indian Coordinator of the IRN MIRA

Amit Singh is Professor in Microbiology and Cell Biology. Amit Singh's group exploits interdisciplinary strategies to dissect the redox basis of persistence in human pathogens *Mycobacterium tuberculosis* and HIV. By taking advantage of redox biosensors, XF-flux analyses, omics-based strategies, and animal studies, his work has helped find new mechanisms of how macrophage's acidic pH mobilizes drug tolerance in *M. tuberculosis*. In 2015, he was awarded the Senior DBT-Wellcome Trust India Alliance fellowship. Most recently, he was awarded the Shanti Swarup Bhatnagar Award, the most prestigious prize by the Council of Scientific and Industrial Research in India. He is a member of the National Academy of Sciences, India.

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Nathalie Campo

CNRS Researcher at Laboratoire de Microbiologie et Génétique Moléculaires (LMGM-CBI), Toulouse, France

Nathalie Campo is a CNRS researcher in the "Pneumococcal Competence and Transformation" team headed by Patrice Polard at the Laboratoire de Microbiologie et Génétique Moléculaires (Toulouse, France). Over the last 15 years, she has developed multidisciplinary approaches including the use of far- end imaging techniques at different levels of resolution to visualize and characterize the process of natural genetic transformation in the human pathogen *Streptococcus pneumoniae*.

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Anjana Badrinarayanan

Associate Professor at National Centre for Biological Sciences (NCBS), Bangalore, India

Anjana's lab employs quantitative live-cell imaging approaches to track, in real-time, the dynamics of single cells and molecules within cells. With this they address frontier and fundamental questions about the regulation of genome duplication and DNA damage repair in microbial systems. Their work has provided new mechanistic insights into the general principles of genome maintenance and its impact on microbial adaptation and survival.

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Julien Vaubourgeix

Assistant Professor at Digestive Health
Research Institute (IRSD), Toulouse, France

After completing his PhD in the laboratory of Dr. Mamadou Daffé at the Institute of Pharmacology and Structural Biology in 2009 in Toulouse, France, Julien joined the laboratory of Dr. Carl Nathan at Weill Cornell Medicine in New York, USA, as a postdoctoral fellow (2009-2014), an instructor (2014-2017) and an assistant professor (2017-2018) before he established his independent laboratory at Imperial College London (2019-2023). He was recruited to Inserm in January 2024 at the Research Institute of Digestive Health, where he is studying antibiotic tolerance in mycobacteria and Gram negative bacteria using a combination of systems biology, genetics and molecular microbiology.

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Sheetal Gandotra

Senior Principal Scientist at CSIR-Institute of Genomics and Integrative Biology (CSIR-IGIB), New Delhi, India

Sheetal Gandotra is a Senior Principal Scientist at the CSIR-Institute of Genomics and Integrative Biology, New Delhi. Her group works to understand the role of lipid metabolism during *Mycobacterium tuberculosis* infection. Using a multipronged approach of cell and molecular biology, genomics and biochemistry, her group studies the role of lipid droplet lipids and proteins in the innate immune defense against mycobacteria as well as lipid remodeling pathways of mycobacteria that help it counter host defense mechanisms.

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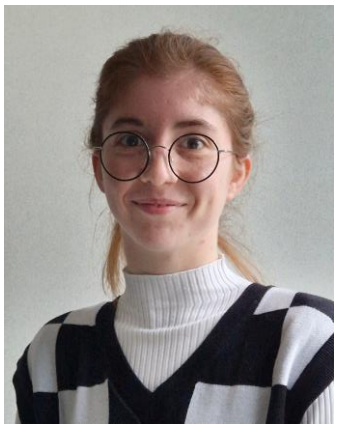

Aélia Farge

Sciences Po Intern in
International Cooperation service
at IPBS and CBI

With the support of:

- Administration and Financial Management service of IPBS:
    - Mélanie Bégué
    - Marcelle Borro-Cosson
    - Julien Brouzes
    - Elodie Gayral

- « Mycobacterial Envelopes and Therapeutic Targets » team of IPBS:
    - Martin Campoy, PhD student
    - Patricia Constant, Research Engineer
    - Chloé Garcia Labaig, PhD student
    - Gwenaëlle Jézéquel, Postdoctoral fellow
    - Hedia Marrakchi, CNRS Research Director
    - Leïla Mefoum, Research engineer
    - Rathies Ravindra, PhD student

- « Transformation of pneumococcus » team of LMGM-CBI:
    - Abel Abli Essowereou, PhD student
    - Anne Boyeldieu, Postdoctoral fellow
    - Nathalie Campo, CNRS Researcher
    - Calum Johnston, Researcher

Sponsors of the event

CEFIPRA - Indo-French Centre for the Promotion of Advanced Research

Title sponsor

Indo-French Centre for the Promotion of Advanced Research (IFCPAR/CEFIPRA) is a model for international collaborative research in advanced areas of Science & Technology. The Centre was established in 1987 and is being supported by the Department of Science & Technology, Government of India and the Ministry for Europe & Foreign Affairs, Government of France. CEFIPRA is actively involved in supporting Indo-French Science, Technology & Innovation (ST&I) system and promoting collaborative research through various activities.

Website: https://www.cefipra.org/Default.aspx

CNRS - National Centre for Scientific Research

Title sponsor

The mission of the National Centre for Scientific Research (CNRS) is to leverage all fields of sciences to tackle current global challenges. The CNRS has over 80 years of experience in basic research, exploring living creatures, space, materials and human societies and can leverage all fields of science to understand current global challenges in all their complexity, in conjunction with organisations in the field.

Website: https://www.cnrs.fr/en

Occitanie Region

Platinum sponsor

Responsible for education, professional training and career orientation, the Region is behind innovative initiatives to ensure its 226,000 high school students, 34,000 apprentices and 270,000 students are able to achieve success. Our aim is to guarantee equality of opportunity and optimum educational conditions, while increasing access to training and employment and getting young people involved in regional policies. The Region supports young people by enhancing the diversity and quality of post-high school options on offer. Regional aid also comes in the form of support for research institutions, in order to boost economic development and equality of access to higher education, spreading innovation through every local district, and promoting and enhancing public research Key Facts: 19,000 students trained for jobs in the aeronautics and space sector per year, 15 competitive clusters, 106 schools offering health and social care training, 3,400 receiving bursaries in the health and social care sector, €5.558 billion invested in research and development, and over 29,400 researchers.

Website: https://www.laregion.fr/

ANTABIO

Silver sponsor

Antabio is a privately-held clinical-stage biopharmaceutical company developing novel and highly differentiated antibacterial treatments for drug-resistant infections due to CDC and WHO critical priority pathogens. The company has a particular focus on life-threatening respiratory infections, including carbapenem-resistant nosocomial pneumonia and chronic pulmonary diseases. Antabio's lead program, MEM-ANT3310, is being developed for the treatment of hospital-acquired infections such as nosocomial pneumonia caused by carbapenem-resistant Acinetobacter baumannii (CRAB) and carbapenem-resistant Enterobacterales (CRE).

Website: https://antabio.com/

University of Toulouse

Silver sponsor

Our university is named after Paul Sabatier, a French scientist born in Carcassonne in 1854. Paul Sabatier won the Nobel Prize in Chemistry in 1912. He was Dean of the Faculty of Sciences in Toulouse and a member of the Academy of Sciences. The Université Toulouse III – Paul Sabatier has more than 37,000 students and 68 research facilities on its campus. Université Toulouse III – Paul Sabatier has a history dating back to the 13th century and is among some of the oldest universities in the world. It was officially founded in 1969 by a merger between the faculties of Medicine, Pharmaceuticals and Science. Its wide range of laboratories and high quality training courses in the fields of science, health, sport, technology and engineering have earned it the reputation of being one of the world's leading scientific universities for over 50 years now. It is ranked among the top 300 institutions for its scientific performance in the Academic Ranking of World Universities (ARWU ranking), also known as the Shanghai Ranking.

Website: https://www.univ-tlse3.fr/home

SFR-B2S - Federative Research Structure «Biology and Biotechnology for Health»

Silver sponsor

The Federative Research Structure « Biology and Biotechnology for Health » (SFR-B2S) was created in 2023 following the merger of the Toulouse Biomedical Research Federative Structure (SFR-BMT, FED 4138) and the Toulouse Biology Research Federation (SFR-BT, FR 3451). In addition, as part of its unit project, the Toulouse Biotechnology Institute (TBI) has decided to join this new federation, bringing biotechnology within its scope. This federative structure plays an active role in the coordinated dissemination of knowledge and information between the various research centres, in order to maintain a centre of excellence in the field of biology and biotechnologies for health. It is also a driving force behind Génotoul and the BABS cluster at Toulouse III – Paul Sabatier University. By bringing together 15 research institutes of the Toulouse site, its mission is not only to encourage exchanges and interactions between the constituent units, but also to give greater national and international visibility to the high-level research carried out in these units.

Website: https://bio-sante-toulouse.fr/

French Embassy in India

Silver sponsor

The French Embassy in India, based in New Delhi, plays a key role in strengthening bilateral relations between France and India across political, economic, scientific, educational, and cultural fields. As part of its mission to foster French-Indian collaboration, the Embassy actively supports initiatives that promote scientific and academic cooperation between institutions, researchers and experts from both countries. It especially encourages joint projects that contribute to research and innovation in the face of today's global challenges.

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Website: https://in.ambafrance.org/

IPBS - Institute of Pharmacology and Structural Biology

Bronze sponsor

The IPBS hosts more than 250 scientific and administrative staff, including more than 60 PhD students and postdoctoral fellows of multiple nationalities, who work in a stimulating and highly collaborative environment. The IPBS currently comprises 18 research groups working in two broad research areas: the biology of tissue and cellular microenvironments, and the molecular and structural mechanisms of disease. Four facilities provide state-of-the-art technology in proteomics, biophysics and structural biology, molecular and cellular imaging, and functional exploration. Several BSL-3 laboratories and animal facilities are available for the study of infectious diseases such as tuberculosis and other pulmonary and enteric infections, and more recently COVID-19.

ipbs TOULOUSE CANCER INFECTION INFLAMMATION

Website: https://www.ipbs.fr/

CBI – Centre for Integrative Biology

Bronze sponsor

Understanding the functioning of living organisms is the ambition of the Centre for Integrative Biology (CBI) in Toulouse. To achieve this goal, the CBI develops multidisciplinary, multi-scale approaches, from isolated molecules to whole organisms and animal societies, and uses numerous model organisms, from bacteria to humans. At its origin in 2016, the Federation of Research in Fundamental Biology was created under the supervision of the National Centre for Scientific Research (CNRS) and the University Toulouse III-Paul Sabatier (UPS). Today, the CBI brings together more than 400 people, working in 38 research groups, in three laboratories: Microbiology (LMGM), Molecular, Cellular and Developmental Biology (MCD), and Animal Cognition (CRCA). CBI groups, platforms and services are composed of researchers, faculty members, PhD and post-doctoral students, administrative and technical staff, not only from the CNRS, but also from the University and INSERM, making it one of the most important scientific research centres in France.

Website: https://cbi-toulouse.fr/eng/

LMGM-CBI – Laboratoire de Microbiologie et Génétique moléculaire

Bronze sponsor

The Laboratoire de Microbiologie et Genetique Moleculaires (LMGM) is part of the Center for Integrative Biology in Toulouse (CBI Toulouse). We study the organization, evolution and expression of the genomes of bacteria.

Website: https://lmgm.cbi-toulouse.fr/en/home/

Partners of the event

Access information

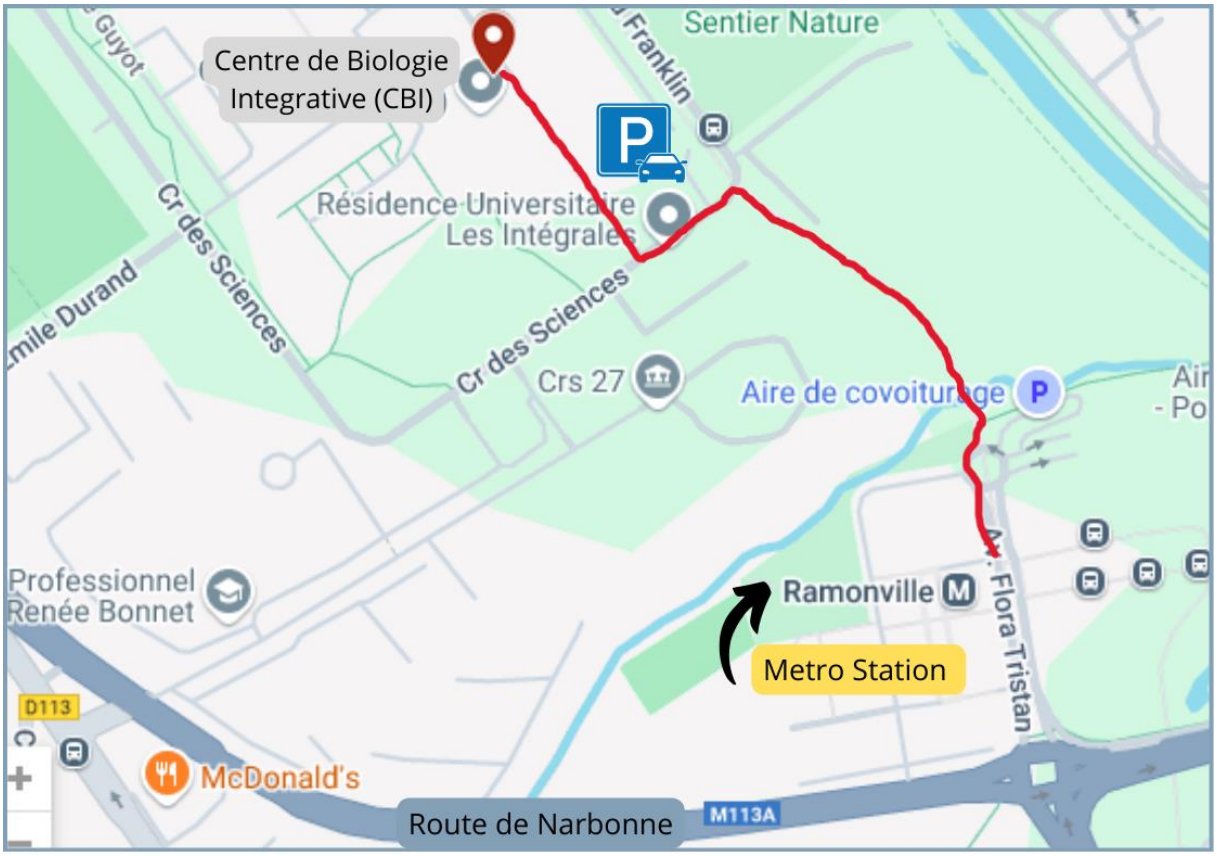
Location

CENTRE DE BIOLOGIE INTEGRATIVE / CENTRE FOR INTEGRATIVE BIOLOGY

Campus Université de Toulouse
169 rue Marianne Grunberg-Manago
31062 Toulouse

Access from metro station

Station Ramonville – Line B



MIRA Workshop

Program

Day 1
26 May 2025

Zoom link:
https://cnrs.zoom.us/j/98609161954?pwd=sU5VzVQJOMdYY4FSxjrAH84d5oUhOe.1

Amphitheatre Nicole Le Douarin and Reception Hall - CBI

Morning: Indian delegates reception – *Upon invitation*

12:00 – 1:30 pm: Lunch with Indian delegates – *Upon invitation*

1:30 – 2:00 pm: Participant reception / Poster installation

2:00 – 2:30 pm: Welcoming speeches

2:30 – 3:30 pm: Opening keynote lecture "The global AMR research landscape" by Prof. Marie-Cécile Ploy, Head of the Bacteriology-Virology-Hygiene Department of Limoges Hospital (France) and Coordinator of the European Joint Action on Antimicrobial Resistance 2024-2027 (EU-JAMRAI 2)

3:30 – 3:45 pm: Coffee break

3:45 – 5:30 pm: Scientific session 1 "Phenotypic AMR/Antibiotic tolerance", chaired by Anjana Badrinarayanan (NCBS, India) and Pierre Genevaux (LMGM-CBI, France)

- Amit Singh (IISc, India)
  Reprogramming mitochondrial bioenergetics to target drug tolerance in *Mycobacterium tuberculosis*
- Julien Vaubourgeix (IRSD, France)
  Multiform AMR in mycobacteria
- Hélène Botella (IPBS, France)
  Use of genetic engineering for forward selection of high survival mutants of *Mycobacterium tuberculosis in vitro* and in host environments
- Matthieu Chavent (LMGM-CBI, France)
  Modelling host-pathogen interactions using multiscale MD simulations: the *Mycobacterium tuberculosis* case
- Asha Joseph (NCBS, India)
  Toggling the replisome: ensuring integrity of the genome across contexts
- Dhiraj Kumar (ICGEB, India)
  Host-pathogen crosstalk at the innate immune driveway

5:30 – 7:00 pm: Social event and poster session

7:30 pm: Dinner of IRN MIRA partners – *Upon invitation*

Day 2
27 May 2025

Zoom link:
https://cnrs.zoom.us/j/98609161954?pwd=sU5VzVQJOMdYY4FSxjrAH84d5oUhOe.1

Amphitheatre Nicole Le Douarin and Reception Hall - CBI

8:30 – 8:45 am: Participant reception

8:45 – 8:55 am: Introduction of Day 2

8:55 – 10:15 am: Scientific session 2 "Surveillance of infectious diseases and antimicrobial resistance: emergence and evolution", chaired by Nathalie Campo (LMGM-CBI, France) and Amit Singh (IISc, India)

- Vinay K. Nandicoori (CCMB, India)
  Evolution of drug resistance *in Mycobacterium tuberculosis*
- Samay Pande (IISc, India) & Olaya Rendueles-García (LMGM-CBI, France)
  Bacterial predators drive the maintenance of antibiotic resistance
- Romane Dusfour-Castan (LMGM-CBI, France)
  Caenorhabditis elegans as a model organism to study the emergence of antimicrobial resistance
- Audrey Goman (IRSD, France)
  War Among Microbes: A Strategy for Antibiotic Discovery?
- Bhupesh Taneja (CSIR-IGIB, India)
  A pathogenomics surveillance unveils widespread AMR in fungal dermatophytic infections in India

10:15 – 10:30 am: Coffee break

10:30 – 12:30 nn: Scientific session 3 "Novel targets for novel antimicrobials", chaired by Hedia Marrakchi (IPBS, France) and Vinay K. Nandicoori (CCMB, India)

- Ranjana Pathania (IIT Roorkee, India)
  Revisiting bacterial pathophysiological fitness determinants as potential drug targets
- Ramandeep Singh (BRIC-THSTI, India)
  Identification of novel therapeutic interventions against *Mycobacterium tuberculosis*
- Anne Boyeldieu (LMGM-CBI, France)
  Deciphering the mode of action of the RumC1 bacteriocin in *Streptococcus pneumoniae*
- Martin Campoy (IPBS, France)
  Structural and enzymatic characterization of Baeyer-Villiger Monooxygenases from *Mycobacterium tuberculosis*: Insights into prodrug activation and structural features
- Harinath Chakrapani (IISER, India)
  Towards identifying new druggable targets through chemoproteomics
- Arunava Dasgupta (CSIR-CDRI, India)
  Discovering new drugs to combat old bugs: a quest for new arsenal against mycobacteria
- Sheetal Gandotra (CSIR-IGIB, India)
  Phospholipid remodelling in mycobacteria

- Xibing Xu (LMGM-CBI, France)
  Nucleotidyltransferase toxins MenT extend tRNA 3'-end to control *Mycobacterium tuberculosis* growth

12:30 nn: Group photo

12:30 – 2:00 pm: Buffet lunch

2:00 – 3:00 pm: Round table on India-France cooperation moderated by Isabelle Saves (IPBS and CBI, France)

- Parvinder Kaur (FNDR, India)
- Marc Lemonnier (ANTABIO, France)
- Leelavati Narlikar (IISER, India)
- Olivier Neyrolles (IPBS, France)
- Marie-Cécile Ploy (University of Limoges, France)
- Didier Raboisson (ENVT, France)
- Amit Singh (IISc, India)

3:00 – 4:00 pm: Coffee break and poster session

4:00 – 6:00 pm: Scientific session 4 "Alternative therapies, host-directed and anti-virulence therapies", chaired by Sheetal Gandotra (CSIR-IGIB, India) and Dhiraj Kumar (ICGEB, India)

- Vivek Rao (CSIR-IGIB, India)
  Importance of host cell metabolism in tuberculosis control - Cues for the development of novel host directed therapies
- Christel Verollet (IPBS, France)
  Deciphering mechanisms of cell-to-cell transfer of HIV-1 towards macrophages and their modulation by *Mycobacterium tuberculosis*
- Rachit Agarwal (IISc, India) – Online presentation
  Engineering approaches to tackle lung infections: bacteriophages and inhalable carriers
- Saurabh Chugh (IPBS, France)
  Exometabolome insights: A new perspective on *Mycobacterium tuberculosis* virulence
- Ashwani Kumar (CSIR-IMTech, India)
  Mycobacterial biofilms; Emerging hypothesis of *in vivo* drug tolerance and resistance
- Debapriya Mukherjee (IISc, India)
  Critical concentration of formate determines the susceptibility of *Salmonella* Typhimurium to meropenem and ciprofloxacin
- Olivier Neyrolles (IPBS, France)
  Sulfur metabolism in *Mycobacterium tuberculosis* response to stress
- Albertus Viljoen (IPBS, France)
  Surface organization of mycobacterial envelope glycans: Implications for immune interactions

6:00 – 6:30 pm: Poster session

7:30 – 10:00 pm: Boat trip and cocktail dinner on the *Canal du Midi – Meeting point: Port Saint-Sauveur, 31000 Toulouse – All registered participants*

Day 3
28 May 2025

Zoom link:
https://cnrs.zoom.us/j/98609161954?pwd=sU5VzVQJOMdYY4FSxjrAH84d5oUhOe.1

Amphitheatre Nicole Le Douarin and Reception Hall - CBI

8:30 – 8:45 am: Participant reception

8:45 – 9:00 am: Introduction of Day 3

9 :00 – 11 :00 am : Scientific session 5 "From early discovery to clinical trial: tricks and opportunities", chaired by Arunava Dasgupta (CSIR-CDRI, India) and Olivier Neyrolles (IPBS, France)

- Sidharth Chopra (CSIR-CDRI, India) – Online presentation
  Discovering drugs for bad bugs: Progress amongst challenges!
- Lionel Mourey (IPBS, France)
  Targeting an old target: Is mycobacterial InhA suitable for rational drug design?
- Parvinder Kaur (FNDR, India)
  Humanised 3-D granuloma predicting reactivation of tuberculosis
- Hugo Lebrette (LMGM-CBI, France)
  Structural dynamics of radical enzymes and metalloenzymes by femtosecond crystallography
- Hedia Marrakchi (IPBS, France)
  Mycobacterial cell envelope as an inspiring target for anti-tuberculosis drug discovery
- Avik Pathak (IIT Roorkee, India)
  Deciphering the role of cysteine biosynthesis pathway in the pathophysiology and virulence of *Acinetobacter baumannii*
- Maxime Pingret (IPBS, France)
  Exploring the role of a novel β3-Tubulin expressing cell population in *Mycobacterium tuberculosis* pathogenesis
- Vijaya Vaishnavi (IISc, India)
  Tuberculosis granuloma organoids for studying host-pathogen interactions

11:00 – 11:15 am: Coffee break

11:15 – 12:15 nn: Closing keynote lecture "Towards antibiotic rescue by identifying and targeting emergent vulnerabilities in drug resistant bacteria" by Prof. Nagasuma Chandra, Professor in the Biochemistry department of the Indian Institute of Science of Bangalore (India)

12:15 – 12:30 nn: Closing remarks

12:30 – 2:00 pm: Lunch – *Upon invitation*

Afternoon: Sightseeing tour in Carcassonne – *Indian delegates and organisers upon invitation*

Keynote lectures

Marie-Cécile Ploy

Professor of Microbiology at the Faculty of Medicine and Limoges Teaching Hospital, Limoges University, France

The global AMR research landscape

Faced with the global threat of antibiotic resistance, the European Union, through its different actions, is demonstrating its commitment to a coordinated and concrete response. The ambitions of European initiatives point to a future where European countries actively exchange best practices in the fight against antibiotic resistance, where healthcare professionals act to ever higher standards, and where citizens are made aware of the importance of preserving the efficacy of antibiotics. By adopting a global, coordinated approach, Europe is putting the "One Health" approach into practice.

Marie-Cecile Ploy, PharmD, PhD, is a Professor of Microbiology at the Faculty of Medicine and Limoges Teaching Hospital, Limoges University, France. She is Head of the Bacteriology-Virology-Hygiene Department at the Limoges Teaching Hospital and director of the Inserm RESINFIT research unit on antimicrobials at the Limoges University (https://www.unilim.fr/resinfit/). She gained her PharmD in 1994 and her PhD on aminoglycoside resistance in 2000 (Institut Pasteur and Paris XI University). She had a postdoctoral position (2003-2004) in Didier Mazel'lab at the Institut Pasteur in Paris, France, where she focused her research on integrons.

She is part of numerous Committees on Antimicrobial resistance at the national level. She was the coordinator of the European Joint Action an Antimicrobial resistance and healthcare-associated infections (www.eu-jamrai.eu) from 2017 to 2021; and she coordinates the second Joint action, EU-JAMRAI 2, from 2024 to 2027.

She is an expert in numerous national and international research programmes on antimicrobial resistance. She is a member of the Management Board of the JPIAMR and vice-chair of the JPIAMR steering committee. She is vice-dean for research at the Faculty of Medicine, Limoges University. She is full member of the French Academy for Veterinary Medicine

Her research addresses the mechanisms and dynamics of mobilization and spread of antimicrobial resistance. Her main research topics are I) the role of the SOS response in antibiotic resistance acquisition and expression, and II) the risk assessment of the antibiotic resistance dissemination in the environment.

Website: https://www.unilim.fr/resinfit

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Publications related to the presentation

- GBD 2021 Antimicrobial Resistance Collaborators. Global burden of bacterial antimicrobial resistance 1990-2021: a systematic analysis with forecasts to 2050. Lancet. 2024 Sep 28;404(10459):1199-1226

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- https://eu-jamrai.eu/

Closing keynote lecture

Nagasuma Chandra

Professor in the Biochemistry Department of the
Indian Institute of Science (IISc), Bangalore, India

Towards antibiotic rescue by identifying and targeting emergent vulnerabilities in drug resistant bacteria

The emergence of drug-resistant bacteria poses a major threat to public health, warranting urgent attention to the problem of tackling antimicrobial resistance (AMR). A number of studies have sought to understand the causative molecular mechanisms such as mutations in key targets, upregulation of drug efflux pumps and drug or target-modifying enzymes. Yet, there is no clear understanding of which mechanisms are operative in a given condition, whether microbes explore multiple mechanisms simultaneously, or if such mechanisms are influenced by each other and lead to alterations in the cell in a synchronized manner. Towards this, an understanding of the global mechanisms leading to resistance becomes necessary. In this talk, I will describe a multi-pronged approach that have been using to tackle AMR. Specifically, I will describe our efforts to get a comprehensive view of the molecular perturbations associated with drug resistance by integrating multi-omic data and network models in *M.tuberculosis*, *E.coli* and *S.aureus*, which have lead to identification of the Achille's heel in each case and thereby the identification of strategic co-targets that can rescue drug sensitivity. Our findings reveal that multiple mechanisms operate simultaneously, requiring a genome-wide systems view to address them. I will also describe our work on the computational design of an antimicrobial peptide using structural models and a new machine learning algorithm that we developed, leading to Omega76, that showed high efficacy against tigecycline and colistin-resistant *Acinetobacter baumannii*.

Nagasuma Chandra is a Professor at the Department of Biochemistry and additionally affiliated with Bioengineering and Mathematical biology initiatives at the Indian Institute of Science. She established the area of molecular systems biology and contributed significantly to the development of bioinformatics and genomic medicine in the Institute. Her research is interdisciplinary involving computational modelling of complex biological processes to address fundamental questions about how genome-wide molecular networks respond to a variety of pathological conditions and translating that knowledge into biomedical applications. She is an elected fellow of the Indian Academy of Sciences and the Indian National Science Academy. Nagasuma Chandra obtained her PhD from the University of Bristol, UK. She has over 175 publications in international journals. She has co-founded a precision medicine company, HealthSeq Precision Medicine, Pvt Ltd, incubated at IISc.

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Publications related to the presentation

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Scientific session 1

Chair

Anjana Badrinarayanan

Associate Professor at National Centre for Biological Sciences (NCBS), Bangalore, India

Anjana's lab employs quantitative live-cell imaging approaches to track, in real-time, the dynamics of single cells and molecules within cells. With this they address frontier and fundamental questions about the regulation of genome duplication and DNA damage repair in microbial systems. Their work has provided new mechanistic insights into the general principles of genome maintenance and its impact on microbial adaptation and survival.

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Chair

Pierre Genevaux

CNRS Group Leader at Laboratoire de Microbiologie
et Génétique Moléculaires (LMGM-CBI), Toulouse, France

Pierre Genevaux performed a PhD in molecular genetics during which he identified key genes involved in biofilm formation in bacteria. He then worked as a postdoc in Geneva performing research on molecular chaperones in bacteria, mostly contributing to the discovery of proteostasis networks. Based on this research, he obtained a CNRS position along with an ATIP young researcher grant to start a team in Toulouse. The main goal of the work currently performed in his group is to shed light on cellular mechanisms that respond to stress in bacteria, especially through the activation protein quality control networks and stress-responsive Toxin-Antitoxin (TA) Systems. Throughout the years, the team has revealed intricate connections between molecular chaperones and TA systems in *M. tuberculosis* and discovered several new TA systems and toxic mechanisms.

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Speaker

Hélène Botella

Assistant Professor at Institute of Pharmacology
and Structural Biology (IPBS), Toulouse, France

Use of genetic engineering for forward selection of high survival mutants of *Mycobacterium tuberculosis in vitro* and in host environments

Bacterial survival to antimicrobials can manifest as either bacterial growth in the presence of an antimicrobial (so-called antimicrobial resistance) or a decreased rate of bacterial death upon exposure to antibiotics (tolerance, persistence and high persistence referred collectively as antibiotic recalcitrance). Antibiotic recalcitrance complicates treatment of many bacterial infections, including tuberculosis. They contribute to treatment duration, treatment failure, disease recurrence, and the emergence of resistance. Therapeutically targeting tolerant and persistent cells could improve outcomes, but the molecular mechanisms underlying tolerance and persistence in bacteria are not well understood, in particular in the context of the stresses imposed by host immunity. To fill this gap, we developed ReMIND— REcombination-Mediated Isolation of Non-Dividers—a unique method developed to enrich and select tolerant and high persistent mutants from resistant mutants in liquid medium and in complex host environments.

Hélène Botella started working on mycobacteria during her PhD in the laboratory of Dr Olivier Neyrolles at the IPBS in Toulouse, France. In 2012, she joined the laboratory of Dr Sabine Ehrt at Weill Cornell Medicine in New York as a postdoctoral fellow. Both during her PhD and her postdoctoral work, she studied various aspects of the mechanisms by which *Mycobacterium tuberculosis* escapes host clearance despite the defense mechanisms deployed by immune cells. In 2019, Hélène was granted a Marie Sklodowska-Curie individual fellowship to work in the laboratory of Dr Julien Vaubourgeix at Imperial College in London where her work focuses on antibiotic tolerance in mycobacteria. In 2023, she was recruited as an assistant professor at the University of Toulouse and joined the laboratory of Dr Christophe Guilhot at IPBS to continue the line of research she initiated in London.

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Publications related to the presentation

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Speaker

Matthieu Chavent

CNRS Group Leader at Laboratoire de Microbiologie et Génétique Moléculaires (LMGM-CBI), Toulouse, France

Modelling host-pathogen interactions using multiscale MD simulations: the *Mycobacterium tuberculosis* case

Mycobacterium tuberculosis, the pathogen responsible for the infectious disease tuberculosis, is known for its thick and waxy envelope constituted of numerous complex lipids (1). These lipids act both as building blocks to organize the envelope (2) and as virulence factors that destabilize the host membrane (3) or interact with host-cell receptors (4). I will present our recent work on modeling mycobacterial lipids and on their organization in the mycobacterial membranes.

Chavent's team research is at the cross-road between computer science, biophysics, and structural biology. The scientific goal of the team is to understand membrane protein interactions with lipids using multiscale MD simulations. For several years, the research of the team focus on understanding host-pathogens interactions, especially the biophysical properties of complex lipids constituting the envelope of *Mycobacterium tuberculosis*.

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Publications related to the presentation

- (1) T. Brown, **M. Chavent**, W. Im, Molecular Modeling and Simulation of the Mycobacterial Cell Envelope: From Individual Components to Cell Envelope Assemblies. *J. Phys. Chem. B* (2023). DOI:https://doi.org/10.1021/acs.jpcb.3c06136

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Speaker

Asha Joseph

Postdoctoral Project Scientist at National Centre for Biological Sciences (NCBS), Bangalore, India

Toggling the replisome: ensuring integrity of the genome across contexts

Maintenance and propagation of life relies on the ability to duplicate cell's genome, a process mediated by the replication machinery or the replisome. In addition to it's core function of synthesizing DNA, the replisome is also responsible for spontaneous and stress-induced mutagenesis, thus contributing to generation of genetic diversity and development of antibiotic resistance in bacteria. Using live-cell imaging strategies to directly visualize replisome in *Caulobacter*, we assessed the activity of replisome under various contexts of bacterial growth, Our observations demonstrate how different contexts of growth lead to contrasting implications on bacterial stress response and mutagenesis.

Asha is a research scientist in Anjana Badrinarayanan's group at National Centre for Biological Sciences, Bengaluru. She earned her PhD from Indian Institute of Science where she investigated the evolution of novel metabolic functions in the pathogenic bacterium Shigella under nutrient stress. By combining classical genetics and microbiology approches with quantitative live-cell microscopy, Asha currently studies the regulation of bacterial genome replication and repair in order to decipher their relevance in bacterial survival and mutagenesis.

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Publications related to the presentation

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Speaker

Dhiraj Kumar

Group Leader at International Centre for Genetic Engineering and Biotechnology (ICGEB), New Delhi, India

Host-pathogen crosstalk at the innate immune driveway

Tuberculosis (TB) is a serious health challenge, with BCG as the only available vaccine and the problem of drug resistance against exisiting anti-TB drugs. *Mycobacterium tuberculosis* (*Mtb*), the pathogen that cause TB in humans, is well-adapted to the host environment. This allows them to alter and evade the host immune responses. My group explores the innate immune arm of the host immune system and how the interactions with the *Mtb* alter the innate immune pathways. The pathways studeid include macro events like cellular recruitment, organization and disease lesion composition ; followed by sub-cellular pathways like trafficking, signaling and metabolism and molecular events like transcription, alternative splicing and post-transcriptional gene regulation. The general understanding is that *Mtb* has evolved intricate mechanisms to specifically target each arm of the innate immune system, allowing it efficiently establish infection, cause disease and also, evade any therapeutic interventions. Understanding molecular regulators of these processes allow novel framework for targeting TB and associated pathologies. To that effect, we have explored host responses in macrophages, mesenchymal stem cells and neutrophils using *ex vivo* and *in vivo* models. Our group strive to develop host-directed approaches against TB with an aim to enhance the efficacy of prevention and therapy.

Dhiraj Kumar is a Group Leader at the International Centre for Genetic Engineering and Biotechnology, New Delhi, where he leads the Cellular Immunology Group. The research focus of his group is to explore diNerent facets of innate immunity during tuberculosis pathogenesis. The research topic includes regulation of autophagy, immune cell infiltration, cell detah and inflammation. The aim is to harness the innate immune arm to develop novel host-directed preventive and therapeutic approaches against TB.

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Publications related to the presentation

- Sharma, P., Sharma, R.D., Das, M., Sarkar, B., Singh, L., Jain, N., Chaturvedi, S., Mehra, L., Rathee, A., Sharma, S., Foo, S., Lee, A., kumar, NP., Das, P., Viswanathan, V., Kornfeld, H., Howland, S.W, Babu, S., Nandicoori, V., Singhal, A., **Kumar, D**. (2024) Ly6G+Granulocytes-derived IL-17 limits protective host responses and promotes tuberculosis pathogenesis. *eLife*, 13:RP100966, DOI: https://doi.org/10.7554/eLife.100966.1

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Speaker

Amit Singh

Professor at Center for Infectious Disease Research
(CIDR), Indian Institute of Science (IISc), Bangalore, India

Reprogramming mitochondrial bioenergetics to target drug tolerance in *Mycobacterium tuberculosis*

Mycobacterium tuberculosis (*Mtb*) persists inside the phagocytic cells despite immune pressures and stresses caused by multiple anti-TB drugs. How *Mtb* tolerates immune and drug pressures and survives is fundamental in the TB field. We have developed a novel biosensor to image the redox physiology of *Mtb* inside the human host during infection and upon chemotherapy. Using this probe, we identified the role of phagosomal acidity and mitochondrial metabolism in promoting the emergence of a redox-altered subpopulation of *Mtb* that tolerates anti-TB drugs. Importantly, we provide empirical evidence linking intramycobacterial redox balance, mitochondrial bioenergetics, and Nrf2 signaling pathway in the emergence of drug tolerance during infection. Based on these innovative findings, we developed strategies to reset immuno-metabolism to improve the efficacy of anti-TB drugs during infection.

Amit Singh is a Professor of Microbiology and Cell Biology. Amit Singh's group exploited interdisciplinary strategies to dissect the redox basis of persistence in human pathogens *Mycobacterium tuberculosis* and HIV. By taking advantage of redox biosensors, XF-flux analyses, omics-based strategies, and animal studies, his work has helped find new mechanisms of how macrophage's acidic pH mobilizes drug tolerance in *M. tuberculosis*. In 2015, he was awarded the Senior DBT-Wellcome Trust India Alliance fellowship. Most recently, he was awarded the Shanti Swarup Bhatnagar Award, the most prestigious prize by the Council of Scientific and Industrial Research in India. He is a member of the National Academy of Sciences, India.

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Publications related to the presentation

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Speaker

Julien Vaubourgeix

Assistant Professor at Digestive Health
Research Institute (IRSD), Toulouse, France

Multiform AMR in mycobacteria

Antibiotic recalcitrance refers to a slower rate of death for either a bacterial population or a subpopulation of cells upon antibiotic exposure. It complicates treatment of many bacterial infections by contributing to treatment length, treatment failure, disease recurrence, and the emergence of antimicrobial resistance (AMR). Thus, blocking antibiotic recalcitrance could be a powerful strategy for improving treatment outcomes and reducing AMR rates. Here, using a forward genetic method for the isolation of antibiotic-recalcitrant mutants, we isolated two *Mycobacterium smegmatis* strains with mutations in the tRNA-modifying enzyme adenine-N(1)-methyltransferase. Both mutants are recalcitrant or resistant to proteostasis-perturbing antibiotics. We linked these phenotypes to upregulation of the transcriptional regulator WhiB7, highlighting its role as a point of convergence in the regulation of multiple mechanisms of antibiotic recalcitrance and resistance.

After completing his PhD in the laboratory of Dr. Mamadou Daffé at the Institute of Pharmacology and Structural Biology in 2009 in Toulouse, France, Julien joined the laboratory of Dr. Carl Nathan at Weill Cornell Medicine in New York, USA, as a postdoctoral fellow (2009-2014), an instructor (2014-2017) and an assistant professor (2017-2018) before he established his independent laboratory at Imperial College London (2019-2023). He was recruited to Inserm in January 2024 at the Research Institute of Digestive Health, where he is studying antibiotic tolerance in mycobacteria and Gram negative bacteria using a combination of systems biology, genetics and molecular microbiology.

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Publications related to the presentation

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Scientific session 2

Chair

Nathalie Campo

CNRS Researcher at Laboratoire de Microbiologie et Génétique Moléculaires (LMGM-CBI), Toulouse, France

Nathalie Campo is a CNRS researcher in the "Pneumococcal Competence and Transformation" team headed by Patrice Polard at the Laboratoire de Microbiologie et Génétique Moléculaires (Toulouse, France). Over the last 15 years, she has developed multidisciplinary approaches including the use of far- end imaging techniques at different levels of resolution to visualize and characterize the process of natural genetic transformation in the human pathogen *Streptococcus pneumoniae*.

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Chair

Amit Singh

Professor at Center for Infectious Disease Research (CIDR), Indian Institute of Science (IISc), Bangalore, India

Amit Singh is a Professor of Microbiology and Cell Biology. Amit Singh's group exploited interdisciplinary strategies to dissect the redox basis of persistence in human pathogens *Mycobacterium tuberculosis* and HIV. By taking advantage of redox biosensors, XF-flux analyses, omics-based strategies, and animal studies, his work has helped find new mechanisms of how macrophage's acidic pH mobilizes drug tolerance in *Mycobacterium tuberculosis*. In 2015, he was awarded the Senior DBT-Wellcome Trust India Alliance fellowship. Most recently, he was awarded the Shanti Swarup Bhatnagar Award, the most prestigious prize by the Council of Scientific and Industrial Research in India. He is a member of the National Academy of Sciences, India.

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Speaker

Romane Dusfour-Castan

PhD student at Laboratoire de Microbiologie et Génétique Moléculaires (LMGM-CBI), Toulouse, France

Caenorhabditis elegans as a model organism to study the emergence of antimicrobial resistance

Bacteria evolve rapidly, adapting to their environment. A key example is antibiotic-resistant bacteria, a global issue driven by antibiotic use since the 1940s. Their evolvability stems from mobile genetic elements (MGEs), which are associated with most clinically relevant antibiotic resistance genes (ARGs). MGEs include plasmids, transposons, and integrons, moving intracellularly (between chromosomes and plasmids) or intercellularly (within or across species) (Frost 2005).
Conjugative plasmids are the most active resistance gene disseminators, facilitating horizontal transfer of MGEs via recombination and transposition (Norman 2009). Transposable elements, including insertion sequences (IS) and transposons, not only transpose ARGs but also modulate their expression (Siguier et al., 2006).
Despite extensive knowledge of MGE propagation mechanisms (Cabezón et al., 2015; Guynet et al., 2020), gaps remain regarding in situ processes within natural bacterial communities such as microbiota. Most data come from metagenomic analyses across ecosystems, which lack spatio-temporal resolution.
To address this question, we are developing an approach using the nematode *Caenorhabditis elegans* and its gut microbiota to study MGE dynamics and antibiotic resistance emergence in a complex ecosystem. *C. elegans* harbors a complex gut microbiota, including groups of bacteria that are also found in the human gut microbiota (ex : Gammaproteobacteria and Bacteroidota) (Dirksen et al., 2016; Zhang et al., 2017). In our approach, we use a simplified gut microbiota, composed of twelve bacterial strains, sequenced, easily cultivable in the lab and capable of colonizing the worm gut (the CeMbio community) (Dirksen et al., 2020).
C. elegans is also a model of choice due to its ease of cultivation, short life cycle, and transparency under light microscopy, allowing direct *in vivo* visualization of bacteria and MGEs dynamics.

Sarah began her scientific pathway with an engineering degree in food science, specializing in microbiology at AgroSup Dijon (Dijon, France). During her studies, she explored academic research through two internships: one at the University of Queensland (Brisbane, Australia) on fermentation processes in frozen human fecal inocula, and the second one at the IFREMER and at the IRCGN (Brest and Cergy-Pontoise, France), on marine microorganisms as bio-indicators of sea drowning. Then, she pursued her scientific pathway by doing a PhD at the LMGM-CBI (Toulouse, France), on host-microbiota interactions using *Caenorhabditis elegans* and its gut microbiota as a model. Using the same model, her work also focused on horizontal gene transfer and antibiotic resistance emergence in complex bacterial communities.

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Publications related to the presentation

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Speaker

Audrey Goman

PhD student at Digestive Health
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War Among Microbes: A Strategy for Antibiotic Discovery?

Polymicrobial infections are shaped by interbacterial competition, often mediated by secreted molecules. In this study, we describe a novel antibacterial mechanism involving lipids derived from bacterial extracellular vesicles (bEVs). bEVs produced by Escherichia coli exhibit potent antibacterial activity against Gram-positive species. Our findings reveal a new strategy of bacterial antagonism and suggest that bEVs play an underappreciated role in shaping polymicrobial ecosystems.

Audrey is currently in the fourth year of her doctoral studies under the supervision of Professor Oswald at the Digestive Health Research Institute (IRSD) in Toulouse, France. Her research is primarily focused on elucidating the virulence mechanisms employed by bacteria. Her research interests are centered on the study of outer membrane vesicles (OMVs) produced by Gram-negative bacteria, particularly *E. coli* and *P. aeruginosa*. She employs bacterial genetic engineering techniques to explore the functionality of virulence factors and to investigate the role of OMVs in toxicity toward eukaryotic cells, their contribution to virulence in animal models, and their involvement in bacterial competition within microbial communities.

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Team 2: Pathogenesis and commensalism of enterobacteria (Bat B Bureau 303a)

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Publications related to the presentation

- **Goman, A.**, et al, (2025). Uncovering a new family of conserved virulence factors that promote the production of host-damaging outer membrane vesicles in Gram-negative bacteria. Journal of Extracellular Vesicles, 14, e70032. DOI: https://doi.org/10.1002/jev2.70032

Speaker

Vinay Kumar Nandicoori

Director at CSIR-Centre for Cellular and
Molecular Biology (CCMB), Hyderabad, India

Evolution of drug resistance in Mycobacterium tuberculosis

The emergence of drug resistance in *Mycobacterium* tuberculosis (*Mtb*) is alarming and demands in-depth knowledge for timely diagnosis. In a study published in *Plos Pathogens* (2021), we demonstrated that compromised repair in *Mtb* drives greater adaptability and provides a tool for facile identification of drug targets. Recently, we performed genome-wide association analysis using 2237 clinical strains of *Mtb* to identify novel genetic factors that evoke drug resistance. We showed that novel variant mutations in the DNA repair genes collectively compromise their functions and contribute to better survival under antibiotic/host stress conditions.

Dr. Vinay Nandicoori is a Molecular and Cellular Biologist who has contributed to delineating the signalling networks in *Mycobacterium tuberculosis* (*Mtb*). He did his Masters in Biotechnology at the Indian Institute of Technology, Bombay and his Ph.D. in Molecular and Cellular Biology from the Indian Institute of Science, Bangalore. He was a Postdoc at Texas A&M University for 3 years and the University of Virginia for three and half years before returning to India in 2004. In 2004, he joined National Institute of Immunology, New Delhi, as a Scientist. In 2021, he moved to the Centre for Cellular and Molecular Biology, Hyderabad, India as the Director. His research interest is to delineate the kinase-mediated signalling networks in *Mtb*. In addition, his lab is also interested in identifying novel drug-resistant mechanisms, deciphering transcription regulation and identifying novel targets for host-directed therapy.

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Publications related to the presentation

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Samay Pande

Assistant Professor at Indian Institute of Science (IISc), Bangalore, India

Olaya Rendueles-García

CNRS Group Leader at Laboratoire de Microbiologie et Génétique Moléculaires (LMGM-CBI), Toulouse, France

Bacterial predators drive the maintenance of antibiotic resistance

The effects of antibiotics on resistance evolution are well known, but the influence of microbial interactions on resistance within communities remains unclear. We demonstrate a correlation between the presence of the bacterial predator *Myxococcus xanthus* in soil and the abundance of antibiotic resistance. This occurs because cell death, a key aspect of the *M. xanthus* life cycle, releases diffusible growth-inhibitory molecules into the environment. These molecules, in turn, drive the enrichment of resistance in natural soil communities. Our work highlights the complex dynamics of resistance evolution, emphasizing the role of interspecies interactions in shaping resistance profiles in pristine environments. Building on these findings we now aim to study how microbial predators impact the transmission of resistance plasmids in pathogens like *Klebsiella pneumoniae*. Predator-induced stress has been shown to select for mucoid phenotypes in prey bacteria, which are often linked to increased capsule production. Since capsule expression modulates plasmid uptake and transfer, our ongoing work investigates how such interactions influence the ecology and evolution of antibiotic resistance.

Samay Pande is an Assistant Professor at The Indian Institute of Science. He did hs PhD at the Max Planck Institute for Chemical Ecology in Germany and Then joined ETH Zurich for Postdoctoral training. Among other topics, one of the interest of his laboratory is to understand how microbial interactions influence the evolution and maintenance of antibiotic resistance in natural microbial communities.

After completing a Masters in Microbiology in my hometown in the North of Spain, Oviedo, Olaya Rendueles-García did a PhD in the Genetics of Biofilms lab in the Institut Pasteur (Paris), supervised by Jean-Marc Ghigo. For her first postdoctoral training, she joined the Evolutionary Biology Lab led by Gregory J. Velicer in ETH Zürich, where she addressed the evolution of a social microbe and how such interactions could shape biodiversity and species evolution. She then joined a computational lab led by Eduardo Rocha in the Institut Pasteur, and took interest on the evolution of a major cellular structure and virulence factor, the extracellular bacterial capsule. At the beginning of 2024, she moved to the Centre of Integrative Biology (CBI), in Toulouse, to study the impact of the capsule on bacterial adaptation.

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- Saheli Saha, Jyotsna Kalathera, Thoniparambil Sunil Sumi, Vishwadeep Mane, Sina Zimmermann, Silvio Waschina, **Samay Pande**. Mass lysis of predatory bacteria drives the enrichment of antibiotic resistance in soil microbial communities. Current Biology, Volume 35, Issue 6, 2025, Pages 1258-1268.e6, ISSN 0960-9822 DOI: https://doi.org/10.1016/j.cub.2025.01.068

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Speaker

Bhupesh Taneja

Senior Principal Scientist at CSIR-Institute of Genomics and Integrative Biology (CSIR-IGIB), New Delhi, India

A pathogenomics surveillance unveils widespread AMR in fungal dermatophytic infections in India

Fungal infections are a growing public health concern worldwide. Although invasive fungal infections are usually associated with high proportion of mortality (especially in "at risk" population with underlying health problems), the largest infectious group worldwide accounting for >70% of all fungal diseases is the superficial infections of skin, hair and nail. These superficial infections, termed dermatophytosis, lead to significant morbidity, pain and a substantial economic burden on the healthcare system due to long-term therapy and several cases of recurrence. This health threat is further compounded by the recent worldwide emergence of drug resistance; with nearly 30% of estimated global load of resistance of dermatophytosis in India. Results from a recent exhaustive literature mining and phylogenomics analysis of dermatophytosis in India will be presented.

Dr. Bhupesh Taneja holds the position of Senior Principal Scientist at CSIR-Institute of Genomics and Integrative Biology and Professor at Academy of Scientific and Innovative Research. The overall focus of his lab is to consolidate the understanding of mechanisms of drug resistance in pathogens and to identify alternate ways to address these problems. Specializing in structural biology and genomics, his work integrates informatics and AI-based methods towards point-of-care diagnostics and quicker decision-making in AMR or drug resistance in clinical settings. The major focus areas of his lab are "Structural investigations of key cellular components involved in host-pathogen interactions" and "Antimicrobial resistance in bacteria and fungi" and has contributed significantly to these fields.

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Publications related to the presentation

- Kumar P, Ramachandran S, Das S, Bhattacharya SN, **Taneja B**. (2023). Insights into changing dermatophyte spectrum in India through analysis of cumulative 161,245 cases between 1939-2021. Mycopathologia. 2023; 188:183-202. DOI: https://doi.org/10.1007/s11046-023-00720-6

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Scientific session 3

Chair

Hedia Marrakchi

CNRS Research Director at Institute of Pharmacology and Structural Biology (IPBS), Toulouse, France

Hedia Marrakchi is a CNRS research director and head of the group "mycobacterial envelopes and therapeutic targets" at the IPBS. She received her PhD in Biochemistry from the University of Toulouse, working on mycobacterial fatty acid synthesis, then completed a postdoctoral training at St Jude Children's research Hospital, Memphis (USA) in the department of infectious diseases, before joining the CNRS as a research scientist.

Her research interests focus on bacterial fatty acid synthesis and regulation, mycobacterial lipid metabolic pathways and cell envelope biogenesis, structure-function characterization of potential drug targets, and exploring innovative molecules and strategies in the fight against antibiotic resistance in TB and mycobacterial infections.

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Chair

Vinay Kumar Nandicoori

Director at CSIR-Centre for Cellular and
Molecular Biology (CCMB), Hyderabad, India

Dr. Vinay Nandicoori is a Molecular and Cellular Biologist who has contributed to delineating the signalling networks in *Mycobacterium tuberculosis* (*Mtb*). He did his Masters in Biotechnology at the Indian Institute of Technology, Bombay and his Ph.D. in Molecular and Cellular Biology from the Indian Institute of Science, Bangalore. He was a Postdoc at Texas A&M University for 3 years and the University of Virginia for three and half years before returning to India in 2004. In 2004, he joined National Institute of Immunology, New Delhi, as a Scientist. In 2021, he moved to the Centre for Cellular and Molecular Biology, Hyderabad, India as the Director. His research interest is to delineate the kinase-mediated signalling networks in *Mtb*. In addition, his lab is also interested in identifying novel drug-resistant mechanisms, deciphering transcription regulation and identifying novel targets for host-directed therapy.

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Speaker

Anne Boyeldieu

Postdoctoral fellow at Laboratoire de Microbiologie et Génétique Moléculaires (LMGM-CBI), Toulouse, France

Deciphering the mode of action of the RumC1 bacteriocin in *Streptococcus pneumoniae*

The RumC1 bacteriocin is a Ribosomally Synthesized and Post-translationally Modified Peptide (RiPP) of the sactipeptide class, identified in the human gut symbiont *Ruminococcus gnavus* E1(1). RumC1 is characterized by its compact double hairpin structure, its remarkable stability properties at high temperatures and extreme pH and its proficient antibacterial activity against a broad spectrum of Gram-positive species, including several clinical pathogens (2)(3). RumC1 does not display pore-forming activity (2) unlike all sactipeptides functionally characterized so far, raising the question of how it intoxicates the cell. Here, we report a comprehensive molecular study of RumC1 mode of action. We conducted this analysis in *Streptococcus pneumoniae* by combining genetical, biochemical and cell biology approaches following two complementary research axes. First, we developed a genetic screen, based on the natural transformation ability of this bacterium, to generate and select mutants resistant to RumC1 toxicity with high efficiency. Strikingly, we found that all pneumococcal mutants able to grow in the presence of RumC1 carried point mutations in the same genetic cluster. Phenotypic characterization of these mutants coupled to analysis of the subcellular localization of RumC1 by microscopy provided important clues about the essential cellular function altered by RumC1.The second research axis was based on the functional characterization in *S. pneumoniae* of the natural immunity system encoded within the RumC1 biosynthetic cluster. This analysis further demonstrated how RumC1 interferes with cell integrity and revealed an unprecedented mechanism of protection not only against RumC1 but also against an unrelated antimicrobial peptide. In all, these findings underly a novel mode of action for a bacteriocin and raise important perspectives in the frame of RumC1 potential application in the ever-growing medical issue of antibioresistance.

Anne Boyeldieu is a postdoc in the "Pneumococcal Competence and Transformation" team headed by Patrice Polard at the Laboratoire de Microbiologie et Génétique Moléculaires (Toulouse, France). During her PhD, she studied the regulatory networks involved in chemotaxis and biofilm formation in the aquatic bacterium Shewanella oneidensis. Over the past 4 years, she has worked on characterizing the mode of action of the promising bacteriocin RumC1.

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Publications related to the presentation

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Speaker

Martin Campoy

PhD student at Institute of Pharmacology
and Structural Biology (IPBS), Toulouse, France

Structural and enzymatic characterization of Baeyer-Villiger Monooxygenases from *Mycobacterium tuberculosis*: Insights into prodrug activation and structural features

Tuberculosis (TB) remains a global health threat, with *Mycobacterium tuberculosis* (*Mtb*) being the leading cause of death from a single infectious agent. The rise of antibiotic resistance underscores the urgent need for new TB treatments. Several anti-TB drugs are prodrugs requiring activation by specific *Mtb* enzymes. Among these, Baeyer-Villiger MonoOxygenase (BVMO) activate ethionamide and thiacetazone. However, in vitro research on these enzymes is hampered by protein production and purification challenges, while the precise enzymatic and structural characteristics of these BVMOs remain poorly understood. We investigated structural and enzymatic features using various biophysical approaches including mass photometry, dynamic light scattering and in vitro enzymatic assays on various substrates and prodrugs. Optimization of the expression and purification conditions for BVMOs resulted in improved purification yields, providing highly pure protein suitable for enzymatic and structural studies. Using an optimized in vitro enzymatic assay, we demonstrated the selectivity of the purified BVMO for linear ketone over commercial prodrugs. We also investigated the selectivity of commercial prodrugs versus a library of ethionamide analogues, confirming the findings through in silico docking on AlphaFold 3 models in both apo form and in complexes with coenzymes. The purity of the samples enabled the initiation of structural studies while applying biophysical techniques improved sample homogeneity by dissociating multimeric species. Negative staining electron microscopy experiments revealed a promising conditions for cryogenic electron microscopy data acquisition to solve the 3D structure of BVMO.

Contributors: Martin Campoy, Dimitri Leonelli, Anne Lemassu, Lionel Mourey, Mamadou Daffé, Valérie Guillet, Hedia Marrakchi

Martin is a PhD student in structural biology, specializing in the study of Baeyer-Villiger Monooxygenases (BVMOs) from *Mycobacterium tuberculosis* (*Mtb*). His research focuses on the enzymatic and structural characterization of these enzymes involved in the activation of prodrugs against tuberculosis, such as ethionamide and thiacetazone. He uses advanced biophysical techniques, including mass photometry, dynamic light scattering, cryo-electron microscopy and *in vitro* enzymatic assays, along with optimized protein expression and purification methods, to study the structural and functional properties of BVMOs.

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Publications related to the presentation

- Tomas N, Leonelli D, **Campoy M**, Marthey S, Le NH, Rengel D, Martin V, Pál A, Korduláková J, Eynard N, Guillet V, Mourey L, Daffé M, Lemassu A, André G, Marrakchi H. Bioinformatic Mining and Structure-Activity Profiling of Baeyer-Villiger Monooxygenases from *Mycobacterium tuberculosis*. mSphere. 2022 Apr 27;7(2):e0048221. DOI: https://doi.org/10.1128/msphere.00482-21 Epub 2022 Mar 17. PMID: 35296143; PMCID: PMC9044951

Speaker

Harinath Chakrapani

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

Towards identifying new druggable targets through chemoproteomics

Multi-drug resistance has emerged as a major threat to public health globally. Tacking this requires a multi-pronged approach. Identifying new leads with novel mechanisms of action becomes the foundation to develop new therapeutic strategies. Using medicinal chemistry approaches and analogue synthesis, we identify lead compounds with high potency against a panel of drug-resistant strains. We then synthesized and validated probes that can be used for chemoproteomics. Using advanced mass-spectrometry platforms as well as molecular biology techniques, we identified unique protein targets for these lead molecules. These proteins become the basis for new drug discovery. Taken together, we use a combination of organic synthesis, chemical biology and advanced mass-spectrometry platforms to identify new druggable targets. Assay development and high-throughput screening are presently underway for these targets.

Harinath Chakrapani completed his undergraduate and post-graduate studies in Chemistry from Loyola College and Indian Institute of Technology Madras, respectively. He then moved to Duke University, USA and completed his PhD in 2005. His post-doctoral research work was carried out at Wake Forest University and the National Cancer Institute. He joined IISER Pune in July 2009 and is currently Professor. His research interests are in developing tools to interogate redox biology, and identify new druggable targets in bacteria.

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Publications related to the presentation

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Speaker

Arunava Dasgupta

Senior Principal Scientist at CSIR-Central Drug Research Institute (CSIR-CDRI), Lucknow, India

Discovering new drugs to combat old bugs: a quest for new arsenal against mycobacteria

Tuberculosis is an ancient disease that has taken a deadly new turn with the emergence of drug-resistant strains. While several clinical candidates are currently in development, there remains an urgent need for new therapeutic options. Additionally, *Mycobacterium abscessus*, a distant relative of *Mycobacterium tuberculosis*, is rapidly emerging as a serious healthcare threat due to its high level of drug resistance and the rising number of reported cases worldwide, particularly among immunocompromised individuals and patients with cystic fibrosis. Unfortunately, the drug development pipeline for *M. abscessus* is virtually empty.

To address the urgent need for effective therapies against mycobacterial infections, especially drug-resistant forms, our lab employs two complementary strategies: traditional phenotypic whole-cell screening and the repurposing of FDA-approved drugs for new clinical applications. In my talk, I will present and highlight several success stories that have emerged from our lab's efforts

Dr Arunava Dasgupta is a Senior Principal Scientist/Professor in the Department of Molecular Microbiology and Immunology and is in charge of the International Science & Technology Affairs Group of CSIR-Central Drug Research Institute, Lucknow. His research focuses on host-pathogen interactionand drug discovery against resistant mycobacteria and ESKAPE pathogens. His lab specialises in biological evaluation (*in vitro*, *ex vivo* and *in vivo* models) of synthetic compounds and natural products for anti-bacterial activity, target-based screening and identification of new drug targets/target assays to combat AMR.

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Speaker

Sheetal Gandotra

Senior Principal Scientist at CSIR-Institute of Genomics and Integrative Biology (CSIR-IGIB), New Delhi, India

Phospholipid remodelling in mycobacteria

Phospholipids perform chief compartmentation functions of any cell. The combination of polar head groups and fatty acid chain length, unsaturation, and modifications, provides the unique structural and biophysical features to individual members of this class of lipids. These unique features dictate membrane shape, protein recruitment/activity and cell signalling. We have identified a phosphatidyl inositol phosphate (PIP) remodelling pathway in mycobacteria, that generates unusually long chain fatty acid PIP species. The loss of this PIP species is accompanied by increased abundance of phosphatidyl ethanolamine and phosphatidyl inositol. We find that this altered phospholipid composition is not resulting from a change in the transcript abundance of genes involved in biosynthesis of these phospholipids, suggesting a remodelling at the metabolite level. We are currently investigating the function of this modified PIP in bacterial physiology in vitro and *in vivo*.

Sheetal Gandotra is a Senior Principal Scientist at the CSIR-Institute of Genomics and Integrative Biology, New Delhi. Her group works to understand the role of lipid metabolism during *Mycobacterium tuberculosis* infection. Using a multipronged approach of cell and molecular biology, genomics and biochemistry, her group studies the role of lipid droplet lipids and proteins in the innate immune defense against mycobacteria as well as lipid remodelling pathways of mycobacteria that help it counter host defense mechanisms.

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Publications related to the presentation

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Speaker

Ranjana Pathania

Professor at Indian Institute of Technology Roorkee (IIT Roorkee), Roorkee, India

Revisiting bacterial pathophysiological fitness determinants as potential drug targets

Antimicrobial resistance (AMR) in bacterial pathogens is a critical global health challenge, aggravated by the slow development of new antibiotics and the emergence of novel resistance mechanisms. To address AMR, our lab employs a dual strategy: chemical genetics to discover innovative antibacterials and fundamental research into sRNA-mediated regulatory networks to uncover potential drug targets. Through chemical genetics, we discovered IITR07865, a novel chemical probe targeting MreB protein of the rod complex. Genomics and transcriptomics studies on *E. coli* suppressor mutants of IITR07865 revealed that upregulation of LdtA, an enzyme crosslinking peptidoglycan to Lpp, renders the rod complex non-essential. Although LdtA is typically considered non-essential, our findings highlight its role in conferring resistance to rod complex inhibitors like mecillinam, a clinical antibiotic used in UTI treatment. This insight opens avenues for combination therapies targeting resistance pathways to restore antibiotic efficacy. In parallel, our fundamental research uncovered the dual role of the RNA-chaperone Hfq in *A. baumannii*. Hfq, with its conserved glycine-rich C-terminal domain, is crucial for pathophysiological fitness in this pathogen. Disruption of hfq or its glycine repeats attenuates fitness and increases antibiotic susceptibility, making Hfq a promising therapeutic target. However, we discovered that Δhfq strain exhibits enhanced antibiotic persistence. Transcriptome analysis revealed pleiotropic perturbations of cellular pathways, leading to altered cellular energetics and increased persister frequency against cefepime, a frontline antibiotic for *A. baumannii*. We establish that owing to their perturbed genetic circuits, *A. baumannii* Hfq mutants are able to tolerate cefepime treatment in a murine lung infection model. Our work highlights the need to revisit conventional and unconventional determinants of pathophysiological fitness to develop better drugs in the future.

Dr. Pathania is a professor at the Department of Biosciences and Bioengineering, IIT Roorkee heading the Molecular Bacteriology and Chemical Genetics (MBCG) group. Her research delves into two aspects of infectious agents- one, using chemical genomics to aid in drug discovery against multidrug-resistant pathogens, and the other, centred on understanding the pathophysiology of the pathogenic *Acinetobacter baumannii*. She has published in reputed journals, holds multiple patents, and serves on editorial boards of Communications Biology and ACS Infectious Diseases. Recognized for her contributions, she received the DBT/Wellcome Trust India Alliance Senior Fellowship (2022) and was elected as a Fellow of the National Academy of Science, India (2022).

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Speaker

Ramandeep Singh

Professor at BRIC-Translational Health Science and Technology Institute (BRIC-THSTI), Faridabad, India

Identification of novel therapeutic interventions against *Mycobacterium tuberculosis*

We have standardized high throughput screening assays to identify small molecules with anti-tubercular activity. Usingh performed phenotypic screening we have identified pyridine carboxamide as a hit molecule. We have now performed a detailed structure activity relationship study and identified a lead molecule that shows efficacy in mice. In another study, we have validated Polyphosphate Kinase -1 as a drug target for *M. tuberculosis*. Using 96-well assay target based screens, we have identified an FDA approved drug that inhibits the enzymatic activity of PPK-1 enzyme. The drug molecules identified from phenotypic and target based screens show activity against both drug-susceptible and drug-resistant strains of *M. tuberculosis*.

Dr. Ramandeep Singh has experience working in the field of Tuberculosis for more than 20 years. The focus of the research lab is to study mechanisms of pathogenesis and validation of drug targets for *Mycobacterium tuberculosis*. He has performed high-throughput screening in phenotypic, target-based and macrophage-based assays and identified small molecules that are active in animal models.

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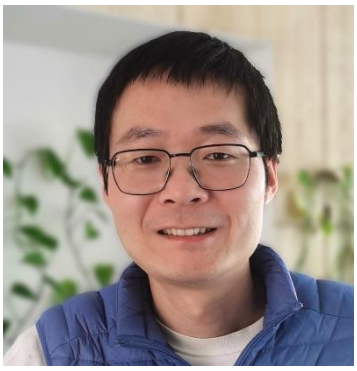
Speaker

Xibing Xu

Postdoctoral fellow at Laboratoire de Microbiologie et Génétique Moléculaires (LMGM-CBI), Toulouse, France

Nucleotidyltransferase toxins MenT extend tRNA 3'-end to control *Mycobacterium tuberculosis* growth

Toxin-antitoxin (TA) systems are evolutionary conserved genetic modules found across bacterial genomes and mobile genetic elements. They play a pivotal role in bacterial stress response, maintenance of genomic stability, and regulation of virulence factors. Under physiological conditions, TA systems maintain a delicate equilibrium between the toxin and antitoxin components, ensuring bacterial growth and survival. However, environmental stressors such as phage infections or plasmid loss can disrupt this balance, leading to activation of the toxin and subsequent inhibition of essential cellular functions. In this study, we characterized the nucleotidyltransferase (NTase) toxins MenT1–4, which belong to the AbiE family of MenAT toxin-antitoxin (TA) systems in *Mycobacterium tuberculosis* (*Mtb*), the causative agent of human tuberculosis. These MenT toxins are capable of adding extra nucleotides to the 3'-CCA end of tRNA. We show that both MenT1 and MenT4 modify all tRNAs tested in vitro without preference. However, while MenT1 uses CTP as nucleotide substrate, MenT4 prefers GTP. Moreover, MenT3 is a robust cytidine specific tRNA NTase *in vitro*, modifying the aminoacyl acceptor ends of most tRNA but with a marked preference for tRNASer, to which long stretches of cytidines are added. To gain insights into the in vivo relevance of MenT toxins, we conduct transcriptomic analysis to identify their targets within *Mtb*. Remarkably, we find that MenT3 predominantly targets tRNASer, highlighting its crucial role in tRNA modification. Furthermore, our data unveil significant detoxification responses by CCA-adding enzyme in response to MenT3 overexpression, underscoring the cellular efforts to mitigate toxin-induced damage. Intriguingly, our investigation discover the unexpected presence of an active pool of endogenous MenT3 targeting $tRNA^{Ser}$ under physiological conditions, only in the presence of the native menAT3 operon. This discovery suggests a key role for MenT3 in *Mtb* biology, possibly influencing pathogenicity and virulence during infection.



Xibing completed his PhD in 2015 at Sichuan University in China, where he studied bacterial stress response mechanisms. Following this, he worked as a lecturer at Henan University of Science and Technology, focusing on ubiquitin-like modification in bacteria. In 2020, he resigned from this position and joined the team of Pierre Genevaux at the Centre for Integrative Biology (CBI) in Toulouse as a postdoctoral researcher. His research has since focused on growth control mechanisms mediated by bacterial toxin-antitoxin systems, with particular interest in ribosome-dependent RNase and tRNA nucleotidyltransferase toxins.

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Publications related to the presentation

- **Xu X**#, Barriot R, Voisin B, Arrowsmith TJ, Usher B, Gutierrez C, Han X, Redder P, Blower TR, Neyrolles O & Genevaux P#. Nucleotidyltransferase toxin MenT extends aminoacyl acceptor ends of serine tRNAs to control *Mycobacterium tuberculosis* growth. Nat Commun 15, 9596 (2024). DOI: https://doi.org/10.1038/s41467-024-53931-w

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Scientific session 4

Chair

Sheetal Gandotra

Senior Principal Scientist at CSIR-Institute of Genomics and Integrative Biology (CSIR-IGIB), New Delhi, India

Sheetal Gandotra is a Senior Principal Scientist at the CSIR-Institute of Genomics and Integrative Biology, New Delhi. Her group works to understand the role of lipid metabolism during *Mycobacterium tuberculosis* infection. Using a multipronged approach of cell and molecular biology, genomics and biochemistry, her group studies the role of lipid droplet lipids and proteins in the innate immune defense against mycobacteria as well as lipid remodelling pathways of mycobacteria that help it counter host defense mechanisms.

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Chair

Dhiraj Kumar

Group Leader at International Centre for Genetic Engineering and Biotechnology (ICGEB), New Delhi, India

Dhiraj Kumar is a Group Leader at the International Centre for Genetic Engineering and Biotechnology, New Delhi, where he leads the Cellular Immunology Group. The research focus of his group is to explore diNerent facets of innate immunity during tuberculosis pathogenesis. The research topic includes regulation of autophagy, immune cell infiltration, cell detah and inflammation. The aim is to harness the innate immune arm to develop novel host-directed preventive and therapeutic approaches against TB.

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Speaker

Rachit Agarwal

Associate Professor at Indian Institute of Science (IISc), Bangalore, India

Engineering approaches to tackle lung infections: bacteriophages and inhalable carriers

Tuberculosis (TB) remains a major global health concern, with drug-resistant strains posing a significant challenge to eOective treatment. Bacteriophage (phage) therapy has emerged as a potential alternative to combat antibiotic resistance. However, two major challenges exist in translating phage therapy for the prevention and treatment of TB. First, the infection dynamics of phages against Mycobacterium tuberculosis (Mtb) have not been studied in various pathophysiological environments. Secondly, the delivery of large amounts of active phages to the lungs is limited due to their poor aerosolization properties. In this talk, I will discuss our results on the eOicacy of widely used mycobacteriophages (D29, TM4, DS6A) against Mtb under pathophysiological conditions associated with TB, such as low pH and hypoxia. Even at low multiplicity of infection (MOI), mycobacteriophages eOectively infected Mtb, got rapidly amplified, and lysed Mtb. We also observed that phages were eOective in lysing bacteria even under low pH and low oxygen concentrations, as well as antibiotic-resistant bacteria. I will also discuss polymer microcarrier-based approaches to facilitate the delivery of phages and other anti-bacterials for inhalation-based delivery in targeting lung immune cells. We found that Mtbinfected macrophages are highly phagocytic, and microparticle surface charge plays a major role in particle internalization by infected cells. Mtb-infected macrophages internalized microparticles of diOerent sizes (0.5–2 μm) in large numbers. Cytocompatibility assay and histological analysis in mice confirmed that the formulations were safe and did not elicit any adverse reaction. Additionally, pulmonary delivery of cationic particles in mice resulted in two-fold higher uptake in resident alveolar macrophages than non-modified particles. This study provides a framework for the future design of phage therapy for the prevention and treatment of TB.

Rachit Agarwal is an associate professor at the Indian Institute of Science, Bangalore, India. He worked in the field of biomaterial-based drug delivery for his PhD. at the University of Texas at Austin, Texas, USA. His post-doctoral fellowship was in regenerative medicine at the Georgia Institute of Technology, Atlanta, USA. His scientific interests are in developing biomaterial-based delivery vehicles for the treatment of inflammatory and infectious diseases. He is a recipient of the prestigious Ramanujan fellowship, Har-Gobind Khorana Young Biotechnologist award, OPPI Young Scientist Award, and DBT/Wellcome Trust India Alliance Intermediate Fellowship.
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Speaker

Saurabh Chugh

Postdoctoral fellow at Institute of Pharmacology and Structural Biology (IPBS), Toulouse, France

Exometabolome Insights : A new perspective on *Mycobacterium tuberculosis* virulence

Mycobacterium tuberculosis (*Mtb*) is the causative agent of tuberculosis and poses a global threat to humanity. While macrophages are naturally equipped with a variety of mechanisms to eliminate potentially harmful invading microorganisms, they nevertheless permit the intracellular replication of *Mtb*. *Mtb* can endure various stresses, including hypoxia, low pH, and nutrient limitation, for prolonged periods in a state known as dormancy or "quiescence." The remarkable ability of Mtb to withstand numerous host-imposed stresses during its life cycle largely depends on the metabolic plasticity of this pathogen, its unique capacity to extract a wide range of nutrients from its host's cells and tissues—even in nutrient-poor microenvironments such as granulomas—and on the myriad virulence factors it can produce to disrupt host functions. Furthermore, microbial metabolic activity leaves an extracellular footprint, and *Mtb* is no exception. Notably, bacteria release small signaling molecules, metabolic intermediates, or end-products into their environment. This process assists in removing cellular metabolic by-products from the intracellular environment to maintain homeostasis. The comprehensive profiling of extracellular metabolites, also known as the exometabolome, is widely employed to assess and compare the metabolic states of different microbial systems. While the role of metabolism in bacterial-host interactions has been extensively studied, the contributions of the bacterial exometabolome to metabolic adaptations and disease pathogenesis remain largely unexamined. We aim to elucidate the role of *Mtb* exometabolites in virulence and pathogenesis.

Saurabh Chugh is a Marie Curie Postdoc Fellow in Prof. Olivier Neyrolles's laboratory at the Institute of Pharmacology and Structural Biology, Toulouse, France. The main objective in his postdoc work is to study the exometabolome of *Mycobacterium tuberculosis* and how exometabolome impacts virulence and pathogenesis. He did his PhD in the laboratory of Prof. Ramandeep Singh at the Translational Health Science and Technology Institute in India. His PhD work focused on how inorganic polyphosphate regulates metabolic pathways in *Mycobacterium tuberculosis*. Additionally, part of his PhD work was also focused on drug and vaccine development against *Mycobacterium tuberculosis*.

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Publications related to the presentation

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Speaker

Ashwani Kumar

Senior Principal Scientist at CSIR-Institute of Microbial Technology (CSIR-IMTech), Chandigarh, India

Mycobacterial biofilms; Emerging hypothesis of *in vivo* drug tolerance and resistance

The capability of *Mycobacterium tuberculosis* (*Mtb*) to form the biofilm could explain the phenotypic drug tolerance displayed by *Mtb* in humans. Recent literature points to the drug tolerance observed in biofilms that may help in the evolution of drug resistance. Here, I will highlight that intracellular thiol reductive stress (TRS) induces *Mtb* biofilm formation. TRS-induced biofilms harbor drug-tolerant but metabolically active bacteria with comparable levels of ATP/ADP, NAD+/NADH, and NADP+/NADPH. Evidence generated through electron microscopy and confocal microscopy revealed that cellulose is a major component of *Mtb* biofilms. Further analyses suggest that cellulose is universally used as a major component of different types of biofilms across slow and fast-growing mycobacterial species. Using cellulose as a biomarker, we demonstrate that *Mtb* forms biofilms in infected mice's lungs, non-human primates, and human lungs. We have observed that cellulose protects resident bacilli from the host immune system and antimycobacterial agents. Interestingly, new modes of drug resistance/tolerance were observed inside the biofilms. Further studies indicate that Mtb forms intracellular biofilms, although the significance of these intracellular biofilms in TB pathogenesis remains to be analyzed.

Keywords: tuberculosis, biofilms, drug tolerance, protection from the immune system

Dr. Ashwani Kumar is currently a Senior Principal Scientist at the CSIR-Institute of Microbial Technology, Chandigarh, India. His laboratory is interested in understanding the basic biology of tuberculosis, which may explain the chronic nature of the disease and the requirement for multiple drugs for at least six months. Ashwani is the recipient of several awards and honors, including the Shanti Swarup Bhatnagar Prize for Science and Technology, the National Bioscience Award for Career Development, the Swarnajayanti Fellowship, DBT/Wellcome Trust India Alliance Senior Fellowship.

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Speaker

Debapriya Mukherjee

PhD student at Indian Institute
of Science (IISc), Bangalore, India

Critical concentration of formate determines the susceptibility of *Salmonella* Typhimurium to meropenem and ciprofloxacin

The global spread of multidrug-resistant (MDR) *Salmonella enterica* serovar Typhimurium (STM) poses a significant healthcare challenge. Beyond traditional antimicrobial resistance (AMR) mechanisms, STM employs non-conventional pathways to resist antibiotics. Our previous study has shown that the enzyme PflB (pyruvate-formate lyase) is crucial for maintaining intracellular formate, which regulates bacterial intracellular pH (ipH) and supports efflux pump activity. Deleting pflB increases the susceptibility of STM to β-lactam antibiotic meropenem and DNA gyrase inhibitor ciprofloxacin, leading to higher reactive oxygen species (ROS) levels and membrane depolarization, recoverable with extracellular formate. Disrupted ipH and associated membrane depolarization in STM ΔpflB strain upregulates the small RNA csrB via sigma factor RpoE, thereby downregulating efflux pumps AcrB and TolC. Extracellular formate also activates the BarA/SirA system, modulating the CsrA/csrB pathway and efflux pump expression. This study highlights the vital role of in *Salmonella* Typhimurium AMR and the importance of its precise intracellular balance.

Debapriya is in the final year of her doctoral studies, having completed her bachelor's in Microbiology before joining the Indian Institute of Science in 2018 for an integrated MS-PhD program. During the latter part of her master's, she began working in the Molecular Pathogenesis Laboratory, led by Prof. Dipshikha Chakravortty, focusing on characterizing the pathogenic traits of the foodborne pathogen *Salmonella*. Her research interests include host-pathogen interactions, molecular biology, cell biology, and immunology

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Publications related to the presentation

- **Debapriya Mukherjee**, Sakshi Wagle, Abhishek Biswas, Raju S Rajmani, Dipshikha Chakravortty. Critical concentration of formate determines the susceptibility of *Salmonella* Typhimurium to meropenem and ciprofloxacin (Manuscript under preparation)

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Speaker

Olivier Neyrolles

CNRS Research Director and Director of the Institute of Pharmacology and Structural Biology (IPBS), Toulouse, France

Sulfur metabolism in *Mycobacterium tuberculosis* response to stress

Tuberculosis remains the deadliest infectious disease caused by a single pathogen, highlighting the need for novel therapeutic strategies. Mycobacterium tuberculosis relies on sulfur metabolism for critical functions such as protein synthesis, redox buffering, and coenzyme production. While M. tuberculosis can utilize various sulfur sources, the primary substrates exploited during infection remain unclear.
Here, I will discuss our recent identification of inorganic sulfate acquisition via the SubI-CysTWA transporter as a key pathway for M. tuberculosis survival in macrophages. Using NanoSIMS, we observed significant sulfate-derived 33S enrichment in intracellular bacteria, correlating with metabolic activity. Deletion of subI abolished sulfate uptake, impairing growth in vitro and reducing M. tuberculosis survival in murine macrophages and infected lungs. This sulfate acquisition pathway is essential for maintaining redox balance in vivo, distinguishing M. tuberculosis from many intracellular pathogens that rely on organic sulfur sources.
M. tuberculosis also faces hypoxia and copper bursts in phagosomes, stresses that act synergistically as copper accumulation increases under hypoxia. A transposon mutant screen in macrophages identified sulfur metabolism, particularly the cysteine synthase CysK2, as critical for intracellular survival under hypoxia. NanoSIMS confirmed sulfate and cystine reach M. tuberculosis-containing phagosomes, with cystine consumption increasing in hypoxia. Mutants lacking subI or cysK2 exhibited reduced survival in mice, reinforcing the importance of this pathway. CysK2, but not CysK1 or CysM, was strongly induced by copper in vitro and in macrophages. A cysK2-KO mutant experienced heightened oxidative stress in macrophages and in mice. Since animal cells lack sulfate assimilation, targeting SubI-CysTWA could impair M. tuberculosis survival or sensitize bacilli to antibiotics by disrupting redox homeostasis.
These findings highlight sulfur metabolism as a promising therapeutic target for tuberculosis.

Olivier Neyrolles, an agricultural engineer by training, earned his PhD in microbiology at the Pasteur Institute, Paris. After postdocs at Imperial College London and Saint-Louis Hospital, he joined Prof. Brigitte Gicquel's lab at the Pasteur Institute, specializing in mycobacterial biology. He became a CNRS Research Associate (2004) and later founded his lab at IPBS (2007). Now CNRS Research Director, he focuses on tuberculosis and respiratory pathogens. His work has earned major awards, including the CNRS Bronze and Silver Medals, the Sanofi-Institut Pasteur Award, and the Coup d'Élan Prize from the Bettencourt Schuller Foundation, and his lab is recognized for its innovation, global collaborations, and mentoring of young scientists.

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Publications related to the presentation

- Le Mouëllic W, Levillain F, Wu T-D, Poquet Y, **O Neyrolles**. Inorganic sulfate is critical for *Mycobacterium tuberculosis* lung tissue colonization and redox balance (Under review)

- Le Mouëllic W, Levillain F, Larrouy-Maumus G, Wu T-D, Poquet Y, *O Neyrolles*. CysK2 cysteine synthase plays a key role in *Mycobacterium tuberculosis* response to hypoxia and copper stress during infection (Under review)

Speaker

Vivek Rao

Senior Principal Scientist at CSIR-Institute of Genomics and Integrative Biology (CSIR-IGIB), New Delhi, India

Importance of host cell metabolism in tubercuosis control - Cues for the development of novel host directed therapies

Protracted tuberculosis (TB) treatment regimens with a combination of drugs presents as a major hurdle to global TB control programs. Conventional methods of identifying molecules that target the pathogen have inherent problem of inducing resistance in the population. Host directed approaches efficiently circumvent this problem and have shown promise in recent studies. We have developed a novel strategy to restrict *Mycobacterium tuberculosis* (*Mtb*) induced type I IFN response with an FDA approved anti-depressant-Sertraline. We demonstrate significant synergy between sertraline and frontline TB drugs in reducing bacterial burdens in cellular and preclinical models, resulting in early bacterial clearance in animal tissues. We also observed a marked improvement of the host morbidity associated with TB infection in preclinical models by the use of sertraline as adjunct to frontline TB drugs. Our results highlight the impact of using a safe and time tested anti-depressant as an adjunct to TB therapy and provides evidence for faster control of infection in preclinical models.

Vivek Rao joined CSIR-IGIB in 2011 after two post-docs at NIMR, London, and MSKCC, New York, follwing his PhD at the Department of Biochemistry of Delhi University. He has worked on recombinant BCG vaccine development for TB in his PhD, then on innate responses to TB lipids at MSKCC and lineage specific responses in *Mycobacterium tuberculosis* lineages at NIMR. At IGIB, he has repurposed an FDA approved antidepressant- sertraline as an adjunct TB treatment regimen and has acheived earlier bacterial clearance in preclinical models. Addifion of Sertraline to ATT imrpoves host recovery after TB infection significantly. Curently working out modalities for phase 2 trial.

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- Shankaran D, et.al (2023). The antidepressant sertraline provides a novel host directed therapy module for augmenting TB therapy. eLife 12:e64834. DOI: https://doi.org/10.7554/eLife.64834

Speaker

Christel Verollet

INSERM Research Director at Institute of Pharmacology and Structural Biology (IPBS), Toulouse, France

Deciphering mechanisms of cell-to-cell transfer of HIV-1 towards macrophages and their modulation by *Mycobacterium tuberculosis*

In addition to CD4+ T lymphocytes , macrophages emerged as essential target cells involved in all stages of HIV-1 pathogenesis and establishment of persistent viral reservoirs. It is thus crucial to better understand how macrophages become infected by HIV-1. In contrast to cell-free virus infection, cell-to-cell transmission is suspected to represent the dominant mode of infection in vivo and may also allow the virus to escape antiretroviral drugs and the immune system. Our projects aim to characterize the mechanisms that govern the intercellular transmission of HIV-1 toward macrophages. While tuberculosis (TB) is a risk factor in HIV-1-infected individuals, the study of these mechanisms in HIV/ Mycobacterium tuberculosis (Mtb) co-infection settings is crucial to better understand the synergy between these two pathogens, which represents a deadly combination.
We showed that HIV-1 can be transferred between macrophages using tunneling nanotubes (TNT) and that this mechanism is exacerbated in macrophages exposed to TB-associated microenvironments. Glycolytic activity in these macrophages triggers both TNT formation and Siglec-1 expression on microtubule-containing TNT that are stable and carry HIV-1 cargo. We also demonstrated that HIV-1 can be transferred in a very efficient manner from infected T cells to macrophages using a heterotypic cell fusion process leading to the formation of highly virus-productive multinucleated giant cells. This mechanism occurs with several human tissue macrophages, including synovial, placental, lung alveolar and tonsil macrophages. Using state-of-the-art biophysic approaches, we want now investigating, at the structural and molecular levels, the mechanisms that govern this intercellular transmission of HIV-1 from T lymphocytes toward macrophages and how it is modulated by TB. It is indeed of great interest to better characterized how these HIV tissue reservoirs, including lung alveolar macrophages in the HIV/Mtb co-infection context, can be infected by the virus in tissue and at different stages of the disease.

Christel Verollet received her PhD in cell biology from Toulouse University in 2007 working on microtubules organization during mitosis, in Drosophila. As a post-doc at IPBS, she conducted work in the characterization of HIV-1 infection of macrophages in terms of cell fusion and cell migration. In 2012, she was recruited as a permanent researcher by Inserm. As such, she continues to investigate the role of macrophages in HIV-1 and HIV-1/*Mycobacterium tuberculosis* co-infection and started new projects on the biology of osteoclasts in physiological and pathological contexts. She received her HDR diploma in 2018 and be awarded a DR2 (Directeur de Recherche) position at Inserm in 2022. From 2021, she co-leads a team with Dr. Renaud Poincloux at IPBS.

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Speaker

Albertus Viljoen

CNRS Research Scientist at Institute of Pharmacology and Structural Biology (IPBS), Toulouse, France

Surface organization of mycobacterial envelope glycans: Implications for immune interactions

Mycobacterium tuberculosis (*Mtb*) produces a large variety of glycans at its surface that interact with C-type lectin pattern recognition receptors (CLRs) on host immune cells. Some of these interactions trigger phagocytosis and/or intracellular signalling that induce a protective immune response to mycobacterial infection, while others favour infection. Using, Atomic Force Microscopy (AFM)-based single-molecule and single-cell force spectroscopy, we recently demonstrated that the organization of glycans at the bacterial surface plays an important part in the effective recognition of *Mtb* by the CLR DC-SIGN. Building on these findings, we have expanded our study to investigate additional CLRs involved in bacterial recognition, including Dectin-1, Dectin-2, and Mincle. Combining AFM with confocal microscopy, we have characterised the distribution of CLR ligands on the surfaces of pathogenic and non-pathogenic mycobacteria. Our preliminary results reveal how ligand density and organisation contribute to efficient CLR binding at the whole-cell level. These insights enhance our understanding of the molecular mechanisms underpinning pathogen recognition by CLRs and may inform strategies to modulate host-pathogen interactions in mycobacterial infections.

Albertus Viljoen earned his PhD in Molecular Biology from Stellenbosch University, South Africa, in 2013, where his research centered on the metabolic pathways that pathogenic bacteria use to survive within host cells. Following his PhD, he pursued postdoctoral research in France and Belgium, investigating the biochemistry and biophysical properties of bacterial envelopes. Currently, as a CNRS research scientist at the Institute of Pharmacology and Structural Biology (IPBS) in Toulouse, he is focused on uncovering novel molecular mechanisms underlying mycobacterial adhesion during infection.

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Publications related to the presentation

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Scientific session 5

Chair

Arunava Dasgupta

Senior Principal Scientist at CSIR-Central Drug Research Institute (CSIR-CDRI), Lucknow, India

Dr Arunava Dasgupta is a Senior Principal Scientist/ Professor in the Department of Molecular Microbiology and Immunology and is in charge of the International Science & Technology Affairs Group of CSIR-Central Drug Research Institute, Lucknow. His research focuses on host-pathogen interactionand drug discovery against resistant mycobacteria and ESKAPE pathogens. His lab specialises in biological evaluation (*in vitro*, *ex vivo* and *in vivo* models) of synthetic compounds and natural products for anti-bacterial activity, target-based screening and identification of new drug targets/target assays to combat AMR.

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Chair

Olivier Neyrolles

CNRS Research Director and Director of the Institute of Pharmacology and Structural Biology (IPBS), Toulouse, France

Olivier Neyrolles, an agricultural engineer by training, earned his PhD in microbiology at the Pasteur Institute, Paris. After postdocs at Imperial College London and Saint-Louis Hospital, he joined Prof. Brigitte Gicquel's lab at the Pasteur Institute, specializing in mycobacterial biology. He became a CNRS Research Associate (2004) and later founded his lab at IPBS (2007). Now CNRS Research Director, he focuses on tuberculosis and respiratory pathogens. His work has earned major awards, including the CNRS Bronze and Silver Medals, the Sanofi-Institut Pasteur Award, and the Coup d'Élan Prize from the Bettencourt Schuller Foundation, and his lab is recognized for its innovation, global collaborations, and mentoring of young scientists.

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Speaker

Sidharth Chopra

Associate Professor at CSIR-Central Drug Research Institute (CSIR-CDRI), Lucknow, India

Discovering drugs for bad bugs: Progress amongst challenges!

Antimicrobial resistance has been internationally recognized as a significant threat to healthcare systems worldwide. Due to multiple reasons including unfavorable economics, increasing emergence of drug-resistant bacteria and expensive and lengthy clinical trials, most of the big pharma has exited the discovery and development of new anti-infectives. In order to augment the non-existent to depleted drug-discovery pipeline targeting drug-resistant bacteria, our lab follows 2 complimentary approaches: conventional phenotypic whole cell screening of diverse chemical scaffold libraries as well as natural products and drug-repurposing of FDA approved drugs for new clinical uses. In my talk, I will discuss and highlight the challenges which we face along with some success stories which have come out of my lab. The talk will highlight the core issue: drug discovery against drug-resistant microbes is a extremely interdependent, interconnected battle involving multiple expertise's against an very intelligent, ruthless and adaptable adversary.

Dr. Sidharth Chopra completed his PhD from ICGEB/JNU in 2004 and moved to Stanford University School of Medicine (USA). From 2008 to 2012, he was Research Scientist at SRI International, Menlo Park, USA. In 2013, he moved to CSIR-Central Drug Research Institute, Lucknow (CSIR-CDRI) to establish his own laboratory working on drug discovery targeting MDR bacterial pathogens. He is Principal Scientist/Associate Professor in Department of Molecular Microbiology and Immunology. His research focuses on complimentary issues of drug discovery for MDR bacterial pathogens and delineating antimicrobial resistance mechanisms prevalent under Indian conditions.

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Publications related to the presentation

- https://scholar.google.com/citations?user=OXpp0UYAAAAJ&hl=en

Speaker

Parvinder Kaur

Principal Scientist at Foundation for Neglected Disease Research (FNDR), Bangalore, India

Humanised 3-D granuloma predicting reactivation of Tuberculosis

Human TB granuloma, a hallmark of *Mtb* infection, are associated with dormant or non-replicating (NRP) *Mtb*, causing latent TB infection. Immunomodulatory drugs prescribed for several autoimmune diseases and cancer immunotherapy alter the immune status of patients which can trigger TB reactivation, converting NRP *Mtb* replicating (REP), that leads to active TB disease. Several such drugs carry black-box warnings from the US FDA, for a risk of patients to develop TB. As more patients in high TB burden countries are prescribed immunomodulatory drugs, TB reactivation cases will lead to a further increase in TB burden. Typically, TB reactivation risk is only assessed during clinical trials. Development of an *in vitro* TB reactivation assay assessing TB reactivation by drug candidates early in drug development pathway will help pharmaceutical companies in selecting clinical candidates with a lower likelihood of reactivating TB in patients.
An *in vitro* 3D human TB granuloma model, mimicing in vivo like/intracellular conditions, can be used to study the risk of latent TB reactivation (Arbues 2020a; 2020b, Kapoor 2013). We have developed an *in vitro* 3D human TB granuloma model-based assay in a 24-well format using human PBMCs and validated it using TNF-α inhibitors like Adalimumab, Etanercept showing clinical evidence of TB reactivation. This assay helps understanding the likelihood of TB reactivation by estimating conversion of NRP to REP *Mtb*.

Dr Parvinder Kaur is a Medical Microbiologist, and worked with AstraZeneca Pharmaceuticals for ~ 20 years. Currently she is Principal Scientist- Microbiology at Foundation for Neglected Disease Research (FNDR), a not-for-profit company, dedicated to discovery and development of new drugs, diagnostics and devices for the neglected tropical diseases of high socio-economic impact.
She manages Microbiology department and drug discovery projects spanning over early to late discovery in FNDR portfolio. Dr. Kaur has been part of several preclinical TB candidates few of which entered into clinical trials like AZD-5847, and recently TBA 7371 another TB drug that completed phase2A clinical trial. She has ~40 publications, and her core interest remains in TB, BSL3facilities/biosafety and AMR.

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Speaker

Hugo Lebrette

CNRS Researcher at Laboratoire de Microbiologie et Génétique Moléculaires (LMGM-CBI), Toulouse, France

Structural dynamics of radical enzymes and metalloenzymes by femtosecond crystallography

Radicals get bad press as they are considered responsible for damaging cells, causing aging and diseases. However, radicals are also essential to many enzymes involved in fundamental biological processes. Half a century ago, the tyrosyl radical in the ribonucleotide reductase (RNR) was the first stable protein radical to be observed. RNR is a drug target for both cancer and infectious diseases as it provides the only pathway for de novo synthesis of deoxyribonucleotides, the building blocks of DNA. Aerobic RNR relies on the R1 subunit to perform the ribonucleotide reductionand on the ferritin-like R2 subunit to produce a catalytic radical upon oxygen activation. The radical is translocated between the subunits via reversible proton-coupled electron transfer.
We are interested in several subclasses of aerobic RNRs with different means of radical generation, which depend on dinuclear iron and/or manganese cofactors (except in particular case of metal-independent RNR such as in mycoplasmas). In order to
study these metalloenzymes in action, we aim to capture snapshots of their cofactors in di`erent redox states by serial femtosecond X-ray crystallography and simultaneous X-ray emission spectroscopy at X-ray free-electron laser (XFEL) sources. Using a drop-on-tape sample delivery system with *in situ* O2-incubation at room temperature, we could obtain high-resolution XFEL crystal structures, including the structure of a R2 subunit with an intact radical present in its core. Here, I will present our recent results and discuss the advantages of XFEL to study metalloenzymes.

Hugo Lebrette obtained a PhD in structural biology from the University of Grenoble, after studying nickel uptake in pathogenic bacteria under the supervision of Christine Cavazza at the IBS in Grenoble, France. He then studied the catalytic mechanisms of several metalloenzymes as a postdoc in Martin Högbom's group at Stockholm University, Sweden. He joined the CNRS in 2022 to start his group 'The Mycoplasma Metalloproteome' at the LMGM-CBI in Toulouse, which aims to understand the role of transition metals and metalloproteins in minimal mycoplasma pathogens. The group is developing an integrative, multidisciplinary approach combining biochemistry, structural biology (in particular with X-ray free electron laser), enzymology, proteomics, bioinformatics, molecular biology and microbiology.

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Speaker

Hedia Marrakchi

CNRS Research Director at Institute of Pharmacology and Structural Biology (IPBS), Toulouse, France

Mycobacterial cell envelope as an inspiring target for anti-tuberculosis drug discovery

The challenge to developing new antibacterial compounds with activity against Mycobacterium tuberculosis (Mtb) is partly due to unique features of this pathogen, especially the composition and structure of its complex cell envelope. Therefore, targeting enzymes involved in cell envelope synthesis has been of major interest for anti-TB drug discovery. We will describe our efforts to target FadD32, a bi-functional enzyme involved in the biosynthesis of the cell wall mycolic acids. While targeting the mycolic acid biosynthesis pathway is a historical strategy against Mtb, the FadD32 enzyme is still underexplored, although a highly relevant and druggable target. The focus will be on the strategy we used to build a miniaturized high-throughput screening platform and exploit it for a drug repurposing campaign. This led to identification of one promising pharmacophore, presenting a salicylanilide scaffold that we are studying further by synthesizing and evaluating derivatives. This compound and some of its derivatives are potent inhibitors of Mtb, which suggests that salicylanilides represent a potentially promising pharmacophore. Exploring structure-activity relationships, we develop a hit to a lead medicinal chemistry approach, searching for novel anti-tubercular candidates targeting FadD32. This work illustrates the relevance of FadD32 as a drug target, and the value of drug repurposing campaigns to discover new leads in the challenging anti-TB drug discovery.

Hedia Marrakchi is a CNRS research director and head of the group "mycobacterial envelopes and therapeutic targets" at the IPBS. She received her PhD in Biochemistry from the University of Toulouse, working on mycobacterial fatty acid synthesis, then completed a postdoctoral training at St Jude Children's research Hospital, Memphis (USA) in the department of infectious diseases, before joining the CNRS as a research scientist.
Her research interests focus on bacterial fatty acid synthesis and regulation, mycobacterial lipid metabolic pathways and cell envelope biogenesis, structure-function characterization of potential drug targets, and exploring innovative molecules and strategies in the fight against antibiotic resistance in TB and mycobacterial infections.

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Publications related to the presentation

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Speaker

Lionel Mourey

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Targeting an old target: is mycobacterial InhA suitable for rational drug design?

InhA, an NAD-dependent enoyl-acyl carrier protein reductase, is involved in the biosynthesis of mycolic acids, major and specific lipids of mycobacteria. InhA is the target of isoniazid, a first-line anti-tuberculosis drug used since the 1950s. Isoniazid is a pro-drug that needs to be activated by the catalase-peroxidase KatG. Due to resistance problems, mainly due to mutations in katG, a substantial amount of work has been carried out to identify or design direct inhibitors of InhA, demonstrating that this enzyme is still considered a relevant target for the discovery of new anti-tuberculosis drugs.
Most of this work incorporates structural studies, and to date over a hundred crystallographic structures have been deposited in the Protein Data Bank. InhA comprises 269 amino acid residues which adopt the classical fold of the enoyl reductase family and associate in a tetramer. One of the special features of the InhA tertiary structure is the movement of its substrate-binding loop (SBL), which comprises a helix-loop-helix motif that contributes to the spatial definition of the active site. Since the beginning of studies on InhA (first structure published in 1995), variations in the SBL have been discussed by some authors in relation to the efficacy of inhibitors, but no rationale has really been established. Our recent efforts to establish structural links of causality will first be presented. Then, we will present our work aimed at designing, synthesising and evaluating inhibitors and characterising their interactions with InhA from a biophysical and structural point of view. In particular, we try to capitalise on advanced organic chemistry methods involving the pre-organisation of fragments by the protein, such as kinetic target-guided synthesis (KTGS) and dynamic combinatorial chemistry combined with X-ray crystallography (DCX), to discover new direct inhibitors.

After graduating as a organic chemist from the Ecole Nationale Supérieure de Chimie in Strasbourg, Lionel Mourey completed a doctorate and a post-doctorate at the Institut de Biologie Moléculaire et Cellulaire in Strasbourg. He was appointed to the CNRS in 1992 to take part in the development of structural biology, in particular bio-crystallography, in Toulouse. Since 2002, he has been heading the structural biophysics team at the IPBS. The main research activity of the team focuses on *Mycobacterium tuberculosis* and *Mycobacterium abscessus*, and their studies concentrate on systems – most of them enzymatic – essential to mycobacterial physiology, which represent potential therapeutic targets.

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Publications related to the presentation

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Speaker

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PhD student at Indian Institute of Technology Roorkee (IIT Roorkee), Roorkee, India

Deciphering the role of cysteine biosynthesis pathway in the pathophysiology and virulence of *Acinetobacter baumannii*

Acinetobacter baumannii is a member of the ESKAPE group of pathogens and a significant threat to healthcare settings. Owing to the plastic genome and a remarkable capability to acquire antibiotic resistance genes, *A. baumannii* has developed resistance to a wide range of clinically relevant antibiotics. In our lab, we have been focusing on identifying pathophysiological fitness determinants in *A. baumannii* and developing novel therapeutics against this pathogen. In our quest to identify non-conventional metabolic pathways critical to *A. baumannii* fitness, we have been focusing on the serine metabolism and cysteine biosynthesis pathways in this pathogen. Cysteine biosynthesis is a two-step pathway; carried out by serine acetyltransferase and cysteine synthases. We found that contrary to other pathogenic bacteria, *A. baumannii* uses two functionally unique serine-acetyltransferases in synthesizing cysteine, an amino acid that is important for stress mitigation in *A. baumannii*. These serine acetyltransferases differ in their transcript abundance, the ability to interact with cysteine synthases and feedback inhibition by the end product, l-cysteine. We found that disruption of cysteine biosynthesis alters colony size and cellular morphology in *A. baumanii*. The disruption of the cysteine biosynthesis pathway at different steps alters biofilm formation, indicating that the cysteine biosynthesis intermediate plays a role in biofilm formation. This observation holds promise to further develop therapeutic strategies to mitigate this pathogen as biofilms play a critical role in the success of *A. baumannii* in healthcare settings. Moreover, we found that the cysteine biosynthesis pathway contributes to the survival and growth in the presence of antibiotics targeting diverse pathways, highlighting the possibility of developing inhibitors of cysteine biosynthesis and their use in combination with conventional antibiotics.

Avik obtained his master's degree in microbiology before joining Prof. Ranjana Pathania's laboratory at IIT Roorkee for his doctoral research. With a background in microbiology and his training in molecular biology, he is working on understanding the pathophysiology of *Acinetobacter baumannii*, a significant threat to the healthcare system. He focuses on identifying key pathways that provide pathophysiological fitness to *A. baumannii* and deciphering their molecular regulations by non-coding RNAs. Through his approach, he aims to uncover pathways that can further be investigated as targets for antibacterial compounds to aid in the drug discovery approach aimed at mitigating this pathogen.

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Speaker

Maxime Pingret

PhD student at Institute of Pharmacology
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Exploring the Role of a Novel β3-Tubulin Expressing Cell Population in *Mycobacterium tuberculosis* Pathogenesis

Mycobacterium tuberculosis (*Mtb*), the causative agent of tuberculosis (TB), remains the leading cause of death from infectious diseases worldwide. While interaction between the immune system and *Mtb* has been extensively studied, emerging evidence suggests that the nervous system also influences disease pathology. Recently, we identified the emergence of a novel non-immune cell population expressing β3-tubulin, a pan-neuronal marker, within the lungs of *Mtb*-infected mice. Intriguingly, these cells localize to inflamed areas and often found near the bacteria-rich zones, hinting a potential role in the disease. Our objective is to define the nature, origin, and function of this novel cell population. Using immunohistology, we detected these cells in additional animal models and in human pulmonary biopsies, underscoring the translational relevance of our findings. Despite extensive co-staining with markers for known resident lung cell types, none match the phenotype of these β3-tubulin-expressing cells. To address this knowledge gap, we will utilize high-resolution spatial transcriptomics on infected mouse lungs to uncover their gene signature and precise identity. In parallel, we are optimizing methods for isolating these cells, which will then undergo single-cell RNA sequencing to characterize their molecular profile and enable ex vivo functional studies. This will allow us to investigate their potential roles in modulating immune responses or facilitating bacterial persistence. By characterizing this novel cell population, our work aims to deepen understanding of non-immune cellular involvement in TB. These insights could pave the way for innovative therapeutic strategies targeting these cells to improve disease outcomes.

Maxime began his university journey in 2018 with a Technician Diploma in Biological Engineering, specializing in the Food Industry, at the University Institute of Technology of Paul Sabatier University in Auch. Following this, he pursued a general bachelor's year in Microbiology before advancing to a master's degree in Immunology, Immunopathologies, and Infections at the University of Toulouse. Currently, he is a PhD student at the IPBS, where he studies the interactions between the immune and nervous systems during *Mycobacterium tuberculosis* infection in mice.

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Speaker

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PhD student at Indian Institute
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Tuberculosis granuloma organoids for studying host-pathogen interactions

Tuberculosis (TB) kills more than a million people yearly. Most of the host-pathogen interaction during the infection happens within tight clusters of host immune cells surrounding the bacteria, called granulomas. Given their indispensable role in bacterial control and treatment success, our understanding of the host dynamics within such structures remains limited due to the lack of human disease-mimicking TB research models. While animal models are routinely used to study TB, they are not primarily hosts of *Mtb* and not all animals recapitulate the hypoxic and necrotic microenvironment of human granulomas. Existing *in vitro* granuloma models also do not capture the entire complexity of human granulomas. We previously reported a static 3D collagen hydrogel culture system as a reliable mimic of the in vivo infection microenvironment (Gupta et al., 2024). This static collagen culture system was further modified into a flow-incorporated granuloma-on-a-chip platform to mimic key events that drive granuloma formation such as immune cell recruitment and cytokine gradient generation. By flowing media and additional human-derived immune cells at lung interstitial velocities into an ECM mimicking matrix, we are able to mimic the infection dynamics as seen *in vivo* and generate granuloma-like large aggregates (>500 µm). We are nowvalidating the granuloma for hypoxia, necrosis, spatial organization, and transcriptional signatures to assess their similarity with primary human granulomas. We hypothesizethat our *in vitro* granulomas will be a successful model of hypoxic and necrotic human granulomas, currently lacking in the field and will hold immense potential to answer intricate questions about how the type of immune response in response to *Mtb* exposure correlates with bacterial control. This will also pave the way for testing small molecule antagonists/ agonists with immunomodulatory functions for developing host-directed therapies.

Vijaya Vaishnavi is a PhD candidate at the Indian Institute of Science, Bengaluru. She completed her undergraduate studies in Biotechnology at the Ramaiah Institute of Technology, Bengaluru where she was recognised with the 'Best Achiever Award.' In the year 2021, she won the first prize and a cash award of Rs. 20,000 in the 'Voice for BT' public speaking South Zone contest organised by ABLE India and Novozymes. Her 'Train water harvesting' idea to conserve water won her USD 1300 under the 'Spirit of Invention (InvEnt) Scholarship' programme by Avery Dennison Foundation and IIE in 2019. Currently, her doctoral thesis under the mentorship of Prof. Rachit Agarwal, aims to understand heterogeneity in tuberculosis infection using three-dimensional *in vitro* granuloma organoids.

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Publications related to the presentation

- V. K. Gupta, **V. V. Vaishnavi**, M. L. Arrieta-Ortiz, A. P.S., J. K.M., S. Jeyasankar, V. Raghunathan, N. S. Baliga, R. Agarwal, 3D Hydrogel Culture System Recapitulates Key Tuberculosis Phenotypes and Demonstrates Pyrazinamide Efficacy. Adv. Healthcare Mater. 2024, 2304299. DOI: https://doi.org/10.1002/adhm.202304299

Round table

International cooperation

This round table will underscore the critical importance of international cooperation in addressing global health challenges, with a particular focus on antimicrobial resistance (AMR). The discussion will highlight the benefits and impact of cross-border collaboration, showcase ongoing international, European, and Indian initiatives in the field, and explore existing mechanisms and funding opportunities that support Franco-Indian cooperation. The round table will bring together perspectives from both academia and the private sector, fostering a comprehensive dialogue on how to strengthen partnerships and drive innovation in global health.

Moderator

Isabelle Saves

Head of International Cooperation at Institute of Pharmacology and Structural Biology (IPBS) and Centre for Integrative Biology (CBI), Toulouse, France

After 15 years' experience in infectious disease research at the CNRS, Dr Isabelle Saves turned to international cooperation. Seconded to the French Ministry of Foreign Affairs, she initially set up and developed the training program for pharmacists at the University of Antananarivo (Madagascar) coordinating a public-private partnership. With a further four years' experience as attaché for scientific and university affairs at the French Consulate General in Hong Kong and Macao (China), she returned to the CNRS-Toulouse in September 2016 as Head of international cooperation for the Centre for Integrative biology (CBI-Toulouse) and the Institute of Pharmacology and Structural biology (IPBS), research units of the CNRS and the University of Toulouse, home to over 600 research staff, or almost 40% of the biological sciences workforce in Toulouse. Since 2024, she is deputy director of the Federative structure of research "Biology and Biotechnology for Health" of Toulouse.

Her ambition: Promoting outstanding basic research activities led at IPBS and CBI and more generally in Life Sciences/Biomedical institutes of Toulouse University, and developing an international strategy toward increased **international visibility and attractiveness, and an expanded scientific network**.

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Speaker

Parvinder Kaur

Principal Scientist at Foundation for Neglected Disease Research (FNDR), Bangalore, India

Dr Parvinder Kaur is a Medical Microbiologist, and worked with AstraZeneca Pharmaceuticals for ~ 20 years. Currently she is Principal Scientist- Microbiology at Foundation for Neglected Disease Research (FNDR), a not-for-profit company, dedicated to discovery and development of new drugs, diagnostics and devices for the neglected tropical diseases of high socio-economic impact.
She manages Microbiology department and drug discovery projects spanning over early to late discovery in FNDR portfolio. Dr. Kaur has been part of several preclinical TB candidates few of which entered into clinical trials like AZD-5847, and recently TBA 7371 another TB drug that completed phase2A clinical trial. She has ~40 publications, and her core interest remains in TB, BSL3facilities/biosafety and AMR.

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Speaker

Marc Lemonnier

CEO of Antabio, Labège, France

Marc, founding CEO of Antabio, is a molecular and cellular microbiologist with over 25 years' experience in academia and biotech. Prior to founding Antablo, Marc held different research positions at various institutions globally such as CNRS and Inserm (France), CSIC (Spain) and Emory University (USA), authoring over 20 peer-reviewed articles in the field of bacterial pathogenesis and antibiotic resistance. Under Marc's leadership, Antabio has raised 44m funding and received numerous awards including CARB-X and Wellcome Trust (twice). Marc is a co-founding and former member of the BEAM Alliance (European Alliance of Biopharmaceutical companies combating Anti-Microbial resistance), as well as a member of the SAB of JPIAMR (the Joint Programming Initiative on Antimicrobial Resistance).

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Speaker

Leelavati Narlikar

Associate Professor at Indian Institute of Science Education and Research Pune (IISER Pune), Pune, India

Leelavati Narlikar received her Bachelors degree in Computer Engineering from University of Pune and a PhD in Computer Science from Duke University, USA. She is a faculty member in the Department of Data Science at IISER Pune, working at the interface of machine learning and computational biology. A large part of her work is focused on discerning roles played by various cellular biomolecules in regulating gene-expression, especially at the transcriptional level. In particular, her group develops methods to gain biological insights from large-scale genome-level data.

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Speaker

Olivier Neyrolles

CNRS Research Director and Director of the Institute of Pharmacology and Structural Biology (IPBS), Toulouse, France

French Coordinator of the IRN MIRA

Olivier Neyrolles, an agricultural engineer by training, earned his PhD in microbiology at the Pasteur Institute, Paris. After postdocs at Imperial College London and Saint-Louis Hospital, he joined Prof. Brigitte Gicquel's lab at the Pasteur Institute, specializing in mycobacterial biology. He became a CNRS Research Associate (2004) and later founded his lab at IPBS (2007). Now CNRS Research Director, he focuses on tuberculosis and respiratory pathogens. His work has earned major awards, including the CNRS Bronze and Silver Medals, the Sanofi-Institut Pasteur Award, and the Coup d'Élan Prize from the Bettencourt Schuller Foundation, and his lab is recognized for its innovation, global collaborations, and mentoring of young scientists.

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Speaker

Marie-Cécile Ploy

Professor of Microbiology at the Faculty of Medicine and Limoges Teaching Hospital, Limoges University, France

Marie-Cecile PLOY, PharmD, PhD, is a Professor of Microbiology at the Faculty of Medicine and Limoges Teaching Hospital, Limoges University, France. She is Head of the Bacteriology-Virology-Hygiene Department at the Limoges Teaching Hospital and director of the Inserm RESINFIT research unit on antimicrobials at the Limoges University (https://www.unilim.fr/resinfit/). She gained her PharmD in 1994 and her PhD on aminoglycoside resistance in 2000 (Institut Pasteur and Paris XI University). She had a postdoctoral position (2003-2004) in Didier Mazel'lab at the Institut Pasteur in Paris, France, where she focused her research on integrons.
She is part of numerous Committees on Antimicrobial resistance at the national level. She was the coordinator of the European Joint Action an Antimicrobial resistance and healthcare-associated infections (www.eu-jamrai.eu) from 2017 to 2021; and she coordinates the second Joint action, EU-JAMRAI 2, from 2024 to 2027.
She is an expert in numerous national and international research programmes on antimicrobial resistance. She is a member of the Management Board of the JPIAMR and vice-chair of the JPIAMR steering committee. She is vice-dean for research at the Faculty of Medicine, Limoges University. She is full member of the French Academy for Veterinary Medicine
Her research addresses the mechanisms and dynamics of mobilization and spread of antimicrobial resistance. Her main research topics are I) the role of the SOS response in antibiotic resistance acquisition and expression, and II) the risk assessment of the antibiotic resistance dissemination in the environment.
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Speaker

Didier Raboisson

Professor at Toulouse National Veterinary School (ENVT), Toulouse, France

Dr Didier Raboisson is professor in Toulouse Veterinary School where he is teaching veterinary medicine and economics. His research is dedicated to economics applied to zoonotic and animal diseases in a one health perspective. Dr Didier Raboisson has been Attaché for Scientific Cooperation at the Embassy of France in India for 4 years, where he was managing the bilateral scientific cooperation, closely with the Indo-French Centre for the Promotion of Advanced Research (Cefipra).

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Speaker

Amit Singh

Professor at Center for Infectious Disease Research (CIDR), Indian Institute of Science (IISc), Bangalore, India

Indian Coordinator of the IRN MIRA

Amit Singhis a Professor of Microbiology and Cell Biology. Amit Singh's group exploited interdisciplinary strategies to dissect the redox basis of persistence in human pathogens *Mycobacterium tuberculosis* and HIV. By taking advantage of redox biosensors, XF-flux analyses, omics-based strategies, and animal studies, his work has helped find new mechanisms of how macrophage's acidic pH mobilizes drug tolerance in *Mycobacterium tuberculosis*. In 2015, he was awarded the Senior DBT-Wellcome Trust India Alliance fellowship. Most recently, he was awarded the Shanti Swarup Bhatnagar Award, the most prestigious prize by the Council of Scientific and Industrial Research in India. He is a member of the National Academy of Sciences, India.

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Poster sessions

Poster 1

Vishawjeet Barik

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Exploring the role of gene Rv1906c in mycobacterial adaptation and host interaction

The secretome of *Mycobacterium tuberculosis* plays a critical role in its pathogenesis, immune evasion, and intracellular survival. Certain secreted proteins modulate host immunity, facilitating prolonged bacterial persistence and effective transmission within the population. This study investigates the function of one such protein, encoded by the Rv1906c gene. Our findings demonstrate that the deletion of Rv1906c in *M. tuberculosis* reduces the pathogen's ability to survive under fatty acid-rich and low-pH conditions. Differential gene expression (DEG) analysis revealed a downregulation of transcripts associated with mycolic acid biosynthesis, lipid metabolism, and proteolytic processes, while genes involved in immune system interactions and hypoxic adaptation were upregulated. Mycobacterial interactome analysis of Rv1906c suggests its involvement in lipid anabolism and the enhancement of detoxification pathways. Notably, *ex vivo* and *in vivo*, murine infection studies revealed that the absence of Rv1906c enhances bacterial fitness within the host. Furthermore, under *in vivo* conditions, the Rv1906c mutant exhibited increased susceptibility to first-line anti-TB drugs. Cytokine profiling of infected mice indicated a mutant-specific shift towards an M2/Th2-skewed immune response. Collectively, these findings suggest that the secretory protein Rv1906c is crucial for establishing a host microenvironment conducive to long-term bacterial persistence. Targeting Rv1906c may enhance the efficacy of current anti-TB therapies and potentially shorten the duration of tuberculosis treatment.

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Poster 2

Emilie Dordet Frisoni

Research Scientist at Therapeutic Innovations and Resistance laboratory (InTheRes), Toulouse, France

Hidden reservoirs of resistance: Contribution of plasmids to antibiotic resistance in *Staphylococcus aureus*

Staphylococcus aureus, a significant human and animal pathogen, exhibits remarkable capacity for antibiotic resistance development. This is closely linked to its acquisition of antibiotic resistance genes (ARGs) through mobile genetic elements (MGEs), constituting 15-20% of its genome. ARGs in S. aureus spread through highly successful epidemic clones but also via horizontal transfer through MGEs. The associations between ARGs and MGEs are still underexplored in *S. aureus*. Yet, horizontal gene transfer appears to play a crucial role in the emergence of multidrug-resistant clones, posing a major threat to human and animal health.

A large-scale comparative genomic study was conducted to explore the associations between the resistome (set of ARGs) and the mobilome (set of MGEs) of over 10,000 S. aureus genomes of human and animal origin available in the NCBI database. To obtain a more precise and dynamic view of an ARG-rich elements, the plasmid content and their associated antibiotic resistance genes in *S. aureus* strains isolated from animals were characterized using long-read sequencing. These strains were collected through the French surveillance network, Resapath.

Our analyses revealed a wide diversity of MGEs and ARGs in *S. aureus*, with plasmids and transposons being the main vectors of resistance genes. Numerous plasmid/ARG associations were identified, suggesting that these MGEs play a crucial role in the dissemination of resistance. The prevalence of certain families of ARG-carrying plasmids is similar in strains of both human and animal origin, highlighting their dissemination potential independently of the host. Furthermore, plasmids and their associated ARGs can propagate across different sequence types (ST). The high variability of plasmid/ARG associations within a single ST and their spread between STs underline the critical role of these MGEs in shaping the resistome of S. aureus.

Our studies provide valuable insights into the complex interactions between MGEs and ARGs in *S. aureus*. Plasmids serve as the main reservoir of antibiotic resistance genes and, as such, drive the evolution and adaptation of various clones under selective environments. These findings underscore the need to elucidate the mechanisms underlying their epidemic success.

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Poster 3

Nelly Dubarry

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A platform to support drug discovery to tackle *Neisseria gonorrhoeae*

Drug-resistant *Neisseria gonorrhoeae*, and particularly increasing incidence of ceftriaxone-resistant *N. gonorrhoeae*, are making treatment of gonorrhea more and more challenging, with the risk of becoming untreatable. If left untreated, gonorrhea, a prevalent sexually transmitted disease causes by the gonococcus, can lead to complications including pelvic inflammatory disease, infertility, ectopic pregnancy, and an increased risk for the acquisition and transmission of HIV [1]. The World Health Organization (WHO) estimates that 82 millions new cases of gonorrhea occurred globally in 2020 [2].
Recognizing the urgency, WHO listed *N. gonorrhoeae* as a priority pathogen for research and development of new antibiotics in 2017 [3] and CDC have classified drug-resistant *N. gonorrhoeae* as "urgent threat" in 2019 [4]. The new antibiotics zoliflodacin and gepotidacin, both in Phase 3, have shown good antibacterial ability against *N. gonorrhoeae in vitro* [5, 6], but their clinical efficacy still needs to be evaluated. Therefore, there is an urgent need to find new antibiotics to treat gonococcal infections.
To support drug discovery addressing *N. gonorrhoeae*, we propose a platform approach to cover *in vitro* and *in vivo* profiling of new molecules, with the goal to identify new promising drug candidates in an efficient way.

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Poster 4

Chloé Garcia Labaig

PhD student at Institute of Pharmacology
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Design of a Mixed-Species Biofilm Model for the Evaluation of Antibiofilm Therapies Targeting *M. abscessus* and *P. aeruginosa* in Cystic Fibrosis

Mycobacterium abscessus, is an emerging prominent pathogen responsible for severe infections in cystic fibrosis (CF) patients, alongside the persistent threat of *Pseudomonas aeruginosa*. These two critical pathogens share the ability to form resistant biofilms within the lungs of CF patients, underscoring the urgent need for new strategies to limit their spread and develop effective treatments. Such strategies require an *in vitro* model to study the interactions of these two bacteria within a mixed biofilm, investigate their response to treatments and evaluate new strategies to fight these resistant pathogens.
This study aims to develop an in vitro mixed *M. abscessus*/*P. aeruginosa* biofilm model, by establishing conditions that allow the co-growth of both species as adherent cells, while limiting their planktonic growth.We first assessed the planktonic growth of *P. aeruginosa* and *M. abscessus* in various media, classically used for the culture of mycobacteria supplemented with different concentrations of glycerol or glucose, as well as the Synthetic Cystic Fibrosis Medium (SCFM). Other parameters, such as the microplate coating, were also evaluated. Both species were cultured individually and as co-cultures, up to 72h in microplates. Biofilm formation was assessed using both the crystal violet staining method, and the colony forming units counting after resuspension of the biofilm cells. Finally, the biofilms obtained were also observed by Confocal laser scanning microscopy (CLSM) in order to compare their organization and architecture between the different media evaluated.
Optimization of the culture conditions and various parameters enabled us to identify the conditions required for the adhesion of mycobacteria, in particular, to the bottom of the wells, and the growth of mono-species and mixed biofilms. For example, the BBM and de SCFM media were selected following the production of mono-species and the observation of their architecture by CLSM. Mixed biofilms of *Mabs* and *Pa* were then produced in the various media selected.
One of the main aims of this work is to use this *in vitro* mixed biofilm model to characterize the interactions between *Mabs* and *Pa*, and to screen chemical libraries of antibiofilm compounds, alone and in combination with first-line antibiotics, in order to identify new treatment strategies for *P. aeruginosa*/*M. abscessus* co-infections in cystic fibrosis.

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Poster 5

Yasmine Hamchaoui

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Towards a Better Understanding of the *Mycobacterium tuberculosis* FAS-II System

With over 10 million new cases and nearly 1.3 million deaths annually, tuberculosis remains the deadliest infectious disease caused by a single pathogen, *Mycobacterium tuberculosis* (*Mtb*). The high prevalence of multidrug-resistant tuberculosis highlights the urgent need for new and effective therapeutic strategies. The Fatty Acid Synthase type II (FAS-II) system, a megacomplex of discrete monofunctional enzymes, stands out as a central and highly attractive target for anti-TB drug development. Indeed, it is the primary target of several specific antibiotics, including the frontline drug isoniazid. FAS-II is involved in the production of extremely long, atypical fatty acids known as mycolic acids (MAs). These lipids of the outer membrane have a strategic role in the architecture and very low permeability of *Mtb* cell envelope. Also, they are essential for bacterial viability and key actors of *Mtb* virulence.

Although most proteins of the FAS-II system have been identified, several specific enzymes remain unknown, and the 3D structure of the complex is yet to be resolved. With the objective to characterize fully the FAS-II system as a basis for the future development of novel therapeutic approaches, a strategy of affinity purification coupled to quantitative mass spectrometry (AP-MS) is adopted. Known FAS-II core proteins are used as baits to capture co-purified proteins, enabling the identification and quantification of potential interacting partners. This provides valuable insights into the composition and interactome of the protein complex.

Interestingly, while the FAS-II thioesterase responsible for the release of FAS-II products remains unknown, one of the potential FAS-II partners identified is a putative acyl-ACP thioesterase (called TE). In order to validate TE as the long sought FAS-II thioesterase, its physical interactions with FAS-II proteins are examined via the AP-MS strategy, using TE as a bait protein. Furthermore, enzymatic assays with the purified TE protein as well as the lipidomic analysis of *Mtb* TE mutants aim to determine its function in the FAS-II system. Finally, X-ray crystallography analysis will provide insights into the structure-function relationships of this atypical acyl-ACP thioesterase with ultra-long chain specificity.

This work should provide a better understanding of FAS-II at the level of the entire protein complex, paving the way for future therapeutic approaches.

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Poster 6

Gwenaëlle Jézéquel

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Re-exploring the salicylanilide scaffold for the inhibition of mycolic acid synthesis in *Mycobacterium tuberculosis*

Tuberculosis (TB) remains a global health crisis, further exacerbated by the slow pace of progress in developing new treatment options, and the emergence of extreme and total drug resistance to existing drugs. Infection cases keep increasing yearly, with a global number of 7.5 million of people diagnosed in 2022. As for other bacterial bugs, the most pressing challenge lies in the urgent need of finding new active compounds, acting through novel modes of action to tackle resistances. A unique feature of *Mycobacterium tuberculosis* (*Mtb*), the causative agent of TB, is its complex and lipid-rich cell envelope. Therefore, targeting the biosynthesis pathway of cell envelope mycolic acids is a historical strategy against *Mycobacterium tuberculosis*, yet the enzyme FadD32 is still an underexplored although highly relevant (and druggable) target. We have previously developed a miniaturized automatized enzymatic assay, which allowed the high-throughput screening of a drug repurposing library containing 1280 approved human or veterinary drugs (Prestwick Chemical Library). We obtained 36 hits that we further validated for their phenotypic activity on *M. tuberculosis* (MIC 0.08 – 10 µM). This led to identification of one promising pharmacophore, presenting a salicylanilide scaffold that we are now studying further by synthesizing and evaluating derivatives. Exploring SARs, we develop a hit to a lead medicinal chemistry project, searching for a novel anti-tubercular candidate targeting FadD32. The preliminary results of this approach will be presented.

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Poster 7

Saravanan Matheshwaran

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Inhibiting Mycobacterial Transcriptional Repressor LexA to Combat Antimicrobial resistance

Antimicrobial resistance (AMR) is a growing global threat that requires innovative treatment strategies. The bacterial SOS response, regulated by the transcriptional repressor LexA and the co-regulator RecA, drives adaptive mutagenesis, contributing to AMR. Suppressing the transcription of SOS genes by preventing LexA autoproteolysis offers a promising approach to limiting resistance development. While RecA inhibitors face challenges due to their broad conservation across species, LexA, absent in eukaryotes, serves as a more specific target. In this study, we identify and characterize a novel inhibitor that directly interacts with the catalytic residues of *Mycobacterium tuberculosis* (*Mtb*) LexA. By blocking LexA self-cleavage, the inhibitor maintains repression of SOS genes, thereby reducing mutation frequency and AMR acquisition. Using bioinformatics, biochemical, biophysical, molecular, and cell-based assays, we demonstrate the potential of LexA inhibitors as a new strategy to enhance tuberculosis (TB) treatment efficacy.

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Poster 8

Hector Mayoral Reyes

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Targeting Autophagy to Improve BCG Vaccine Efficacy Against Tuberculosis

Introduction: Tuberculosis (TB) is a major health concern due to its high incidence and mortality. The Bacillus Calmette-Guérin (BCG) vaccine, derived from *Mycobacterium bovis*, has been used for over a century but exhibits suboptimal efficacy against pulmonary TB. Therefore, development of more effective TB vaccines is a top research priority.
Autophagy is a lysosomal degradative process implicated in intracellular pathogen elimination, cytokine modulation and antigen presentation, playing a major role in vaccine efficacy. However, BCG's ability to induce autophagy is limited. Thus, enhancing autophagy has emerged as a promising strategy to improve BCG vaccine.
Objectives: The aim of this project is to evaluate autophagy as a potential target for BCG improvement by testing two recombinant strains of BCG (rBCG) that produce specific pro-autophagy peptides. The first objective is to assess immune responses to these rBCGs, *in vitro*, in macrophages and dendritic cells. The second objective is to evaluate immunogenicity, protection and safety of these rBCGs *in vivo*.
Methods: Murine bone marrow-derived macrophages (BMDMs) and dendritic cells (BMDCs) were infected with varying multiplicities of infection using rBCG or control strains. Intracellular bacterial growth was measured in BMDMs by colony forming unit. Cytokine levels were quantified by ELISA in BMDMs and BMDCs.The effects of autophagy modulation were assessed by using autophagy inhibitors and autophagy-deficient cells. Antigen presentation of mycobacterial Ag85A peptide was evaluated in BMDCs using DE10 T-cell hydridoma.
Results: Our data so far indicates that rBCG strains producing pro-autophagy peptides display lower intracellular growth, induce higher levels of proinflammatory cytokines compared to the control strain. Importantly, autophagy inhibitors and autophagy-deficient cells prevent upregulation of cytokines production by rBCG strains. Moreover, rBCG strains enhance MHC class II-mediated antigen presentation by BMDCs. In ongoing studies, we are evaluating in mice models these rBCG immunogenicity, safety and protection against *M. tuberculosis*.
Conclusions: So far, this work shows, that BCG secreting pro-autophagy peptides improve immune responses in antigen presenting cells. These findings suggest that targeting specifically autophagy is a pertinent approach to improve BCG immunomodulatory properties which may have potential consequences for vaccine efficacy and safety.

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Poster 9

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Deciphering the mode of action of promising bioinspired lipidic compounds for antituberculosis drug development

Tuberculosis (TB) is one of the most devastating infectious diseases. The causative agent *Mycobacterium tuberculosis* (*Mtb*) adapts easily to the human host environment and can persist for years inside macrophages. Therapeutic treatments against drug-sensitive TB forms, combining 4 drugs, are 6-month long. The alarming increase in drug-resistant *Mtb* strains highlights the urgent need to identify novel anti-TB agents, acting through new mechanisms of action (MoA).

In this context, we aim to evaluate the anti-TB potential of lipids called Lipidic AlkynylCarbinols (LACs) inspired by the natural falcarindiol, showing moderate anti-*Mtb* activity *in vitro*. Given their unusual chemical structure, LACs could represent an innovative source of anti-TB agents with a previously unexplored MoA. By combining multidisciplinary approaches, our objectives are to: i) generate evolved hits with optimal anti-TB-, pharmacokinetic- and toxicity-profiles through structure-activity relationship studies; ii) elucidate their MoA and identify the associated bacterial targets.

We have evolved the chemical structure by pharmacomodulation and by assessing both anti-*Mtb* minimal inhibitory concentration (MIC) and cytotoxicity against human cell lines.To identify the LACs target proteins and elucidate their MoA, we applied a strategy relying on the isolation of LACs resistant mutants and on a click-chemistry based approach, combining imaging and chemoproteomics.

We selected three hits based on their activity in broth culture and inside infected macrophages and on their low toxicity for human cells, resulting in high selectivity index. The isolation of resistant mutants combined with genome sequencing led to the identification of genes whose mutation or inactivation conferred resistance to selected hits. Using clickable active analogues, we validated their covalent binding to *Mtb* proteins by in gel fluorescence and started the chemoproteomics analyses.

Altogether, we have identified a bioinspired lipidic series with strong anti-*Mtb* activity and high selectivity. We also developed a multi-approach strategy to characterize their MoA, leading to the discovery of new potential *Mtb* targets. The ambition of this project is to validate these synthetic LACs as leads with promising pharmacological properties.

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Poster 10

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Targeting sulfur metabolism to tackle drug resistance in *Mycobacterium tuberculosis*

Inorganic sulfur is the fourth most abundant element in human blood serum and bacteria derive 0.5-1% of their total dry weight from sulfur. It is required for the biosynthesis of several key metabolites – amino acids (methionine, cysteine), coenzymes (Coenzyme A), co-factors (lipoic acid, biotin), and lipids (SL-1) which are required for the maintenance of bacterial physiology, metabolism and for the virulence of *M. tuberculosis in vivo*. Several studies have described the role of different *M. tuberculosis* genes involved in sulfur uptake and assimilation affecting the growth, survival, and virulence of *M. tuberculosis*. However, little is known about the landscape of gene expression in the absence of sulfur in *M. tuberculosis* and the pathways that are largely affected. In our current study, we examined the effects of sulfur depletion on bacterial transcriptome, *in vitro* growth, *in vivo* pathogenicity, and drug susceptibility. We utilised sulfur-depleted media *in vitro* and found that genes encoding components of the bacterial electron transport chain (ETC), proteostasis machinery, central carbon metabolism, major porins, and transporters were down-regulated. Conversely, genes involved in sulfur uptake and assimilation, as well as redox homeostasis, were upregulated. The differential gene expression profile data supported the observed phenotypes, including severe *in vitro* growth attenuation, reduced protein content, decreased ATP levels, and lower rates of replication in the absence of sulfur. Further, CRISPRi-mediated knockdown of sulfate transporter assembly in *M. tuberculosis* revealed a severe *in vitro* and *in vivo* attenuation phenotype and enhanced sensitivity to anti-TB drugs. The absence of sulfate uptake in *M. tuberculosis* resulted in the complete sterilization of infected animals upon 2 weeks of INH treatment. Additionally, blocking sulfate uptake led to *in vitro* as well as *in vivo* fitness of MDR and XDR strains of *M. tuberculosis*. Overall, our findings suggest that targeting sulfate uptake in *M. tuberculosis* can be used as an approach to potentiate the activity of existing anti-TB drugs and tackle the problem of drug resistance in *M. tuberculosis*.

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Poster 11

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Machine learning on high-throughput sequence data reveals novel promoter architectures in *M. tuberculosis*

Gene-regulation in bacteria largely happens at the transcriptional level. Signals within promoters are believed to trigger this process in response to environmental stimuli. However, the identity and function of these regulatory signals on a genome-wide scale are as yet poorly understood. A step forward has been the development of high-throughput technologies that map active transcription start sites (TSSs) at high resolution. Indeed, several studies have reported such maps across different bacteria and conditions. However, the subsequent step of characterizing promoters is typically done on the basis of well-established elements like the -10 box, which precludes the possibility of any novel discovery. Alternatively, overrepresented elements are sought after, a strategy that fails to identify small, diverse promoter-classes.

We have developed a new unsupervised machine learning-based method designed to explicitly characterize architectures of promoters from TSS data. In *M. tuberculosis*, the method provides convincing evidence that the spacing between the -10 box and the TSS is utilized for dynamic regulation of gene-expression by the pathogen. This relationship between the spacing and transcription activity has never been noted before and does not exist in the other bacteria we studied. We believe this result has implications in the physiology of replicating versus non-replicating *M. tuberculosis*. Furthermore, we detect the presence of a pyrimidine preceding the TSS under very specific circumstances, which appears to be a general feature in most bacteria, but missed in all earlier analyses.

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Poster 12

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Impact of chemicals and microbiota from hospital wastewater on the emergence of resistance within sewer systems

Hospital wastewater (HWW) contains antibiotic-resistant bacteria (ARB) and a complex mix of chemical agents, including antibiotics (ATBs). Understanding how hospital-derived microbiota and chemical compounds contribute to antimicrobial resistance in domestic wastewater (DWW) is essential. This study aimed to assess their respective roles in resistance selection within municipal wastewater systems.
The SELECT method [1] was adapted to assess the risk of resistance selection in DWW following exposure to HWW-derived chemicals. Additionally, controlled *in vitro* microcosm experiments were conducted, where DWW was mixed with either whole HWW, its chemical fraction or its microbiota. Resistance selection was evaluated through proportions of ciprofloxacin- and cefotaxim-resistant ARB, relative antibiotic resistance genes (ARGs) abundance via HT-qPCR and ARG sequence variants using multiplex amplicon sequencing.
The adapted SELECT method showed potential resistance selection in DWW microbiota exposed to HWW's chemicals fraction, and that antibiotics were not the only chemicals driving this selection. Microcosm experiments revealed that CIP- and CTX-resistant E. coli proportions were similar when exposed to whole HWW or its chemical fraction, but lower with microbiota alone. This suggests selective pressure from chemical exposure rather than community coalescence drove resistance for this bacterial population. During the first set of microcosm experiments, some ARGs variants were wastewater-type specific, while others were shared. Certain hospital-derived ARG variants (e.g., blaMIR, mdtG) persisted and coexisted alongside DWW variants, whereas others (e.g., blaFOX) were not maintained. These findings indicate that genes from HWW can persist in DWW.
This study highlights the complex interplay between hospital-derived ARGs and chemical agents in municipal wastewater. It provides insights into potential dissemination pathways and persistence patterns, improving our understanding of antimicrobial resistance risks in wastewater systems.

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Poster 13

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Understanding signal transduction mediated by the Mincle receptor

Mincle is a C-type lectin receptor that plays key roles in the innate immune system by acting as a sensor of pathogen-associated molecular patterns. Mincle is a transmembrane protein that consists of a short cytosolic N-terminal sequence and an extracellular domain including a C-terminal carbohydrate recognition domain (CRD). The extracellular and cytosolic domains are connected by a single-pass transmembrane domain. The extracellular binding of trehalose dimycolate, a glycolipid from Mycobacterium tuberculosis, to human Mincle leads to intracellular activation of NF-κB via the Syk-Card9-Bcl10-Malt1 pathway [1,2]. Signal transduction requires FcRγ, a single-pass transmembrane protein bearing cytosolic motifs that are phosphorylated by Src family kinases in the first step of this signalling pathway [3].
We are currently carrying out ligand interaction studies and characterisation of the dynamic properties of Mincle CRD through NMR 15N relaxation measurements and isothermal titration calorimetry. In parallel, a theoretical approach employing multi-scale molecular dynamics simulations is used.
The philosophy behind this project is to implement an integrative approach involving these multiscale simulations and a range of biophysical techniques to understand the molecular mechanisms underlying this signalling event and, potentially, to contribute to the development of new therapies against tuberculosis.

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Poster 14

Laurent Poirel

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The emerging concern of IMP variants being resistant to the only IMP-type metallo-ß-lactamase inhibitor, Xeruborbactam

Objectives: Production of metallo-β-lactamases (MBLs) of IMP-type usually lead to very limited treatment options since MBL hydrolyzing almost all β-lactams and being not inactivated by currently commercialized ß-lactamase inhibitors, including taniborbactam that conversely well inhibit other MBLs of the NDM- and VIM-types. However, the development of xeruborbactam (XER), in combination with meropenem (MER-XER) may shortly offer hope in providing effective combinations against MBL producers. In the present study we evaluated the in-vitro activity of the promising combination under clinical development, MER-XER, against a wide range of IMP-producing clinical isolates, to identify and determine potential resistance mechanisms to XER and the respective BL/BLI combination MER-XER.

Methods: A collection of 32 IMP-producing clinical Gram-negative isolates were included in the study. Susceptibility testing of cefepime, meropenem in combination with TAN or XER at 4 m/L or 8mg/L, was performed by broth microdilution. Furthermore, genes encoding various acquired IMP enzymes from clinical isolates exhibiting resistance to MER-XER were cloned into plasmid pUCP24 and expressed in Escherichia coli TOP10. Additionally, 50% inhibitory concentrations (IC50) of IMP variants was determined. Site-directed mutagenesis and docking simulations were conducted to elucidate the mechanism of resistance to XER of some IMP variants.

Results: All IMP-producing clinical isolates exhibited low MIC values for XER-based combinations, with the exception of isolates that produced IMP-6, IMP-10, IMP-14 and IMP-26. No reduction in MIC values was observed with XER-based combinations in testing recombinant E. coli strains producing IMP-6, IMP-10, IMP-14 and IMP-26, respectively. Interestingly, all IMP-59 producers were susceptible to both TAN and XER combinations. In line with the observed MIC results, a >15-fold higher IC50 value of XER was found for IMP-6, IMP-10, IMP-14 and IMP-26 in comparison to IMP-1, while IC50 value of IMP-59 of TAN was found to be similar to NDM-1. Interestingly, protein secondary structure analysis in comparison to IMP-1revealed that S262G and V67F substitutions are involved in resistance of IMP-6, IMP-10 and IMP-26 to XER. On the other hand, IMP-14 and IMP-4 differing by multiple amino-acid substitutions (H172N, N175S, S65G, Y233N, V280A) remained susceptible to XER). Finally, additional preliminary docking simulations between XER and the IMP enzyme indicated that.

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Poster 15

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The Role of Membrane Fluidity and TCA Cycle in Mycobacterial Bedaquiline Resistance

Mycobacterium tuberculosis (*Mtb*) causes tuberculosis (TB), a chronic disease that requires prolonged treatment with multiple drugs. Eradicating TB still remains a major health management goal. Prolonged TB treatment is associated with compliance issues and leads to the emergence of drug resistance. To map the resistome of *Mtb*, we employed unbiased genetic screening using transposon sequencing (TnSeq) to study the conditional gene essentiality by comprehensively analyzing the effect of loss-of-function or gain-of-function mutations during antibiotic-induced stress conditions. This screen identified multiple genes essential for drug tolerance and resistance towards Rifampicin, Isoniazid, and Bedaquiline (BDQ) in *Mtb*. Many of the genes identified from this screen have shown to be involved with drug resistance/tolerance for INH and RIF, validating the success of this screen. Thus, we focused on BDQ, for which the mechanisms of resistance are not well defined. We found that the TCA cycle plays an important role in protecting Mtb from BDQ-induced cell death. We found that alpha-ketoglutarate decarboxylase (kgd) and isocitrate dehydrogenase (icd2) mutants are hypersusceptible to BDQ treatment. Furthermore, we found that the membrane fluidity of *Mtb* is crucial for drug accumulation within cells and, thus, its anti-TB activity. We found that the deletion mutant of cmaA2 (rv0503c), a gene encoding cyclopropane-fatty-acyl-phospholipid synthase 2, shows resistance towards BDQ. Nonpathogenic strains of mycobacteria, including *M. smegmatis*, lack a functional CmaA2, and BDQ is able to show only bacteriostatic effect in these strains. However, these become sensitive towards BDQ upon the introduction of rv0503c. These findings delineate a new dimension to our understanding of how the TCA cycle is linked with BDQ-mediated killing in mycobacteria, and membrane fluidity is a crucial determinant of anti-TB drug accumulation and subsequent killing.

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The MIRA* International Research Network (2023-2027): a France-India Initiative Against Microbial AMR

Recent decades have seen unprecedented changes in human-environment interactions and increasing disruption of ecosystems and climate, creating a particularly favourable context for the emergence of infectious diseases. In addition to this threat, the worldwide spread of (multi-)antibiotic resistant bacteria represents a major short-term threat to global health. It is therefore essential and urgent to strengthen our efforts to put in place all the necessary means for prevention, surveillance and response to emerging infectious diseases and antibiotic resistance.

By combining their strengths and complementary resources, the 18 partners (4 French and 14 Indian) making up the MIRA network share the common objective of strengthening their research and innovation capacities, from the most fundamental research to applied research for the discovery of new therapeutic and preventive approaches against pathogenic bacteria, through an international collaborative approach.

The partners will endeavour to structure and develop the various areas of cooperation identified and to implement the various levers to perpetuate their joint efforts in favour of global health.

* **MIRA** = **Maladies Infectieuses émergentes et Résistance aux Antibiotiques** / Emerging Infectious Diseases and Antimicrobial Resistance

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University of Toulouse graduate school on Emerging Infectious Diseases (UNITEID)

Strengthening our ability to prevent, detect, and respond to emerging infectious disease threats is crucial. The Toulouse University Graduate School of Emerging Infectious Diseases (UNITEID) aims to achieve this goal and has won the "Skills and Professions of the Future" call under the France 2030 plan. Led by Prof. Pierre Delobel from the University of Toulouse, the project unites ten institutional partners, scientists from ten research laboratories, and an international network of partner institutes, universities, companies, and start-ups.

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Poster 18

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SIG.b: A French academic department for customized methodological development in bacterial genetic engineering, combining conventional tools and CRISPR technologies

The SIG.b department (Service d'Ingénierie Génétique bactérienne) develops engineering tools to modify bacteria chromosomes or transcripts, to specifically increase or decrease gene expression and to carry out arrayed or whole genome functional screens. To do so, we combine conventional approaches including various recombination or repair systems with innovative technologies derived from CRISPR-Cas systems such as interference, display or killing. Taking advantage of the tool box for which we have the handling expertise, of our competence to design new methodologies potentially adapted to any bacterial strain and of our scientific network, we collaborate with research teams (academic or industrial, in France or abroad) facing any challenge in microbial genetics. SIG.b is based in two Toulouse's leading microbiology research institutes: LMGM (Laboratoire de Microbiologie et Génétique Moléculaires), part of the CBI (Centre de Biologie Intégrative) and IPBS (Institut de Pharmacologie et Biologie Structurale). We have access to all microbiology and molecular biology equipments, including BioSafety Laboratories levels 1, 2 and 3, to develop engineering of model, non-model and clinical strains. In order to widely share our theoretical and practical know-how, we also propose professional trainings in partnership with CNRS Formation Entreprises:

- CRISPRi: an innovative application for modulating gene expression in bacteria
- CRISPR genome engineering to generate scarless mutations in bacteria

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Advancing preclinical Tuberculosis research: An expert end-to-end TB platform within a global organization

Tuberculosis (TB) continues to be a major global health challenge, with growing concerns over drug-resistant strains and the need for more effective treatment regimens. Our group utilises a range of well-established and novel preclinical TB models that allows assessment of drug and/or vaccine efficacy and explorations of new treatment and prophylactic options. These models offer distinct advantages for evaluating the pharmacodynamics, bactericidal and sterilising effects of TB drug candidates or immunogenicity and protection of vaccines. *In vivo* acute and highly acute TB models were developed to facilitate rapid "screening" of novel drug candidates, providing preliminary insights into the potential efficacy of therapeutics. In addition, we implemented a relapse mouse model (RMM) that is specifically designed to measure the bactericidal and sterilising effects of drugs alone or in combinations, enabling the identification of compounds with potential for shorten the treatment regimens. This model has been widely used in the PAN-TB consortium (www.pan-tb.org/), with successfully conducted large-scale studies, demonstrating our capacity to manage and execute extensive preclinical trials, ensuring the highest standards. Also, we have developed an advanced Kramnik mouse model, which forms necrotic granulomas that closely mimic the pathology seen in humans, providing a more accurate translation of the disease's progression and the corresponding response to treatment. Finally, to address the increasing challenge of emerging resistant strains, we have developed studies using a bedaquiline-resistant Mtb strain, allowing us to assess the efficacy of new compounds or regimens to overcome resistance. In all cases, the pharmacodynamic data generated from these *in vivo* infection models are integrated with data from *in vitro* profiling and mechanistic assays, as well as pharmacokinetic assessments within phase appropriate modelling frameworks translation. The integration of these models enables us to conduct rigorous evaluations of drug combinations for novel therapeutics, contributing to the advancement of TB treatment strategies. Our platform underscores the value of using multiple TB models in preclinical breakthrough research and their potential to accelerate the development of effective and innovative therapies to fight TB and AMR.

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Boat cruise and cocktail dinner
Access information

May 27th – 7:30 pm

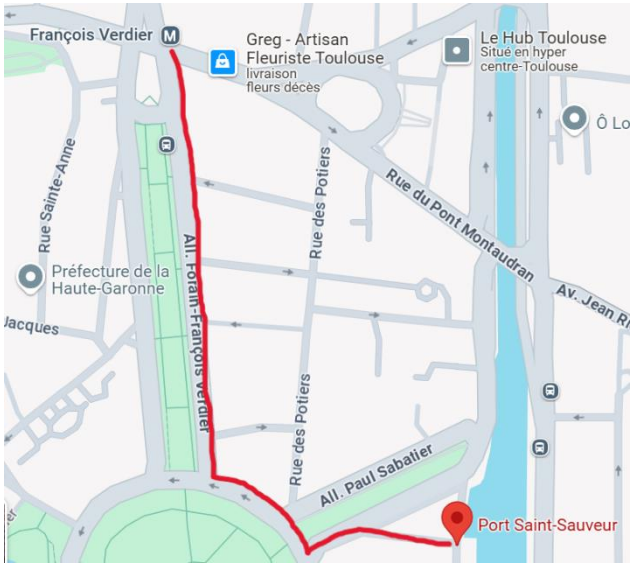
For registered participants

Location

Port Saint-Sauveur
31000 Toulouse

Access from metro station

Station François Verdier – Line B



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